Page 215 to 218

Volume 2 • Issue 1 • 2018

Editorial

Therapeutic Advances in Cardiology

ISSN: 2575-5161

The 'Silent Arterial Duct': Current Controversies

Ageliki A Karatza*

Department of Paediatrics, University of Patras Medical School, Patras, Greece

***Corresponding Author:** Ageliki A Karatza, Department of Paediatrics, University of Patras Medical School, Patras, Greece.

Received: April 09, 2018; **Published:** May 24, 2018

Key words: *Silent arterial duct; Patent arterial duct; Congenital heart disease; Transcatheter occlusion; Surgical ligation; Subacute infective endocarditis/endarteritis; Pulmonary hypertension; Eisenmenger syndrome*

Abbreviations: PDA: Patent arterial duct; AHA: American Heart Association; European Society of Cardiology (ESC)

Volume 2 Issue 1 May 2018

Introduction

The arterial duct is a short vessel which derives from the distal portion of the left sixth embryonic arch and connects the junction of the main and left pulmonary artery to the descending aorta just distal to origin of the left subclavian artery [1-3]. This process is necessary for normal fetal circulation and intra-uterine survival [1]. In fetal life, the arterial duct allows for oxygenated blood to bypass the hypertensive pulmonary circulation, since the lungs are not involved in gas exchange and enter the systemic circulation. After birth, the arterial duct closes and becomes the *ligamentum arteriosum* [4].

The arterial duct closes spontaneously in about 95% of full-term infants during the first 72 hours of life [5]. Persistent patency beyond the third month of life in term infants is a common form of congenital heart disease with an incidence of 1 to 2000 subjects and represents 5% to 10% of all congenital cardiovascular abnormalities [1-3]. Patients with a patent arterial duct (PDA) may be symptomatic or present with failure to thrive and congestive heart failure in infancy, signs of volume overload, recurrent lower respiratory tract infections, pulmonary hypertension/Eisenmenger syndrome, atrial fibrillation, or infective endocarditis/endarteritis [6,7].

The silent arterial duct

With the advent of novel ultrasound technologies, the incidence of persistent patency of the arterial duct is expected to become much higher. If cases detected incidentally on transthoracic echocardiography performed for other purposes are included the incidence is estimated to be 1:500 individuals [1,3,7]. When the duct becomes very small, flow through is diminished and nonturbulent and thus no murmur is audible. The very small duct which is identified incidentally in asymptomatic subjects and produces no murmur on auscultation is termed "*the silent arterial duct*" [6,7].

Over the past 15–20 years, transcatheter closure of PDAs has become the standard of care for most patients and surgery is reserved for those with very large ducts or low weight babies [8]. A number of studies have been published reporting experience with transcatheter PDA closure, in particular using detachable coils and the Amplatzer ductal occluder device [8,9]. In the reported series of transcatheter closure major procedural events occurred in 1.0% of cases, a risk not related to ductal size [9]. The procedural risks specifically associated with closure of a silent duct are not known.

Arterial duct occlusion eliminates volume overload of the left ventricle and pulmonary over-circulation, treats congestive heart failure and prevents both the development of obstructive pulmonary vascular disease/Eisenmenger syndrome and subacute endocarditis/endarteritis. According to the scientific statement from the American Heart Association (AHA) concerning cardiac catheterization and intervention in paediatric cardiac disease, transcatheter PDA closure is indicated for the treatment of a moderate-sized or large PDA with left-to-right shunt associated with congestive heart failure, failure to thrive, an enlarged left atrium or left ventricle or pulmonary over-circulation, provided the anatomy and patient size are suitable [6,9].

Transcatheter PDA occlusion is considered reasonable in the presence of a small left-to-right shunt with normal-sized heart chambers when the PDA is audible by standard auscultation techniques [6]. Small PADs without haemodynamic overload are generally closed because of the risk of subacute bacterial endocarditis [6,9].

There is still controversy related to the need of closure of a silent arterial duct which is associated with a small left-to-right shunt, a normal heart size and an inaudible murmur (Class of recommendation IIb; Level of evidence C) [6]. There are few data supporting significant benefits of occluding it solely to prevent subacute infective endocarditis. As a tiny patent arterial duct does not have a high enough velocity of flow through it to cause endothelial damage which is the substrate for bacterial growth [5]. According to the European Society of Cardiology (ESC) device closure should be considered in small PDAs with continuous murmur, normal LV and pulmonary artery pressure (Level of Evidence IIa; Class of Recommendation C C), but should be avoided in the silent duct (Level of Evidence III; Class of Reccommendation C) [11].

Indeed, infective endocarditis in subjects with silent ducts has been reported only in single-case reports [12-14]. In the adult patients routine follow-up is recommended every 3 to 5 years for those with a small PDA without evidence of left-heart volume overload (Class of recommendation I; Level of evidence C). Closure of a PDA either percutaneously or surgically is indicated in cases previously complicated by infective endocarditis [7].

Infective endocarditis is an uncommon but life-threatening disease and prevention is preferable to treatment of established infection. Bacteraemia with organisms known to cause infective endocarditis occurs commonly in association with invasive dental, gastrointestinal, or genitourinary tract procedures. However, infective endocarditis is much more likely to result from frequent exposure to random bacteraemias associated with daily activities. Although prophylaxis may prevent an exceedingly small number of cases of infective endocarditis in individuals who undergo a dental, gastrointestinal tract or genitourinary tract procedure the risk of antibiotic-associated adverse events exceeds the benefit. Therefore, maintenance of optimal oral health and hygiene may reduce the incidence of bacteraemia from daily activities and is more important than prophylactic antibiotics for a dental procedure to reduce the risk of infective endocarditis. These were the primary reasons for the revision of the Infective endocarditis prophylaxis guidelines of the American Heart Association Committee on Rheumatic Fever, Endocarditis and Kawasaki disease who do not recommend routine subacute bacterial endocarditis prophylaxis for unrepaired PDA [8].

The impacts of the application of the 2007 AHA antibiotic prophylaxis guidelines for Infective endocarditis were studied using a nationally-representative cohort of paediatric patients in the US. The data did not demonstrate significant changes on the overall incidence or severity of pediatric infective endocarditis in the period 2001-2012 [15]. However, a significant increase in disease incidence trend due to viridans group Streptococci was observed in the 10-17 year-old group in the post-guideline period. Infective endocarditis due to viridans group streptococci is presumed to result principally from bacteraemia during dental work, which is more common in older children. It should be noted that the absence of overall change in infective endocarditis incidence from pre- to post-guideline might in part reflect poor adherence to the 2007 AHA policy [16].

Conclusion

The frequency, natural history and best management of the 'silent arterial duct' are unknown [1]. Tra7nscatheter closure is probably not necessary, but remains controversial and requires further investigation. As subacute bacterial endocarditis has been reported in both symptomatic and "silent" PADs, the necessity for subacute endocarditis prophylaxis in an unrepaired small PDA remains to be defined.

Appendix

The application of classification of recommendations and level of evidence according to AHA/ACC is published as: Jacobs AK., *et al*. 'The evolution and future of ACC/AHA clinical practice guidelines: a 30-year journey: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines'. *Circulation* 130.14 (2014): 1208–1217.

The recommendations for formulating and issuing ESC Guidelines can be found on the ESC Web Site (http://www.escardio.org/guidelines-surveys/esc-guidelines/about/Pages/rules-writing.aspx).

References

1. Schneider DJ. "The patent ductus arteriosus in term infants, children and adults". *Seminars in Perinatology* 36.2 (2012): 146-153.
2. Schneider DJ., et al. "Patent ductus arteriosus". *Circulation* 114.17 (2006):1873-1882.
3. Anilkumar M. "Patent ductus arteriosus". *Cardiology Clinics* 31.3 (2013): 417-430.
4. Huff T., *et al*. "Anatomy, thorax, heart, ductus arteriosus". StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing (2018-2017):
5. Kang C., *et al*. "Dynamic changes of pulmonary arterial pressure and ductus arteriosus in human newborns from birth to 72 hours of age". *Medicine (Baltimore)* 95.3 (2016): e2599.
6. Feltes TF., *et al*. "On behalf of the American Heart Association Congenital Cardiac Defects Committee of the Council on Cardiovascular Disease in the Young, Council on Clinical Cardiology, and Council on Cardiovascular Radiology and Intervention. 'Indications for cardiac catheterization and intervention in pediatric cardiac disease: a scientific statement from the American Heart Association". *Circulation* 123.22 (2011): 2607-2652.
7. Warnes CA., *et al*. "ACC/AHA 2008 Guidelines for the Management of Adults With Congenital Heart Disease. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines on the Management of Adults With Congenital Heart Disease)". *Circulation* 118.23 (2008): 2395-2451.
8. Baruteau AE., *et al*. "Transcatheter closure of patent ductus arteriosus. Past, present and future". *Archives of Cardiovascular Disease* 107.2 (2014): 122-132.
9. Wilson W., *et al*. "Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group". *Circulation* 116.15 (2007): 1736-1754.
10. Fortescue EB., *et al*. "To Close or Not to Close: The Very Small Patent Ductus Arteriosus". *Congenital Heart Disease* 5.4 (2010): 354-365.
11. Baumgartner H., *et al*. "The Task Force on the Management of Grown-up Congenital Heart Disease of the European Society of Cardiology (ESC)". *European Heart Journal* 31.23 (2010): 2915-2957.
12. Ozkokeli M., *et al*. "Pulmonary and aortic valve endocarditis in an adult patient with silent patent ductus arteriosus". *Japanese Heart Journal* 45.6 (2004): 1057-1061.
13. Balzer DT., *et al*. "Endocarditis associated with a clinically silent PDA". *American Heart Journal* 125.4 (1993): 1192-1193.
14. Parthenakis FI., *et al*. "Silent patent ductus arteriosus". *Heart* 84.6 (2000): 619.
15. Sakai Bizmark R., *et al*. "Impact of AHA's 2007 guideline change on incidence of infective endocarditis in infants and children". *American Heart Journal* 189.7 (2017): 110-119.

16. Naik RJ., *et al*. "Infective endocarditis prophylaxis: current practice trend among paediatric cardiologists: are we following the 2007 guidelines?" *Cardiology in the Young* 26.6 (2016): 1176-1182.
17. Hoffman JI. "Is spontaneous closure of a patent arterial duct common?" *Cardiology in the Young* 27.1 (2017): 55-58.