Review Article

Interventional Cardiology

Interventional device closure of perimembranous ventricular septal defects: challenges, pitfalls, and advancements in device technology

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Abstract

Percutaneous closure is the standard therapy for muscular Ventricular Septal Defects (VSDs) beyond infancy with a low rate of major complications. The story is somewhat different for Perimembranous Ventricular Septal Defects (PmVSDs) where surgery remains in 2021 the preferable treatment approach in some centers, due to the historical incidence of Complete Atrioventricular Block (CAVB) that has been associated with the asymmetrical Amplatzer Membranous VSD Occluder. It is certain that transcatheter closure of PmVSD is one of the most complex cardiac interventions and have stringent demands on device design due to several challenging considerations. Despite that, experienced interventionists have been continuously reporting successful experiences with PmVSD closure using a variety of device occluders in an off-label indication. Recent meta-analyses confirmed the very good outcomes of this approach and the non-inferiority compared to surgery. However, these devices represent a compromise, as they are not specifically designed to be placed in the perimembranous position. To date, no device achieved market approval in the United States. The need for a device dedicated to PmVSD transcatheter closure is mandatory to standardize the technique and we are very close to achieving this goal. The most recent KONAR-Multifunctional Occluder (MFO) has been smartly designed, combining technical features of previous devices, to tackle encountered difficulties and the outcomes of emerging clinical reports are consecutively encouraging. The MFO specifications are particular but limitations are present and need to be highlighted. This continuous advancement in device technology through continuous physician input will lead to the birth of the ideal device for this intervention.

Keywords: Congenital heart disease • Device closure • Multifunctional occluder • Perimembranous • Ventricular septal defect

Abbreviations

ADO: Amplatzer Duct Occluder; AoV: Aortic valve; CAVB: Complete Atrioventricular Block; LRD: Left Retention Disk, LV: Left Ventricle; MFO: Multifunctional Occluder, PmVSD: Perimembranous Ventricular Septal Defect; RRD: Right Retention Disk; RV: Right Ventricle; SAR: Sub-Aortic Rim; TEE: Transesophageal Echocardiography; TV: Tricuspid Valve; VSD: Ventricular Septal Defect

Introduction

Transcatheter therapy offers unbeatable advantages when compared to cardiac surgery [1-3]. Several devices have been designed and tested over the years to percutaneously close PmVSDs [4-7]. However, the Amplatzer asymmetrical device has been associated with a high risk of CAVB, prompting many to abandon this intervention and pushing other interventionists to use devices designed for other applications in an off-label basis

[8-12]. The debate remains ongoing as the challenges are present, and the pitfalls are numerous [1-6]. Yet, advancements in device technology are major and we are very close to the re-birth of the ideal device dedicated for PmVSD closure [6,13,14].

Literature Review

Challenges of interventional device closure of PmVSD

The history of VSD interventional closure dates back to 1988 when Lock, et al. from Boston reported the first case series of successful closure of muscular VSDs using the Rashkind device [15]. Afterward, other cardiologists followed his lead and used various devices to percutaneously close VSDs but residual leaks and device embolization were not uncommon [6]. The introduction of the Amplatzer muscular VSD Occluder solved this problem as it achieved market approval in the United States and is to date the most commonly used device for percutaneous closure of muscular VSDs, with good reported results [16,17]. Experience in transcatheter closure of PmVSDs was quite different. The intervention presents three specific challenges related to the perimembranous location of the defect and carries a significant risk of complications. Besides the highly heterogeneous anatomical morphology, perimembranous defects are very close to the aortic valve and some PmVSDs can extend beneath the septal leaflet of the tricuspid valve towards the inlet septum [5]. Moreover, PmVSDs amenable to closure beyond early infancy are hemodynamically restrictive but the high-pressure nature of the left-to-right shunt increases the risk of device instability. The double-disc design with septal rims was necessary to hold the device in place, but the nearby aortic valve raised the level of the challenge. The proximity of the conducting tissue to the margins of the PmVSD made the procedure even more challenging as sometimes, a simple wire manipulation across the defect may cause conduction abnormalities [5].

In an attempt to address these challenges, the leader market Amplatzer introduced a specially designed eccentric device, called Amplatzer Membranous VSD Occluder, making the closure of PmVSDs possible, effective and standardized. The device was designed with the aortic edge of the asymmetric left ventricular disc shorter (0.5 mm) than the long apical one (5.5 mm). The lower pole of the left ventricular disc contained a platinum marker to orient device positioning. Its first use was reported by Hijazi, et al. and closure rates were excellent when pooling data from literature [8-10,12,17,18]. However, the procedure was described as challenging as the implantation required an arterio-venous circuit and there was a certain difficulty in properly orienting the occluder. Most importantly, the device never achieved pre-market approval due to the high incidence of CAVB [8-12]. This serious complication was more frequently seen in children, occurred acutely (transiently, during the procedure, or permanently) or quite unpredictably months to years after the procedure but more permanently. The exact mechanism of CAVB remains unclear [4-7]. It has been postulated that the device stretches the defect and possibly exerts pressure on the nearby conduction system with possible impingement against its vascular supply system. Others said that mechanical rubbing of the retention disks to the proximal conduction system leads to localized areas of inflammation or even direct damage with scar formation [19]. A second-generation device with softer edges was redesigned to prevent conduction abnormalities with a 75% reduction in radial force, 45% reduction in clamping force, and 10% increase in stability albeit its use was limited to very small reported numbers [7,20,21].

Looking for an alternative, motivated interventionists started closing PmVSDs using pre-existing devices in an off-label fashion. A very large number of clinical reports documented the feasibility, safety, and short-to-long-term efficacy of this approach in many centers worldwide [22-26]. However, researchers faced two major problems. This off-label practice was initially not encouraged in developed western countries, fearing legal pursuit in case of complication. On another side, interventionists who faced patient death secondary to device-related CAVB no longer dared to recommend this approach. To overcome this fear, recent well-conducted meta-analyses confirmed the non-inferiority of percutaneous PmVSD closure compared to surgery, regarding the safety and the long-term outcomes [1-3,24,25]. The fear of CAVB became quite inexistent and was mostly limited to oversized defects as newer devices are significantly less stiff with improved profiles (incidence of CAVB at a rate of around 1%) [24,25,27,28]. Nevertheless, there was always a variable degree of residual shunting, aortic, and tricuspid injuries [22,23,28]. Most importantly, to date, there is no approved device available that has consistently shown to allow closure of PmVSD with complete safety and efficacy. The lack of one device approach for all anatomies keeps the procedure complex and pushes the interventionist into non-standardized decision making.

Cardiovascular impact of VSD closure

Significant and chronic left-to-right shunting across the VSD leads to left-side chambers volume overload and dilatation with eventually eccentric Left Ventricle (LV) hypertrophy due to amplification of LV wall stress [29]. Moreover, the alteration in systemic hemodynamics related to non-corrected cardiac defects can influence both the systemic and pulmonary vascular performance and theoretically promote the development of endothelial dysfunction and morphological vascular wall

alterations [30,31]. Previous studies showed that timely surgical closure of VSD is associated with a positive outcome on various LV parameters [32,33]. However, the time-related reduction of these parameters is not uniform with a slower pace of LV mass regression compared to other parameters [34]. Closure of cardiac shunts can promote early and long-term amelioration in the systemic vascular endothelial function [35], speculating that left cardiac chambers remodeling can influence the systemic performances of the endothelium [36]. Previous studies demonstrated amelioration in nitric oxide production in the vascular stream after percutaneous closure of cardiac defects [37], partially explaining the complex relationship between the left chambers remodeling and endothelial function [31,38]. With this in mind, the impact of transcatheter PmVSD closure on LV remodeling and systemic hemodynamics also deserves to be fully investigated with a focus on the time course and degree of expected changes.

Discussion

Multifunctional occluder: Advantages and limitations

As a result of a long-standing effort to solve the complex challenges of transcatheter PmVSD closure, the MFO was recently released as the latest addition to the armamentarium of Lifetech (Shenzhen, China) combining the advantages of previous devices to tackle most of the aforementioned difficulties [13,14,39,40]. The device is made of new soft woven mesh with 144 threads of 0.002 inch Nitinol wires to offer high conformability to the defect with a reduction in the theoretical risk of conduction abnormalities and improvement in the profile (Figure 1). The self-expanding device is hybridly designed to combine the advantages of single and double-disc Amplatzer duct occluder devices. The two retention discs are joined by a cone-shaped 4 mm long waist. The Right Retention Disk (RRD) is connected to the central waist by a millimetric connection making it articulated, thus allowing the operator to accommodate the device to various aneurysmal anatomies reducing interference with the Tricuspid Valve (TV). The retention rims exceed the largest waist diameter by 2 mm or 2.5 mm allowing closure of more complex PmVSDs (with deficient or absent Sub-Aortic Rim (SAR)). The availability of eight incremental sizes helps in tailoring the patch as much as possible to the size of the hole, covering a large spectrum of defects even in small patients as the delivery system is low profile (5 to 7 Fr.) and partnered with a slim cable to minimize unwanted damage. The waist of the four large models is securely sewn with a PTFE-membrane to increase occlusion capacity and the double-sided screw increased procedural flexibility with both side deliverability.

That being said, drawbacks still exist. First and foremost, there is a size limitation as the largest Left Retention Disk (LRD) diameter is 18 mm. Moreover, in defects with deficient or absent SAR and LV entry-to-Right Ventricle (RV) exist diameters ratio<2, device oversizing is needed for stability. Therefore, transvenous delivery (arterio-venous circuit) is required to control the deployment of the LRD first within the aneurysm and the possible interference with the Aortic Valve (AoV). In fact, the non-articulating LRD didn't allow to extrapolate the maneuver of pushing on the delivery cable in an attempt to re-orientate the LRD in the LV far away from the AoV. This maneuver successfully used with retrogradely delivered Amplatzer Duct Occluder (ADO) II devices would displace the MFO's central waist solidly connected to the LRD. Furthermore, the RRD is a bit too large and can easily interfere with the TV movement if not properly deployed. The delivery system remains relatively large for the retrograde approach, especially in small-sized patients. Finally and most importantly, the incidence of CAVB is not completely eliminated. Leong, et al. recently reported the early occurrence of CAVB following PmVSD closure using MFO in a 26-year-old woman with subsequent device surgical removal [41]. We believe that unwanted conduction tissue damages are well controlled with the device's very soft and flexible design along with the avoidance of unreasonable oversizing [14,42,43]. However,

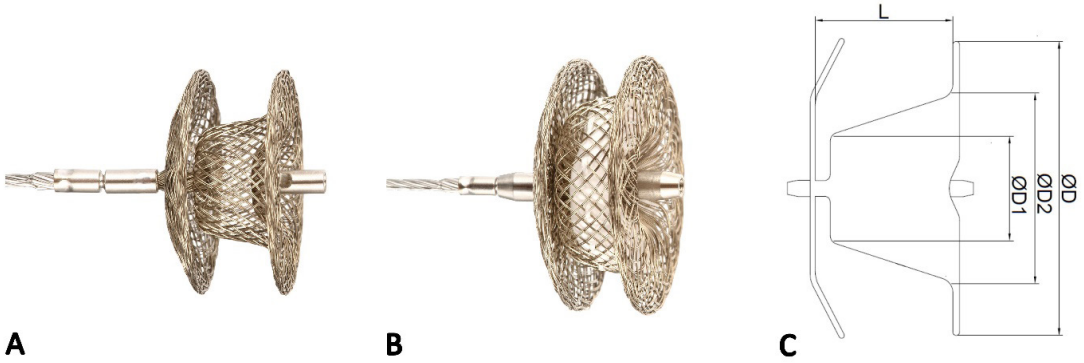

Figure 1: Small (A) and larger (with PTFE membrane) (B) models of KONAR-MF™ VSD occluder connected to the delivery cable (from the right side). Schematic presentation of the different parts of the device: D=left retention disk diameter; D1=waist diameter (right ventricle side); D2=waist diameter (left ventricle side); L=4 mm waist length (C).

caution is always the best statement and only long-term follow-up will reveal the real risk [44,45].

Recommendations for PmVSD closure with MFO

Pre-procedural transthoracic echocardiography planning is highly advisable with a focus on 3 measurements to guide the primary selection of the device size to be later reassessed, by angiography and intra-procedural transesophageal echocardiography (TEE). The LV entry diameter is measured using 3 views (parasternal short-axis, apical 3 chambers view, and subcostal LV-to-Aorta). The SAR, defined as the distance between the AoV annulus and the upper margin of the color flow across the defect is measured using 4 views (parasternal long-axis view, apical 3-chambers, apical 5-chambers, and subcostal LV-to-Aorta). The parasternal short-axis view is also used to define the number and diameters of the RV exit(s) taking into account the largest color Doppler flow. The measurement of the depth is not reliable on transthoracic ultrasound. Single plane 55°-60° LAO/20° cranial projection with per-procedural TEE imaging is sufficient to delineate the anatomy and to precisely guide the entire intervention. SAR length and LV entry diameter are measured on angiography at the largest diastolic phase on the LV side and compared to TEE measurements to average both measurements. The RV exit diameter is evaluated on TEE especially in aneurysmal anatomy and is defined as the narrowest color Doppler diameter during diastole, while the number of exits is better assessed on angiography. Angiography is also used to precisely evaluate the defect depth.

The most common anatomical variant in PmVSDs is the presence of an aneurysm. In case of a large aneurysmal pouch and especially when SAR is deficient or absent, we preferably implant the device within the aneurysm itself to close the true anatomical hole and not to place the device at the entrance of the LV side, avoiding the insertion of an oversized device [14]. This attitude was previously reported to reduce the risk of CAVB as the device is placed far from the conduction system [5]. If the redundant tissue of the aneurysm is relatively small and the device could cover the hole along with the aneurysm it is better to close the true anatomical LV entry with the most appropriate device.

The retrograde implantation of MFO is our first choice of approach as it allows simple deliverability with a decrease in radiation exposure and proper control of valvular interferences [13,46]. However, the prograde transvenous approach offers the possibility to precisely control AoV-to-LRD interference, especially in challenging cases with deficient or absent SAR. In tall adult patients, the delivery sheath inserted transarterially is not long enough to pass across the defect and thus transvenous delivery is required.

Future directions and improvements

We expect in the near future to see an ideal device being marketed for PmVSD closure in children and adults with complete safety. The ideal device is foreseeable as a hybrid fusion of the most promising occluders, the ADO II and the MFO with some additional features. It should be as soft as ADO II or MFO to limit the risk of conduction abnormalities. The double disk design is a must. In fact, the single-disk design of ADO I that was supposed to be an advantage in reducing TV injury can turn out in some cases a disadvantage related to the prograde implantation process. When passing a PmVSD (with multiple exits) from the LV side, the wire might pass through the non-targeted (small) exit. Therefore, when deploying the ADO I transvenously, the secondarily deployed right side of the device might squeeze in that small exit and interfere with the TV movement, causing stenosis and or regurgitation. The retention disks should be asymmetrical with the RRD 2 to 4 mm smaller than the LRD as the main target is only to occlude the LV entry. Both retention disks should be smartly flexible to offer high anatomical conformability (i.e. ADO II) and allow the operator to position each of the retention disks independently for the waist and far away from the nearby valves. The LRD flexibility is highly needed in defects with deficient or absent SAR that are closed retrogradely. The tapered design of the connecting waist is advantageous to accommodate the PmVSD most common conical shape. However, an anatomically adaptable waist (conforming not only to the size but also to the shape, therefore, precisely filling the hole and not only covering it) is ideal. This might eliminate the common attitude of device oversizing thereby avoiding defect "stenting" as previously seen with the asymmetrical Amplatzer device, where the expansion of the device over time exerted pressure on the conducting tissue. Small 2 mm retention rims would be sufficient for device stability (no need for the 2.5 mm rims in odd-sized MFO or the 3 mm rims in ADO II). Both side deliverability is as mandatory as the low profile to allow procedural flexibility and retrograde closure in very small babies with limited risk of tissue damages. Longer delivery sheathes are needed for retrograde delivery in adults. A larger portfolio with LRD diameters up to 20 mm is necessary to cover larger defects. Regarding the delivery cable, the slimness of the MFO cable is excellent for reducing the system profile. When it comes to flexibility, a heavy cable will certainly lead to destabilizing the implanted device with an unwanted rebound effect post-release, meanwhile, a very floppy cable (i.e. Trevisio) will impede appropriate positioning especially in retrograde deliveries.

Conclusion

The addition of the MFO device to the armamentarium of "off-

label" devices used for percutaneous closure of complex PmVSDs is highly valuable, yet some technical limitations are present. Until we witness the birth of the ideal device, patients should benefit from the unbeatable benefits of transcatheter therapy. Albeit a high level of expertise is needed as operators should be very flexible in using a variety of devices and able to modify implantation techniques that are most suited for the anatomy. Appropriate patient selection is of paramount importance to the success of the procedure.

Conflict of Interest

R. Haddad has no conflict of interest to declare. Z. Saliba is a proctor and consultant for Abbott Vascular and Lifetech.

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References

1. Li D, Zhou X, Li M, et al. Comparisons of perventricular device closure, conventional surgical repair, and transcatheter device closure in patients with perimembranous ventricular septal defects: A network meta-analysis. BMC Surg. 20(1): 115 (2020).
2. Saurav A, Kaushik M, Alla VM, et al. Comparison of percutaneous device closure versus surgical closure of peri-membranous ventricular septal defects: A systematic review and meta-analysis. Catheter Cardiovasc Interv. 86(6): 1048-56 (2015).
3. Yang J, Yang L, Yu S, et al. Transcatheter versus surgical closure of perimembranous ventricular septal defects in children: A randomized controlled trial. J Am Coll Cardiol. 63(12): 1159-1168 (2014).
4. Rao PS, Harris AD. Recent advances in managing septal defects: Ventricular septal defects and atrioventricular septal defects. F1000Res. 7: F1000 Faculty Rev-498 (2018).
5. Cinteză EE, Butera G. Complex ventricular septal defects. Update on percutaneous closure. Rom J Morphol Embryol. 57(4): 1195-1205 (2016).
6. Holzer RJ, Sallehuddin A, Hijazi ZM. Surgical strategies and novel alternatives for the closure of ventricular septal defects. Expert Rev Cardiovasc Ther. 14(7): 831-41 (2016).
7. Butera G, Chessa M, Eicken A, et al. Cardiac catheterization for congenital heart disease from fetal life to adulthood. (2015).
8. Holzer R, de Giovanni J, Walsh KP, et al. Transcatheter closure of perimembranous ventricular septal defects using the amplatzer membranous VSD occluder: Immediate and midterm results of an international registry. Catheter Cardiovasc Interv. 68(4): 620-8 (2006).
9. Fu YC, Bass J, Amin Z, et al. Transcatheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: Results of the U.S. phase I trial. J Am Coll Cardiol. 47(2): 319-25 (2006).
10. Masura J, Gao W, Gavora P, et al. Percutaneous closure of perimembranous ventricular septal defects with the eccentric Amplatzer device: Multicenter follow-up study. Pediatr Cardiol. 26(3): 216-9 (2005).
11. Yip WC, Zimmerman F, Hijazi ZM. Heart block and empirical therapy after transcatheter closure of perimembranous ventricular septal defect. Catheter Cardiovasc Interv. 66(3): 436-41 (2005).
12. Butera G, Carminati M, Chessa M, et al. Transcatheter closure of perimembranous ventricular septal defects: Early and long-term results. J Am Coll Cardiol. 50(12): 1189-95 (2007).
13. Schubert S, Kelm M, Koneti NR, et al. First European experience of percutaneous closure of ventricular septal defects using a new CE-marked VSD occluder. EuroIntervention. 15(3): e242-e243 (2019).
14. Haddad RN, Daou LS, Saliba ZS. Percutaneous closure of restrictive-type perimembranous ventricular septal defect using the new KONAR multifunctional occluder: Midterm outcomes of the first middle-eastern experience. Catheter Cardiovasc Interv. 96(3): E295-E302 (2020).
15. Lock JE, Block PC, McKay RG, et al. Transcatheter closure of ventricular septal defects. Circulation. 78(2): 361-8 (1988).
16. Holzer R, Balzer D, Cao QL, et al. Amplatzer muscular ventricular septal defect investigators. Device closure of muscular ventricular septal defects using the Amplatzer muscular ventricular septal defect occluder: Immediate and mid-term results of a U.S. registry. J Am Coll Cardiol. 43(7): 1257-63 (2004).
17. Yang L, Tai BC, Khin LW, et al. A systematic review on the efficacy and safety of transcatheter device closure of Ventricular Septal Defects (VSD). J Interv Cardiol. 27(3): 260-72 (2014).
18. Hijazi ZM, Hakim F, Haweleh AA, et al. Catheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: Initial clinical experience. Catheter Cardiovasc Interv. 56(4): 508-15 (2002).
19. Rao PS. Perimembranous ventricular septal defect closure with the amplatzer device. J Invasive Cardiol. 20(5): 217-8 (2008).
20. Velasco-Sanchez D, Tzikas A, Ibrahim R, et al. Transcatheter closure of perimembranous ventricular septal defects: Initial human experience with the Amplatzer® membranous VSD occluder 2. Catheter Cardiovasc Interv. 82(3): 474-9 (2013).
21. Tzikas A, Ibrahim R, Velasco-Sanchez D, et al. Transcatheter closure of perimembranous ventricular septal defect with the Amplatzer(®) membranous VSD occluder 2: Initial world experience and one-year follow-up. Catheter Cardiovasc Interv. 83(4): 571-80 (2014).
22. Bergmann M, Germann CP, Nordmeyer J, et al. Short- and Long-term outcome after interventional VSD closure: A single-center experience in pediatric and adult patients. Pediatr Cardiol. 42(1): 78-88 (2021).
23. Shah JH, Saraiya SP, Nikam TS, et al. Transcatheter device closure of perimembranous ventricular septal defect in pediatric patients: Long-term outcomes. Heart Views. 21(1): 17-21 (2020).
24. Hong ZN, Chen Q, Huang LQ, et al. A meta-analysis of perventricular device closure of perimembranous ventricular septal defect. J Cardiothorac Surg. 14(1): 119 (2019).
25. Santhanam H, Yang L, Chen Z, et al. A meta-analysis of transcatheter device closure of perimembranous ventricular septal defect. Int J Cardiol. 254: 75-83 (2018).
26. Haddad RN, Daou L, Saliba Z. Device closure of perimembranous ventricular septal defect: Choosing between amplatzer occluders. Front Pediatr. 7: 300 (2019).
27. Bai Y, Xu XD, Li CY, et al. Complete atrioventricular block after percutaneous device closure of perimembranous ventricular septal defect: A single-center experience on 1046 cases. Heart Rhythm. 12(10): 2132-40 (2015).
28. Kozlik-Feldmann R, Lorber A, Sievert H, et al. Long-term outcome of perimembranous VSD closure using the Nit-Occlud® Lê VSD coil system.

Clin Res Cardiol. 110(3): 382-390 (2021).

29. Penny DJ, Vick GW. Ventricular septal defect. Lancet. 377(9771): 1103-12 (2011).
30. Baeyens N, Schwartz MA. Biomechanics of vascular mechanosensation and remodeling. Mol Biol Cell. 27(1): 7-11 (2016).
31. Werner N, Wassmann S, Ahlers P, et al. Endothelial progenitor cells correlate with endothelial function in patients with coronary artery disease. Basic Res Cardiol. 102(6): 565-71 (2007).
32. Yoshikawa M, Sato T. Left ventricular end-systolic wall stress to volume relationship before and after surgical closure of ventricular septal defect. Pediatr Cardiol. 8(2): 93-8 (1987).
33. Cho HJ, Ma JS, Cho YK, et al. Timing in resolution of left heart dilation according to the degree of mitral regurgitation in children with ventricular septal defect after surgical closure. J Pediatr (Rio J). 90(1): 71-7 (2014).
34. Rychik J, Jacobs ML, Norwood WI Jr. Acute changes in left ventricular geometry after volume reduction operation. Ann Thorac Surg. 60(5): 1267-73 (1995).
35. Scicchitano P, Gesualdo M, Cortese F, et al. Atrial septal defect and patent foramen ovale: Early and long-term effects on endothelial function after percutaneous occlusion procedure. Heart Vessels. 34(9): 1499-1508 (2019).
36. Chirinos JA, Akers SR, Trieu L, et al. Heart failure, left ventricular remodeling, and circulating nitric oxide metabolites. J Am Heart Assoc. 5(10): e004133 (2016).
37. Humpl T, Campbell R, Stephens D, et al. Levels of exhaled nitric oxide before and after surgical and transcatheter device closure of atrial septal defects in children. J Thorac Cardiovasc Surg. 124(4): 806-10 (2002).
38. Bissinger A, Grycewicz T, Grabowicz W, et al. Endothelial function and left ventricular remodeling in diabetic and non-diabetic patients after acute coronary syndrome. Med Sci Monit. 17(2): CR73-7 (2011).
39. Bjorkman KR, Aldoss O, Maldonado JR, et al. Transcatheter utilisation of lifetech multifunction™ occluder device for closure of perimembranous and muscular ventricular septal defects: First use in North America. Cardiol Young. 1-3 (2021).
40. Mukherji A, Chattopadhyay A, Ghosh S, et al. Novel use of Konar-multifunctional occluder. Cardiol Young. 30(11): 1722-1727 (2020).
41. Leong MC, Alwi M. Complete atrio-ventricular heart block, a not to be forgotten complication in transcatheter closure of perimembranous ventricular septal defect: A case report and review of literature. Cardiol Young. 1-4 (2021).
42. Boukaram L, Haddad RN, Daou L, et al. Transcatheter closure of congenital Gerbode-type ventricular septal defect using the new multifunctional occluder device. J Card Surg. 36(8): 2986-2988 (2021).
43. Haddad RN, Gaudin R, Bonnet D, et al. Hybrid perventricular muscular ventricular septal defect closure using the new multi-functional occluder. Cardiol Young. 30(10): 1517-1520 (2020).
44. Gozar JD, Cheng DED, Del Rosario JJD. Procedural and short-term outcomes of transcatheter closure of ventricular septal defect using lifetech multifunctional occluder: Initial experience. Cardiol Young. 31(3): 435-445 (2021).
45. Tanidir IC, Baspinar O, Saygi M, et al. Use of Lifetech™ Konar-MF, a device for both perimembranous and muscular ventricular septal defects: A multicentre study. Int J Cardiol. 310: 43-50 (2020).
46. Haddad RN, Rizk C, Saliba Z, et al. Percutaneous closure of ventricular septal defects in children: Key parameters affecting patient radiation exposure. Am J Cardiovasc Dis. 11(1): 65-72 (2021).