Review

Long-term Complications After Transcatheter Atrial Septal Defect Closure: A Review of the Medical Literature

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ABSTRACT

Percutaneous closure has evolved to become the first-line treatment strategy for most cases of secundum atrial septal defect (ASD) in both adults and children. Its safety and efficacy have been proved; percutaneous ASD occlusion offers many advantages over surgical closure, including avoidance of cardiopulmonary bypass, avoidance of sternotomy scar, shorter hospitalization, and a potentially lower incidence of postprocedural complications. Periprocedural course and short-term outcome have been widely described, with low mortality and morbidity rates. However, the wide use of ASD closure devices and the growing experience worldwide brought some delayed and rare complications to light. Device thrombosis and cardiac erosion are the most severe late complications of device closure, whereas atrial arrhythmias are the most common. Other delayed complications include nickel allergy, cardiac conduction abnormalities, valvular damage, and device endocarditis. The long-term complication rate is not null and, although rare,

RÉSUMÉ

La fermeture par voie percutanée est devenue la stratégie de traitement de première intention de la plupart des cas de communications interauriculaires (CIA) de type *ostium secundum* chez les adultes et les enfants. Son innocuité et son efficacité ont été démontrées; l'occlusion par voie percutanée de la CIA offre plusieurs avantages par rapport à la fermeture par voie chirurgicale, y compris l'évitement du pontage cardiopulmonaire, l'évitement de la cicatrice de sternotomie, l'hospitalisation plus courte et une fréquence potentiellement plus faible des complications postopératoires. L'évolution périopératoire et les résultats à court terme ont largement démontré de faibles taux de mortalité et de morbidité. Cependant, la vaste utilisation des dispositifs de fermeture de la CIA et l'expérience accumulée dans le monde entier ont mis en lumière quelques complications tardives et rares. La thrombose sur dispositif et l'érosion cardiaque sont les complications tardives les plus graves de la fermeture par dispositif,

Secundum atrial septal defects (ASDs) account for approximately 6%-10% of congenital heart defects in children and 30% of such defects in adults.[1,2] Percutaneous ASD closure was described in 1974 by King and Mills.[3] Since then, multiple devices have been used and others are still under development before their clinical application.[4-6] At the time of this writing, the most commonly used and US Food and Drug Administration (FDA)-approved devices are the Amplatzer Septal Occluder (ASO) (St. Jude Medical, St. Paul, MN) and the Gore HELEX (WL Gore & Associates, Flagstaff, AZ). The ASO, a self-expandable double disk con-sisting of a nitinol wire mesh, is the most studied device in the literature.[7]

Although surgical closure of ASDs is known to be associated with very low mortality (0%-3%), transcatheter ASD device occlusion has evolved to be the currently preferred treatment strategy, whereas the traditional surgical approach has been dedicated to patients with unsuitable anatomic features or associated cardiac malformations.[8] Percutaneous ASD

some of these complications may be sudden and potentially life-threatening. Moreover, the occurrence and rate of these complications vary with the different devices used currently or in the past. Therefore, both operators and patients need to be aware of these issues to assist them in the choice of intervention or device, or both, and to adapt follow-up modalities. In this review, we sought to describe the type, incidence, and outcome of these rare but potentially serious device closure delayed complications.

alors que les arythmies auriculaires sont les plus fréquentes. Parmi les autres complications tardives, on note l'allergie au nickel, les troubles de conduction cardiaque, les lésions valvulaires et les endocardites liées à un dispositif. Le taux de complications à long terme n'est pas nul. Toutefois, bien que les complications soient rares, certaines sont subites et peuvent potentiellement mettre la vie en danger. De plus, la survenue et la fréquence de ces complications varient selon les dispositifs utilisés actuellement ou par le passé. Par conséquent, les opérateurs et les patients doivent être conscients de ces problèmes pour faire un choix d'intervention ou de dispositif, ou les deux, et adapter les modalités du suivi. Dans cette revue, nous avons cherché à décrire le type, la fréquence et les résultats concernant ces complications tardives liées à la fermeture par dispositif, qui s'avèrent rares, mais potentiellement sérieuses.

occlusion is considered to be safe and offers many advantages over surgical closure, including avoidance of cardiopulmonary bypass and its potential adverse neurologic events, avoidance of a sternotomy scar, shorter mechanical ventilation and intensive care unit and hospitalization duration, a potentially lower incidence of postprocedural complications, and lower cost.[9,10] Short- and long-term device-related mortality rates are known to be low (0.01% and 0.1%, respectively) as reported in a meta-analysis of 28,142 patients from 203 studies.[11] Despite excellent early results, the wide use of these devices and the growing perspective brought some delayed and rare complications to light, such as device thromboembolic events, cardiac erosion (CE), nickel allergy, conduction abnormalities, valvular damage, atrial arrhythmias, and device endocarditis.

Given the frequency of ASD closure, a careful description of the risk of device-related complications can have important implications for physicians in patients counselling and management (choice of surgery vs percutaneous intervention and choice of device) and follow-up. This review aims to describe the type, incidence, and outcome of rare but potentially life-threatening delayed complications—ie, those occurring beyond 6 months after the percutaneous closure—reported in the medical literature.

Thromboembolic Events

In a recent meta-analysis, the estimated rate of device thrombosis was 1.0% (95% confidence interval [CI], 0.8%-1.0%) after ASD closure.[11] This complication has been reported with the ASO but tended to occur more frequently with NTM devices like the CardioSEAL and the STARflex (NMT Medical, Boston, MA).[12] In this analysis of 54 cases of device thrombosis, the ASO was involved in 4 cases of thrombus (7%), whereas the CardioSEAL and STARflex devices were responsible for 13 device thrombosis cases (24%).

In an old report from the Frankfurt team, the incidence and clinical course of thrombus formation after percutaneous ASD closure were assessed in a series of 407 patients.[13] The incidence of thrombus was 1.2%, with significant differences between different devices: the AMPLATZER had the lowest incidence of thrombi (0%) compared with the CardioSEAL (7.1%), STARflex (5.7%), or the HELEX (0.8%) devices. Postprocedural atrial fibrillation was a significant risk factor

for thrombus formation. In most cases, thrombus resolved with medical therapy without clinical consequences.

Most of the device-related thrombi are associated with the healing response of the ASD closure prosthesis. The biocompatibility and histopathologic characteristics of the healing response of various septal closure devices have been investigated only in preclinical animal studies performed for regulatory premarket device approval.[4-6] The corresponding time frame of the neoendothelialization of devices in humans and experimental animals substantiates the common clinical practice of providing antiplatelet treatment for at least 6 months after device placement. Nevertheless, cases of incomplete neoendothelialization from 18 months up to 7 years after device implantation have been reported with the ASO.[14,15] Thus, if more than 6 months is necessary for complete neoendocardial coverage of the device, the risk of thrombus formation persists after the standard 6 months of antiplatelet therapy. To date, no human study exists defining the accurate endothelialization duration of ASD devices.

Abaci et al.[11] specifically looked at the risk of stroke and found that the pooled estimate rate of cerebrovascular events after ASD device closure was 1.1%. This rate of stroke is consistent with other studies,[16] whereas Kutty et al.[17] reported a cumulative incidence of 3% after a median duration of 8.1 years after percutaneous ASD closure. A recent Danish study revealed that the risk of stroke was higher for patients with ASD both before and after closure compared with a control cohort from the general population. Moreover, the stroke risk after closure was significantly related to the presence of atrial arrhythmia. Device-related thrombus was the other main risk factor for stroke after ASD closure.[18]

Erosion

Although no cases of CE were reported after ASO implantation in the first pivotal studies, this potentially lethal complication rapidly became a point of major concern.

The first series reporting CE was published in 2004 and described 28 patients in whom hemodynamic compromise developed after ASO placement.[19] The incidence of CE was 0.1%. Eight patients (29%) were diagnosed between 5 days and 8 months after the procedure, and 1 patient experienced pericardial effusion 3 years after implantation. All erosions occurred at the dome of the atria near the aortic root. A deficient aortic rim was seen in 89% of cases. In the erosion

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group, mean native and balloon-stretched diameters of the defects were 3.6 mm and 5 mm larger, respectively, than in the FDA-approval trials group ($P < 0.001$). Moreover, the device-to−unstretched ASD ratio was significantly larger in this study when compared with that in the FDA trial group (148% vs 138%).

Erosion management varied, with 21 patients requiring surgery and 7 patients managed medically with pericardiocentesis or observation, or both. Among surgically managed patients, 16 had device removal in addition to perforation or fistula repair. In the 5 remaining patients, the device was left in place because it seemed to be in an optimal position, but the perforation was repaired.

Based on their observations, the authors made some recommendations to minimize the risk of device-related CE. These included avoidance of overstretching the defect during balloon sizing by using the stop-flow technique during defect sizing, avoidance of oversizing the device beyond 2 mm to avoid straddling of the aorta, or a mandatory 24-hour follow-up in every patient.

Subsequently, Divekar et al.[20] reported 24 cases of CE after percutaneous ASD closure. Erosion occurred 1.5 hours-3 years after intervention (66% after hospital discharge). The ASO size ranged from 12-38 mm (13 devices ≤ 25 mm; 11 devices > 25 mm). Device malposition was not reported. Ten patients had devices sized equal to the balloon-stretched diameter. All but 1 perforation occurred in the anterosuperior atrial wall or the adjacent aorta, or both. Three patients had neurologic deficits, and 3 patients died; the remaining patients had favourable clinical outcomes.

When analyzing the characteristics of 223 adverse events in patients undergoing ASD closure submitted to the FDA between 2002 and 2007, DiBardino et al.[21] showed that cardiac perforation/erosion/rupture was the second most commonly reported adverse event (22.9%) after device embolization (51%). Extrapolating the rate of ASO implantations in the United States during the study period, the authors concluded that the rate of erosion/perforation/rupture was 0.28% (51 of 18,333 cases). The majority were reported within the first 6 months (16 within 24 hours, 11 within 1 month, and 8 between 1 and 6 months), but erosions were still being reported as late as 3 years after deployment. CE was the most frequent complication−resulting in mortality (10 of 17 overall deaths) in that cohort. From this database analysis, the CE-related mortality rate was 0.05%, which as an isolated cause of mortality was lower than the overall surgical mortality of 0.13% after ASD closure.

In 2014, Amin reviewed 12 cases of erosion that occurred between 2005 and 2012 despite full compliance with the 2004 expert panel recommendations.[19] The author focused specifically on the preprocedural, intraprocedural, or postprocedural echocardiographic data (or a combination of these factors) of the patients. The main preimplantation echocardiographic risk factors for device erosion were a poor posterior rim consistency, the absence of the aortic rim in multiple views, a septal malalignment, and a dynamic ASD (defined as at least 50% decrease in size of the ASD during atrial systole). Once the device was placed, echocardiographic predictors of erosion were (1) a tenting of the atrial free wall into the transverse sinus caused by the edge of the device, (2) wedging of the disks between the posterior wall and the

aorta, and (3) early pericardial effusion.[22] Device erosion has been reported mainly with the ASO device. It has also been described after use of other devices that have a similar design (ie, self-centreing devices like the Cardia ATRIASEPT occluder, CardioLogic, Thirsk, UK) or devices with protruding arms, such as the Clamshell/CardioSEAL device (NMT Medical), which are no longer used.[23,24] Indeed, the ASO is made of nitinol, which is a shape memory alloy. When an oversized device is implanted in a defect, the waist tries to recover its nominal diameter over time. Moreover, the profile of the device improves and the disks of the device flatten over time. This flat profile, which is associated with a very small increase in diameter, may lead to a shearing of the atrial free wall by the edge of the device. An oversized device is wedged between the ascending aorta and the posterior rim of the ASD. The edge of the device near the superior rim may move like a seesaw with every cardiac cycle, leading to a stretching of the free atrial wall and the erosion of the cardiac structure. The proximity of the aorta to the anterosuperior rim of the defect may also make the aorta vulnerable to erosion once the atrial roof has been eroded. This can lead to hemopericardium, tamponade, or aortic fistula.[24] Interestingly, to date no case of erosion has been reported with the HELEX device, which has a different design (helicoidal nitinol wires with no sharp edges on disks). This may be partially explained by the fact that this device is usually not recommended for defects > 18 mm or balloon occlusion−sized defect diameters > 22 mm. Conversely, wire frame fractures have been described with the HELEX device, especially with large devices.[25] This complication usually does not alter the function of the device but has been associated with mitral valve damage. Conversely, no device fracture has been reported with the ASO.

Although no general conclusive data or consensus can be drawn after reviewing the available literature regarding CE after percutaneous ASD closure, several points deserve to be underlined:

1. Cardiac erosion is a rare complication. Although calculation of erosion incidence is speculative and based on estimates (and probably underestimated; some devices may be implanted without an implant card returned to the manufacturer, some may not be used for ASD closure, some cases of erosion may resolve spontaneously), the estimated incidence ranges from 0.04%-0.28%.[21]
2. Erosion events are of particular concern, because they may occur in a vulnerable pediatric population (40% of erosion cases occurred in pediatric patients) and may be an urgent life-threatening event.
3. There are no evident root causes, but several reported erosion risk factors included an absent or deficient aortic rim, protrusion of the device into the atrial or aortic wall (or both), flaring of the device around the aortic root, device oversizing (although erosion has been observed in patients who did not have oversized devices implanted), and early pericardial effusion.
4. There is insufficient evidence to confidently assess which patient subgroups are at increased risk for CE; however, in May 2012, the FDA Circulatory System Devices branch made new recommendations, including frequent follow-up in the first year (ie, serial echocardiography at 1 day, 1

week, 1 month, and 6 months) and yearly thereafter; mandatory device tracking; modifications of the ASO instructions for use (a warning related to the absence of a 5-mm anterosuperior aortic rim was changed to a contraindication); standard training of echocardiographers; additional measures to ensure that patients are informed of the risks/benefits of the procedure.[25,26]

Nickel Hypersensitivity and Migraines

The use of nitinol (an alloy composed of 45% titanium and 55% nickel) devices can predispose the patient to or reveal nickel hypersensitivity. This alloy is widely used in medical products because of its good radiopacity and shape memory properties. Therefore, the high nickel content of such devices is a matter of controversy, because concerns have been raised about the potential release and hypersensitivity reactions to nickel. Nickel hypersensitivity associated with ASD occluders is caused by immune—ie, allergic—reactions.

After ASD closure using an ASO, an increased incidence of migraine headache has been observed during the postprocedural course and was shown to be correlated with nickel hypersensitivity and large device size. Discomfort in the chest, rash/urticaria, difficulty breathing, fever, or pericardial effusion with tamponade after ASD closure have also been described as possible symptoms related to nickel release.[27]

In a series of 150 patients who underwent patent foramen ovale or ASD closure, 7 patients (5%) noted new-onset or increased postprocedural migraine headaches. Of these patients, 4 (67%) tested positive for nickel hypersensitivity, and 2 were not allergic to nickel but had large ASD devices (38 mm).[28] The suggested mechanism was induction of a local inflammatory reaction by the device that may result either in the formation of platelet adhesions that could then embolize to the brain, causing microinfarcts and migraine headache, or release of inflammatory mediators into the left atrium, which then travel to the cerebral circulation and induce migraine headache. Five patients experienced a significant increase in the frequency of migraine headache shortly after clopidogrel discontinuation, suggesting that the pharmacologic suppression of platelet aggregation on the implanted device may be preventing embolization.[29] In line with these hypotheses is the fact that plasma calcitonin gene-related peptide levels, a protein known to be released from specific cardiac tissue, has been shown to increase during migraine attacks occurring after ASD closure, suggesting a key role of inflammatory mediators.[30]

Nickel hypersensitivity—related symptoms might persist for several months but usually respond to medical therapy, including antihistamines, steroids, or the addition of clopidogrel for 3 months.[31]

In rare cases in which medical management fails, surgical removal of the device may be considered. Of note, when compared with the ASO, the HELEX device was not associated with nickel hypersensitivity.[32,33] This may be partially explained by the fact that nickel elution properties and release during in vitro experiments are lower with the HELEX device compared with other occluders.

When performing intracardiac implantation with nickel-based devices, a patch test for nickel hypersensitivity does not appear to be applied systematically because of its lack of sensitivity and specificity. In patients with documented nickel hypersensitivity, ASO implantation is not a contraindication; however, other devices may be considered if available.

Conduction Abnormalities

The proximity of the atrioventricular (AV) node—ie, in the triangle of Koch—to the rims of the ASD makes it at risk of injury after device placement. Although commonly described, conduction abnormalities, including complete AV block (AVB), are scarce after transcatheter ASD closure, with a reported prevalence of less than 1%.[33] The occurrence of complete AVB is classically an acute periprocedural complication; most of the time it is transient and recovers within a short period after corticosteroid therapy. Conversely, only 5 reports of late complete AVB have been described in the literature. The first case of late AVB leading to permanent pacemaker implantation was described by Hill et al.[34] in a 6-year-old patient. Another case of a 2-year-old patient treated with 2 ASDs showed that after initial improvement of an early second-degree AVB (Mobitz type II) with steroids, over a course of 4 years there was a subsequent deterioration of AV conduction to complete AVB, which required an epicardial dual-chamber pacemaker.[35] Szkutnik et al.[36] described in their series 2 patients, aged 15 and 16 years, with complete AVB diagnosed 4.3 and 1.5 years, respectively, after ASO implantation leading to pacemaker implantation. Recently, Dittrich et al.[37] published the first case of complete AVB occurring after GORE Septal Occluder (W.L. Gore and Associates, Flagstaff, AZ) implantation, which occurred 11 months after device implantation in a 2-year-old patient. In contrast, late development of AVB with ASD, corrected or not, is a recognized complication caused by a well-described genetic association (*NKX2-5* mutation). These observations underline the fact that careful cardiac rhythm monitoring is critical during long-term follow-up after percutaneous ASD closure, even in patients without early postprocedural cardiac conduction abnormalities. Some authors also recommend that early removal of a pushing device be considered if the use of a smaller device or surgical closure of the defect is feasible to give the best chances to the AV conduction to recover and above all to avoid pacemaker implantation and its inherent morbidity.

Valvular Damage

An onset or worsening of mitral regurgitation (MR) may occur in 10%-37% of patients after percutaneous ASD closure, but a pre-existing MR may also improve after the procedure.[38] The MR is usually trivial to moderate and without clinical significance. The alterations of atrial function, including atrial stiffness from the device, and geometric changes of the left side of the heart after atrial shunt disappearance may explain this observation.

Wilson et al.[39] described a series of 194 patients with a mean follow-up of 1.2 years after ASO placement. Among those patients, the degree of MR was unchanged in 160 patients (88%), increased in 20 patients (10%), and decreased in 13 (7%) patients, including 1 patient who had severe MR, which decreased to trivial MR. Similar results were found recently in a series of 288 patients, which also showed that patients with MR deterioration were older and more likely to

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be women. MR was also related to significant mitral annulus echographic geometric changes. After a median follow-up of 24 months, 7 patients had cardiovascular events. These complications occurred exclusively in patients without MR deterioration after ASD closure, showing that MR deterioration was not related to an altered prognosis in those patients.[40]

Aortic regurgitation (AR) has also been described as a potential long-term complication after transcatheter ASD closure; however, published data are scarce and have contradictory findings. Schoen et al.[41] reported a series of 70 patients who underwent ASD closure using ASO (57%) and Cardia devices (Cardia, Eagan, MN) (43%). Sixteen percent of the patients had pre-existing mild AR. The authors found novel or worsened AR in 9% of patients after ASD closure independent of age, sex, type of device, or size of the defect. They hypothesized that AR was likely caused by the modification of the septal geometry from the device, leading to traction at the noncoronary aortic valve sinus.[41] Conversely, in another series of 200 patients in whom an ASO was implanted, mild AR occurred in only 1% and was correlated with a device-to-defect ratio of > 1.3:1, suggesting a detrimental role of oversizing.[42]

The most recent study that has investigated the impact of ASD device closure on AR was published by a Mayo Clinic team and reported an incidence of 0.8% (1 of 118 cases) of AR after a mean follow-up of 1.2 years.[43]

Regarding the tricuspid valve, functional regurgitation is common before ASD closure but was shown to persist in nearly half of patients because of excessive structural changes in tricuspid valve anatomy (annular dilatation and enlarged septal leaflet).[44]

In summary, valvular dysfunction, whether mitral, tricuspid, or aortic, may occur with a relatively low incidence and negligible clinical impact; however, physicians have to pay attention to these specific complications because they can lead to surgical valvular repair.

Atrial Arrhythmias

It is well described that in unclosed ASD, the incidence of atrial fibrillation (AF) increases with age to reach 50% in individuals older than 60 years. After percutaneous closure, atrial arrhythmias appear to be the most common complication in patients without pre-existent arrhythmias.

Vecht et al.[45] suggested a beneficial effect of the closure on pre-existing AF on midterm follow-up (up to 5 years). This analysis included both surgical and transcatheter approaches, but when the authors focused on the percutaneous closure subgroup analysis, the beneficial effect of closure remained significant (odds ratio [OR], 0.49; 95% CI, 0.32-0.76).

Conversely, in patients without pre-existent arrhythmias, the rate of AF increases after closure, as shown in the recently published Danish nationwide cohort.[18] Among these 1167 patients, 300 had a percutaneous closure and a median follow up of 5.2 years. These authors showed that patients with ASD had a higher risk of new-onset of atrial arrhythmias (adjusted hazard ratio, 8.2) after closure than the comparison cohort but with no difference between transcatheter and surgical approaches. In patients with closure before the age of 25 years, AF developed in 21% during follow-up.

There are few data on how to manage such arrhythmias.[46] A few articles suggest management that includes classic anticoagulation and antiarrhythmic drugs, whereas refractory cases might be treated using catheter ablation, which is feasible in experienced hands using trans-septal access through the device.[47]

Endocarditis

The ASD closure procedure is typically performed under strict asepsis with the administration of prophylactic antibiotics. However, device-related endocarditis may rarely occur after ASD closure and, excluding reports from the manufacturer and User Facility Device Experience database, has been described 6 times in the literature.[48-52] Patient age ranged from 4-71 years. Infective endocarditis involved the ASO device in all cases and occurred from 11 months up to 4 years after device implantation. The culprit bacterium was mainly *Staphylococcus aureus* (4 of 6 cases). Of note, 1 patient had undergone periodontal scaling without antibiotic prophylaxis 1 month before his febrile episode, based on the current guidelines.[53] Surgical removal of the device was performed in 4 cases, showing an incomplete neoendothelialization of the device. No deaths occurred.

This complication, although rare, underscores 2 main issues: (1) as mentioned in the section on thromboembolic complications, there is an undetermined proportion of cases of delayed/incomplete endothelialization of the device, and to

Table 1. Summary of the main long-term complications after percutaneous ASD closure

Complication	Incidence	Delay from closure	Complications	Risk factors	Treatment
Cardiac erosion	0.04%-0.28%	Up to 9 y	Death (0.05%) Stroke	Absent or deficient aortic rim Device oversizing Device protrusion into atrial or aortic wall, or both Flaring of the device around aortic root	Surgical repair of erosion ± device removal Medical management (pericardiocentesis)
Device thrombosis	0.8%-1.2%	Up to 2 y	Stroke (10%-15%)	Postprocedural AF Coagulopathies	Antithrombotic therapy Surgical thrombectomy
Atrial arrhythmias	11% 10 years after closure	—	Stroke	Early ASD closure	Antiarrhythmic agents Catheter ablation
Complete AV block	5 published cases	Up to 4 y	—	Early conduction abnormalities AV conduction deterioration	PM implantation
Infective endocarditis	6 published cases	Up to 4 y	—	Lack of antibiotic prophylaxis	Antibiotics ± surgical device removal

AF, atrial fibrillation; ASD, atrial septal defect; AV, atrioventricular; PM, pacemaker.

date, there is no specific method for confirming complete endothelialization on the surface of the device in individual patients and (2) in the case of incomplete endothelialization, there is still a delayed risk of device infection, suggesting the prolongation of antibiotic prophylaxis in these patients.[14,15] Currently, antiplatelet therapy and prophylaxis of endocarditis are recommended for 6 months after device implantation; however, these guidelines are based on the results of animal experiments. Further experimental and clinical studies are needed to better address these points of concern.

Table 1 summarizes the main long-term complications after percutaneous ASD closure with their risk factors and treatment.

Follow-up Guidelines

Most of the complications discussed in this article involve the ASO device. Indeed, the ASO represents the vast majority of implants in developed countries; it is the only device used in larger defects (at least in North America), and its scrutiny is important (in part because of the erosion issue). Conversely, some emerging devices (like the Figulla Flex, Occlutech GmbH, Jena, Germany, or the Cera Occluder, Lifetech Scientific, Shenzen, Republic of China) may become more than alternative options to the ASO, because large published series reported promising results.[54,55]

The American College of Cardiology/American Heart Association guidelines for the management of adults with congenital heart disease published in 2008 state that after a percutaneous ASD closure, the recommended frequency of clinical and transesophageal echocardiographic follow-up is 24 hours, 1 month, 6 months, and 1 year and at regular intervals thereafter. In addition, electrocardiographic surveillance for recurrent or new-onset arrhythmia is also mentioned as an important feature.[56] Similarly, European Society of Cardiology guidelines published in 2010 specify that regular follow-up during the first 2 years and then, depending on results, every 2-4 years is recommended.[57]

Regarding the possibility of potentially serious long-term adverse events, we do think that the key aspect of follow-up is to inform both patients and their primary care physicians about (1) the requirement of lifelong follow-up, (2) the different types of complications, and (3) the need to report symptoms such as fever, chest pain, or syncope, because they might represent early signs of device-related complications.

Finally, there are few data available on the long-term tolerance of devices implanted in growing children. However, despite this lack of literature, recent echocardiographic and magnetic resonance imaging studies that focused on large implanted devices in children showed that the distance between the device and the surrounding structures increases with growth, likely decreasing the risk of long-term complications.[58,59]

Conclusions

Percutaneous ASD closure has already proved safe and effective using a great number of device types. The early complication rate is quite low when compared with its surgical alternative. Most reported complications involve the ASO device because it is most commonly implanted, especially in large defects. Device thrombosis and CE are the most important late complications of device ASD closure, whereas atrial arrhythmias are the most common. The long-term complication rate is not null, and, although rare, some of these complications can be sudden and potentially lethal. Therefore, both operators and patients need to be aware of these issues. Long-term follow-up of patients after ASD closure is mandatory to detect these potentially serious late events.

Disclosures

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