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Editorial

Percutaneous Occlusion of Atrial Septal Defect – As You Please!

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Atrial septal defects (ASDs) correspond to 10% of birth defects in newborns and to 30% to 40% of defects in adults.[1] Children are generally asymptomatic, but only 4% of patients above 40 years deny symptoms.[2]

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Although ASDs are generally well tolerated, some complications can arise in adulthood; the most feared is the onset of lung vascular disease. Significant pulmonary arterial hypertension can occur in approximately 22% of adults, with the possibility of vascular pulmonary hyper-resistance development in 15% of these patients.[3] Other problems that may occur in the natural history of ASDs are: increase of right cavities with right ventricular dysfunction, systemic desaturation, paradoxical embolism, and atrial arrhythmias, whose incidence increase with age.

Introduced in 1948 by Murray, the surgical correction of *ostium secundum* ASD is a well established, safe, and effective procedure, with a satisfactorily low mortality rate. However, it presents complications (major and minor), requires a longer hospitalization, causes pain, provokes visible scarring, and demands greater recovery time compared to percutaneous implants.[4]

Percutaneous occlusion, initially described by King and Mills in 1976, paved the way for modern interventional cardiology to treat congenital defects, and it is perhaps the most commonly performed procedure in this field. The first report of percutaneous occlusion of an *ostium secundum* ASD in Brazilian literature was published by Haddad et al., in 1996.[5] At that time, double disk prostheses without centralizing mechanism (Rashkind's double umbrella, CardioSEAL®, StarFLEX®, and Sideris Buttoned Device®) prevailed. Difficult to handle, not repositionable, and with only reasonable results, these devices did not encourage Brazilian interventionists, and their use was limited to a few cases in Rio de Janeiro and São Paulo.

The release of prostheses made of nitinol mesh (Amplatzer Septal Occluder® [ASO]; AGA Medical Corp. – Golden Valley, USA), in 1997, was responsible for an exponential increase in the number of procedures worldwide. The ASO was composed of fine nitinol wires forming two disks, and a central part that acted as an expansible centering mechanism, occupying the defect in its entirety, and corresponding to the nominal size of the prosthesis. It was a very elastic material and with large mechanical memory, allowing for the compression of the devices into a long sheath, in order to restore their original shape when externalized. They had polyester flaps sutured at three points of the device, for the purpose of inducing thrombosis in its interior. The ASO prostheses also had major advantages compared to previous devices: they were able to occlude larger defects (up to 40 mm), did not need the aortic edge for fixation, were easy to handle, and were fully retrievable and repositionable while joined to the delivery cable, enabling a smaller learning curve. Thus, the ASO quickly became the device of choice for occlusion of *ostium secundum* ASDs worldwide. Its manufacturers obtained approval from the Food and Drug Administration (FDA) for use in the United States and are, to date, the most referred devices in the international medical literature.[6] The Brazilian experience with this device is vast and well documented.

Despite the excellent results presented, in association with the large number of implants, some problems arose: the emergence or worsening of headache, significant arrhythmias (especially total atrioventricular blocks [TAVB], and atrial fibrillation [AF]), late embolization

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of the device, pericardial effusions, thrombi formation in the prosthesis' left disc, and the extremely troubling erosions. General complications of the procedure with the use of the ASO are few (8.6%), but may occur immediately, or several years after its implantation.[7]

The ASO is composed of 55% nickel by weight, an element comprising part of the nitinol alloy. The release of nickel from the device starts immediately after its implantation and reaches its maximum in one month. This may cause allergic and toxic reactions in sensitive patients. While this has not proven to be a problem of great magnitude, some cases of occurrence or worsening of migraine in patients who underwent implantation of the ASO have been attributed to an allergic reaction to nickel. Alternatively, the platelet activation on the surface of the device has been proposed as a triggering mechanism of migraine; this hypothesis is supported by the therapeutic response to clopidogrel in some cases, but not in all. The stretching of the atrial septum caused by the presence of the device and leading to the release of atrial natriuretic peptide, a vasoactive antagonist of vasopressin, by myocytes, has been suggested as another theory for the onset of migraine.

The presence of atrial arrhythmias is not uncommon in patients with *ostium secundum* ASD. Electrophysiological changes in the sinus node or in the system of atrial conduction may be due to a volumetric overload, or to the atrial distension in patients with hemodynamically significant ASDs and with increased right cavities. Studies with dynamic electrocardiography (Holter) revealed no changes in basal rhythm in 90% of patients after the procedure, but there have been changes in atrioventricular conduction in 7% of patients studied.

Other trials have demonstrated that the percutaneous occlusion with ASO may be associated with TAVB, which, in turn, may occur during or immediately after the procedure, requiring electrocardiographic monitoring. Nonetheless, most cases are transient, with spontaneous reversion; they mainly occur in defects that need the largest prostheses. The incidence of TAVB in children fluctuates at around 6% of cases. In adults, the incidence is even lower, probably due to the larger size of the patients compared to the prostheses.

The onset of AF after the procedure entails increased risk of thromboembolic complications before a complete endothelialization of the device. The incidence of AF appears to increase after percutaneous occlusions of *ostium secundum*-type ASD, but this is also a well-described complication in untreated patients. These data led some authors to speculate whether the presence of postprocedural AF would be a consequence of the device, or just a complication expected from the defect. Patients with pre-existing paroxysmal AF appear to only be able to maintain sinus rhythm after the occlusion of the defect. However, in isolation, the exclusive closure of the ASD does not appear to be able to produce these results, and the use of associated electrophysiological procedures becomes necessary.[8]

Device embolization has been reported in most case series, with an overall incidence of 0.1 to 3.5%. This complication can occur immediately or days, months, or years after implantation. When it occurs during the procedure, rescue can usually be performed by capturing the device with a snare catheter and pulling it through a long sheath of larger size compared to that used for the implantation of the prosthesis. Some time after implantation, the device becomes rigid due to the formation of a thrombus inside it, and thus no longer allows for the capture and percutaneous removal. In such cases, the treatment option is surgical remotion, with elective closure of the defect, or as an emergency procedure.[9] When embolized, the device tends to shift to the right atrium, from where it crosses the tricuspid valve and usually transits to the pulmonary trunk or its branches. Depending on its position relative to the axis of the vessel, it may not cause obstruction.

Pericardial effusions are described in most series, but without a convincing explanation for its apparition. These complications may occur immediately after the procedure, as a result of heart perforation, or days after the procedure, without a specific cause; but it is speculated that it may be due to an immune reaction.[7]

The presence of thrombi attached to the device, a complication feared due to its consequences, appears to be less frequent in nitinol mesh devices than in previously existing devices. Post-procedural AF and presence of atrial septal aneurysms appear to be predictive of thrombus formation, but dual antiplatelet therapy has been shown to be quite effective in preventing this complication.[10]

Erosions caused by ASO are rare, but potentially fatal events. These are unpredictable situations of extreme risk to patients, and can be of late occurrence. Although its mechanism is still unknown, erosion appears to be related to the abrasion of adjacent heart tissue by the metal prosthesis, and may have some facilitating agents, for instance, associated structural defects (as in Marfan syndrome), changes in tissue thickness and composition related to age, and variations in the ability of the tissue to resist to abrasive forces.[11] Overestimation of the size of the device and contact of the edges of the prosthesis with the aortic or atrial wall, in association with heart movements, may act similarly to the mechanism of a circular saw.

The thickness of nitinol wire increases in certain sizes of this device (at 11, 18, 26, and 32 mm), to maintain its integrity and shape. Most erosions in the cases described occurred in patients who received the 26 mm ASO, which is proportionally the most rigid

type for the nitinol wire thickness used in prostheses of 26-32 mm.[12]

In 2004, the estimated incidence of cases of erosion in United States using the ASO device was 0.1%. Other publications relate erosion rates ranging between 0.07 and 0.28%, based on the number of ASOs sold or implanted. Clearly, these numbers are only estimates, since many patients do not have their implants registered, and many authors don't report their complications; in any case, the incidence of this complication is fortunately low.

As to the time of appearance, erosions can occur days after implantation or up to six years after the procedure. The mortality rate caused by erosion varies from 0.004 to 0.015%.[13] With respect to the location, the erosions reported occurred at the top (roof) of the right and left atria (with or without aortic involvement), in the aortic wall, and in the wall of both atria; in some cases, the site of the lesion was not identified. Erosions appear to be more frequent in defects of large diameter, located more superiorly, without aortic rim, and in those receiving oversized prostheses.[13] Nevertheless, the majority of interventionists dedicated to the closure of *ostium secundum* ASDs believe that the primary culprit of the erosions is the movement of the prosthesis in the atrial septum.

Although the causes of erosion are not yet fully clarified, some recommendations were proposed by a panel of experts in an attempt to minimize these episodes, such as avoiding the stretching of septum with the measuring balloon using the technique of flow occlusion (stop-flow), trying to identify patients at risk (high, large defects, without aortic rim), and closely following small pericardial effusions in the first 24 hours after the procedure.[14]

The consensus is that, despite the complications reported, undoubtably the percutaneous occlusion of *ostium secundum*-type ASD with ASO is a safe and effective procedure. In the wake of the success of the AGA prostheses, a new generation of devices was released, aiming to correct deficiencies or introducing some interesting modifications in the design of the prostheses, their composition, in the amount and thickness of the nitinol wire, in its external coating to prevent release of nickel, and in different release mechanisms. Unfortunately, the number of publications in the literature worldwide focusing on these new devices is scarce, perhaps due to the fact that these devices have not been approved yet by the FDA. Even though most of these devices were approved by the Brazilian Health Surveillance Agency (Agência Nacional de Vigilância Sanitária [ANVISA]) and have been used in this country by several renowned experts with excellent results, the number of Brazilian publications on this subject is almost nonexistent. Thus, the publication of the article by Haddad et al.,[15] in this issue of **Revista Brasileira de Cardiologia Invasiva (RBCI)** is quite auspicious, which reports the initial experience of the authors with a new prosthesis for closure of *ostium secundum*-type ASD.

For each of these new devices, there are advantages and disadvantages regarding technical aspects and prosthesis profile. All of them are within our reach, and offer interesting options for safety, effectiveness, and reproducibility, so that the professional may choose the one that best fits his/her aims. Certainly, with the various options currently available, the choice of prosthesis depends on the preference of the customer!

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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