Incomplete Endothelialization and Late Dislocation After Implantation of an Amplatzer Septal Occluder Device

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A 64-year-old woman with a history of a large-sized secundum atrial septal defect (ASD) underwent successful percutaneous closure with a 34-mm Amplatzer septal occluder (ASO) without apparent complications on April 25, 2002. Transthoracic echocardiography immediately following the closing procedure revealed that her ASO was well wedged, with no residual shunt or murmurs (Figure 1). At the third year of follow-up, a small volume of split flow was detected by transthoracic echocardiography. No intervention was attempted at that time because the shunt was not clinically significant and the patient had no uncomfortable symptoms. However, at the last follow-up visit in 2009, the patient had a chest distress and palpitation after daily activity; transthoracic echocardiography showed that the ASO was dislodged into the left atrium and that only a part of the ASO remained at the defect border (Figure 2).

A decision was made to surgically remove the ASO (Figure 3). During surgery, we found that the surface of the ASO was only slightly endothelialized on the right side of the disc with approximately 1 cm of the device still adherent to the inferior border of the atrial septum (Figure 4A). There was minimal endothelialization on the left side (Figure 4B). Pathological examination of the explanted ASO showed fibrous connective tissue without thrombi or neoplastic formation on either surface.

ASO has become one of the most frequently used systems for transcatheter repair of ASDs. It has been thought that the percutaneous closure of type II ASD with ASO is safe and effective during long-term follow-up.1 Device thrombus for-
ation and dislodgement are the most common early complications of percutaneous ASD closure. To date, the data on late complications following percutaneous closure of ASDs are scarce. Limited experience has shown that occluder surface thrombus formation and infectious endocarditis are the major late complications. Andreas et al reported that the incidence of thrombus formation was 1% with the Amplatzer device. Zahr et al reported that a patient had poor endothelialization and developed endocarditis 30 months after implantation. This finding suggests the importance of antibiotic prophylaxis until endothelialization is completed.

It has been shown that most ASD repair devices fully endothelialize within 3 months in animals. However, this may not be the case in humans, as in our patient. This is consistent with the case reported by Chessa et al. They reported that an implanted ASO was not endothelialized after 18 months.

To our knowledge, early dislocation has only been reported into the right atrium or the right ventricle, and there are no reports on incomplete endothelialization and late-stage device dislocation after the implantation of an ASO. In our patient, the ASO was dislodged into the left atrium and partially adhered to the inferior border of the atrial septum. Several risk factors might have contributed to this complication. First, the ASD was large in this case (3.21 cm), and the waist diameter of the used ASO was 34 mm (the largest ASO available at that time). Therefore, it is reasonable to assume that the occluder was unable to complete effective closure of the patient’s ASD and subsequent complete endothelialization over the device. Second, preoperative transthoracic echocardiography showed increased right ventricular volume (73 mL) and moderate tricuspid regurgitation (5.84 mL). Transthoracic echocardiography in 2009 showed worsened right ventricular dilatation (109 mL) and increased tricuspid regurgitation (16 mL). Enlarged right heart chambers and increased tricuspid regurgitation and insufficient endothelialization, as well, may contribute to the dislodgement of the ASO into the left atrium, although these are also the results of a dislodged ASO.

Our report emphasizes the importance of carefully assessing the size and borders of the ASD and the degree of tricuspid regurgitation, especially in patients with large ASDs. It is critical to use a matched occluder. It may be necessary to extend the anticoagulation treatment time from 6 months currently to more, with longer follow-up after ASO closure of large ASDs.

Disclosures

None.

References

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