Case Report

SURGICAL REMOVAL OF EMBOLISED ASD DEVICE FROM PULMONARY ARTERY

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ABSTRACT
Embolization of the ASD closure device is a rare but potentially fatal complication of percutaneous atrial septal defect (ASD) closure. We report a case of 22-year-old man who underwent ASD device closure with 32 mm Amplatzer device, which was embolized to the pulmonary artery without symptom one day after successful device implantation.

Keywords: Atrial Septal Defect, Amplatzer Device

INTRODUCTION
Atrial septal defect (ASD) is one of the most common congenital heart defect found in adults. Percutaneous device closure using Amplatzer ASD occluder (AGA Medical Corp., Golden Valley, MN, USA) is widely used for treatment of ASD and proven to be effective and safe as traditional surgical repair. However, procedure or device related complications can occur, which can be potentially fatal. We report a rare case of subacute and silent embolization of Amplatzer device to the pulmonary artery, on the next day of successful percutaneous ASD closure.

CASES
A 22-year-old male was admitted due to symptom of dyspnea developed several months before admission.

Figure 1: Large secundum atrial septal defect measuring 28 mm on transthoracic echocardiography (A). Transesophageal echocardiographic findings of sufficient superior and inferior vena caval rim (B) and preserved atrioventricular rim (C). Posterior rim was sufficient in length but relatively thin in nature, and aortic rim was nearly absent (D). RA: right atrium, LA: left atrium, IVC: inferior vena caval rim, SVC: superior vena caval rim, RV: right ventricle, AV: atrioventricular rim, SP: superoposterior rim, P: posterior rim, Ao: aortic rim.
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Transthoracic echocardiography (TTE) showed a secundum ASD measuring 28 mm anterior-posteriorly at apical 4 chamber view (figure 1A). Cardiac catheterization revealed pulmonary hypertension with pulmonary artery systolic pressure 42 mmhg and a large left to right shunt with a Qp/Qs 3.1. Transesophageal echocardiography (TEE) was performed and revealed a large secundum ASD measuring 30 mm, with sufficient superior vena cava (11 mm), inferior vena cava (14 mm) rim (figure 1B) and relative small atrioventricular rim (5 mm) (FIGURE 1C). Posterior rim was sufficient in length (13 mm) but relatively thin in nature, and aortic rim was nearly absent (Figure 1D). Despite relative large size of ASD with insufficient aortic rim, percutaneous device closure with Amplatzer was planned because she refused surgical treatment. During procedure, intracardiac echocardiography (ICE) was used for guiding intervention instead of TEE as our routine practice. ASD size measured by sizing balloon under fluoroscopy was 28 mm. An oversized 32 mm Amplatzer ASD device was selected because of insufficient aortic rim and deployed successfully after several failure of capturing atrioventricular rim. Prior to final release of the device, a secure and stable position of the device within the defect was checked by a push-pull maneuver and cessation of flow across the inter-atrial septum was confirmed by ICE and TTE (Figure 2).

FIGURE 2: Intracardiac echocardiographic still image during procedure (A). The Amplatzer device was successfully deployed and its secure and stable position was confirmed by push-pull maneuver. Inferior vena caval rim (white arrow) and superior vena caval rim (white arrowhead) were captured and well positioned between left and right discs. Still frame of apical 4 chamber view by transthoracic echocardiography after procedure (B). Amplatzer occluder (white arrow) is well positioned in interatrial septum without residual shunt. RA: right atrium, LA: left atrium

FIGURE 3 One day after the procedure, the embolized Amplatzer device was seen in pulmonary artery (white arrow) on chest X-ray (A), which was lodged on right pulmonary artery ostium (white arrow) on transthoracic echocardiography (B). MPA: main pulmonary artery, RPA: right pulmonary artery, Ao: aorta

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On the day following device closure, she was asymptomatic and routine follow up chest X-ray and TTE was performed. On chest X-ray, Amplatzer device shadow was found at the main pulmonary trunk area (Figure 3A) and TTE revealed reappearance of the large ASD with embolized Amplatzer device in the ostium of right pulmonary artery (FIGURE 3B). The right ventricular systolic pressure was increased to 65 mmHg, but she still remained asymptomatic and hemodynamically stable. We started intravenous infusion of heparin and asked for emergency surgery to remove the Amplatzer device and repair of the ASD. The operation was carried out with mediline sternotomy. After cardiopulmonary bypass, pulmonary artery was opened and the Amplatzer occlude was identified in the bifurcation site of pulmonary artery trunk (Figure 4). Amplatzer device slowly removed avoiding trauma to surrounding tissue (figure 5). Some of the marginal tissue in inferoposterial portion of the atrial septum was composed with friable membranous tissue. After removing the thin and friable tissue, the size of ASD was measured up to 30 × 40 mm. The ASD was closed using pericardial patch and he discharged from the hospital on the 7th postoperative day without other complications.

Figure 4: Amplatzer Device in MPA (main pulmonary artery)

Figure 5: Removed Amplatzer Device
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DISCUSSION

Atrial septal defect (ASD) is the prevalent congenital heart disease (3.78 per 10000 live birth). Surgical treatment is safe and effective but the complications relate to thoracotomy, bleeding, arrhythmia, post pericardectomy syndrome, and residual defects. There have been some creative efforts by interventional cardiologists in closing ASD using various devices. ASD closure device was first described by Ring et al., in 1976. The devices used for closure are composed of cardioseal, amplatz septal occluder (ASO), and the angeliwings. The amplatz septal occluder (ASO) is a device approved by FDA for transcatheter closure of secundum atrial septal defects and fenestrations post Fontan operation.

The ASO is a self-expandable, double disc device made from a Nitinol wire mesh. Nitinol is a metal alloy used in many medical appliances. The two discs are linked together by a short connecting waist corresponding to the size of ASD. In order to increase its closing ability, the discs and the waist are filled with polyester fabric.

Since the introduction in 1974, percutaneous device closure of secundum type ASD is increasing and became an alternative to surgical treatment (King and Mills, 1974). Complications of percutaneous ASD closure are air embolism, vascular trauma resulting from large sheathes, device embolization, clot embolization through AV valve, occlusion of pulmonary or systemic venous return, perforation of atrial septum, aortic perforation, infective endocarditis, atrial arrhythmia, device malposition necessitating its removal, delayed breakdown of device and residual atrial tear. Although immediate procedural success rate of Amplatzer septal occluder is 95-98%, adverse events including arrhythmia, cerebral embolism, cardiac tamponade and device embolization requiring immediate surgical removal can occur (Majunke et al., 2009). Among them, device embolization is a potential life threatening complication requiring immediate removal via percutaneous or surgical intervention. Although the reported incidence is 0.01-0.55%, it would be higher in less experienced operators (Majunke et al., 2009; Levi and Moore, 2004). The common reasons for the device embolization are undersized ASD device, small left atrium to accommodate the device, inadequate or floppy rim and operator-related technical issues. Most of the device embolization occurs during or several days after the procedures (Majunke et al., 2009). Immediate embolization occurs in the procedural field and thought to be caused by malposition or incorrect device size. Undersizing of the device is the most common reason for the embolization in such case (Levi and Moore, 2004). However, subacute embolization within several days of the procedure is thought to be associated in large part with aortic rim erosion or floppy septum (Misra et al., 2007).

In present case, nearly absent aortic rim and large defect size (30 mm) could be one reason for device migration. However, large ASD size and the small or deficient aortic rim itself is not a contraindication and often considered as suitable for device closure with Amplatzer occluder (Braga et al., 2004; Lin et al., 2003). Another important reason for the device migration is thin and floppy membranous nature of posterial portion of the atrial septum which was confirmed in operation field. Combination of small aortic rim and floppy membranous nature of counterpart rim (inferoposterior rim) increased the instability of oversized Amplatzer device and may lead to migration and embolization of device in our patient.

In case of complicated ASD, as our present case, it is often difficult to reconstruct the spatial structure with the use of two dimensional (2D) images. In this occasion, 2D TEE or balloon sizing may not be sufficient and additional imaging modalities such as cardiac multiddetector computed tomography or three dimensional TEE can be used complementary option to 2D TEE for morphologic evaluation of ASD and guidance of transcatheter closure (Kijima et al., 2010; Ko et al., 2009).

About 50% of cases with Amplatzer occluder embolization, percutaneous retrieval is possible by using the devices including large sheaths, gooseneck snares, or endomyocardial biopsy forcep (Balbi et al., 2008). However, surgical removal and repair of the ASD is more preferable in the situation of inappropriate ASD rims for the second procedure as present case.

Conclusion

Application of the strict criteria for selecting the device closure by comprehensive evaluation of ASD. Careful monitoring for the possible delayed embolization of device are mandatory in the case of complicated ASD.
Device embolization is an uncommon complication of Amplatzer ASD closure, that can be managed surgically or by percutaneous extraction methods in experienced centre.

REFERENCES


