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INTRODUCTION

Atrial septal defect (ASD) is one of the most common congenital heart defects (1). Secundum ASD accounts for 80% of ASDs. It is located in the area in and around fossa ovalis. This defect is in the fossa ovalis area and occurs as a result of excessive fenestration or resorption of the septum primum, failure of the septum secundum to develop, or a combination of the 2 conditions (2). Although open heart surgery is considered to be the classical and reliable treatment method for ASD, percutaneous ASD closure has recently been superior to surgery in appropriate cases due to its easy applicability, low complication rate, low cost, short hospital stay, less interventional, and successful long-term results. Surgical and non-surgical closure of this septal defect is possible, provided that certain indications and criteria are met for each procedure (3). Embolization, among the complications, is the most common reason for surgical intervention. We report a case of successful surgical removal of an atrial septal occluder device embolized to the left atrium 3 weeks after percutaneous intervention. In our opinion, what made this case interesting

Does every ASD closure device stay in the atrial septum?

Abstract

Atrial septal defect (ASD) is one of the most common congenital heart defects and the most common type is secundum type ASD. There are 2 approaches to its treatment: open surgical repair and percutaneous intervention. In this article, we describe the complication and intervention performed on a patient whose ASD was repaired percutaneously. In our opinion, what made this case interesting was that a complex case was handled by cardiology without a Cardiology-Cardiovascular Surgery council.

Keywords: Atrial septal defect, complication of percutaneous repair, closure device

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CASE

A 56-year-old male patient was admitted to the cardiology outpatient clinic with complaints of fatigue. He had no known comorbidities. Transthoracic Echocardiography (TTE) revealed moderate mitral regurgitation (MR), severe tricuspid regurgitation (TR), pulmonary Hypertension, and a 26 mm septal defect in the interatrial septum (IAS). Transesophageal Echocardiography (TEE) performed for ASD revealed a 29x32x26 mm secundum type ASD with a left-to-right shunt in the IAS. Coronary angiography revealed 90% stenosis in the mid-proximal LAD. The patient was decided by the cardiology to have his ASD closed percutaneous, elective Percutaneous Coronary Intervention (PCI) of the stenosis in the LAD 3 weeks after the procedure and to follow up his TR and MR with medical treatment since he was not symptomatic. ASD was closed with a 39 mm septal occluder device (Figure 1). The next day, control TEE was performed and the patient was sent home for PCI with clopidogrel 3 weeks later.

1 month later during the PCI procedure, it was noticed that the IAS occluder device was moving during the fluoroscopy (Figure 2) and Emergency TTE was performed and it was seen that the device was in the left atrium. The patient was consulted to cardiovascular surgery department urgently.



Figure 1. ASD occluder seen on coroner angiography

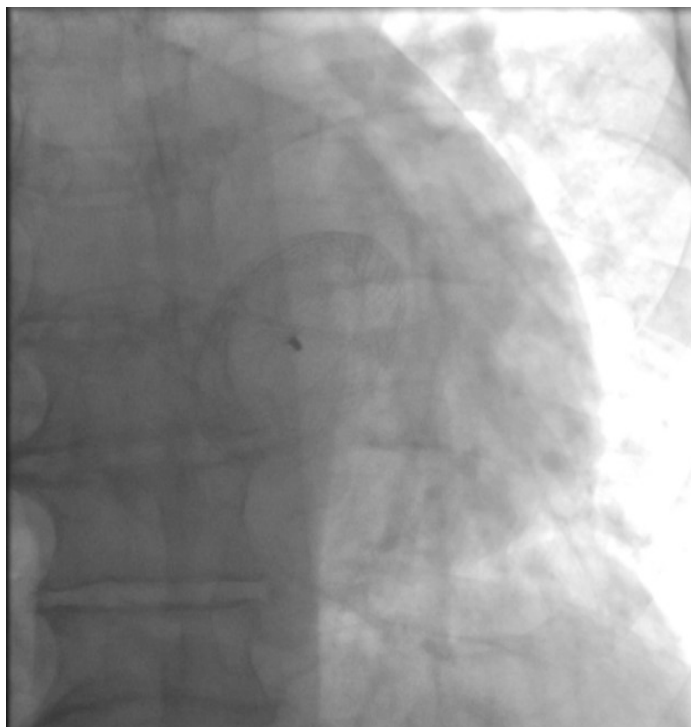


Figure 2. ASD occluder seen on angiography

The patient was operated on urgently. A median sternotomy was performed under general anesthesia. Left internal mammary artery (LIMA) and saphenous graft was prepared. The pericardium was opened. Aorta bicaval cannulation was performed. A cardiopulmonary bypass was performed. Cross clamping was performed. The diastolic cardiac arrest was achieved with antegrade cardioplegia. Right atriotomy was performed. Secundum type cribriform ASD in the IAS and the occluder device was present in the left atrium. The occluder device was removed (Figures 3-5). IAS was closed with a pericardial patch. The right atriotomy was closed. Ao-LAD bypass was performed using a saphenous graft because the Lima flow was not sufficient. The cross-clamp was removed. Cardiopulmonary bypass was exited with dopamine support. The patient was intubated with ventilator support and admitted to the PVC intensive care unit. The patient was transferred to the ward without any complications during follow-up.



Figure 3. ASD occluder device



Figure 4. ASD occluder device



Figure 5. ASD occluder device

DISCUSSION

Complications such as device migration/embolization, cardiac erosion, perforation, arrhythmias, thrombosis, sepsis, etc. may occur in percutaneous closure. The incidence of occluded device embolization after ASD closure is 0.55%-1.4% (4). Reported sites of embolization include the right ventricle, RVOT, pulmonary artery, left atrium, LVOT, aortic arch, descending thoracic aorta, abdominal aorta, and common iliac artery (5). In our case, the atrial septal occluder device was embolized to the left atrium. Boysan et al., in a case they presented, attributed the cause of the ASD closure device embolized to the pulmonary artery to cough (6). Depending on the area of embolism and the complications that develop, various symptoms (cough, shortness of breath, hypotension, etc.) may occur. In our case, there was no symptom to explain the embolization.

Epithelialization is expected to occur within 2 weeks after the percutaneous closure of ASD. The ASD closure device we removed from the patient also had epithelial tissue. Patients are discharged after control echocardiography 24 hours after the procedure and are called for controls at 1, 3, 6 and 12 months. In our patient, control TEE was taken at 24 hours and it was found to be normal. When she was called for PCI 3 weeks later, she had been processed without echocardiography.

Common causes of embolization include very large defects, undersized ASD devices, a small left atrium for device placement, insufficient margin of the ASD, and technical problems. In patients with large ASD defects (> 38 mm) with insufficient margins, transcatheter closure is not considered appropriate and surgical closure is appropriate. In our case, the defect size was 29x32x26mm. The mobility of the device after implantation and an aortic margin narrower than 5 mm also increase the risk of early and late embolization (7).

Retrieval of the ASD device can be performed by both percutaneous and surgical approaches. In this case, surgical retrieval of the device was performed because the occluder device was free in the left atrium and there was a possibility of thrombosis and endothelialization of the left atrial wall and a lesion in the LAD. The ASD was closed with a pericardial patch and a LAD-Ao bypass was performed. Simple Transthoracic

Echocardiographic evaluation in the early follow-up period can confirm the position of the device.

CONCLUSION

Permanent closure of ASD is nowadays commonly performed by percutaneous intervention. Percutaneous ASD closure offers many advantages such as easy applicability, low complication rate, low cost, and short hospital stay, but there are some serious complications such as embolization. This requires careful monitoring of patients and more careful selection of suitable patients to avoid complications. Surgical closure of cribriform ASDs would be more appropriate.

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REFERENCES

1. Smilowitz NR, Subashchandran V, Berger JS. Atrial septal defect and the risk of ischemic stroke in the perioperative period of noncardiac surgery. *Am J Cardiol.* 2019;124:1120-4.
2. Yong G, Khairy P, De Guise P, Dore A, Marcotte F, Mercier LA, et al. Pulmonary arterial hypertension in patients with transcatheter closure of secundum atrial septal defects: a longitudinal study. *Circ Cardiovasc Interv.* 2009;2:455-62.
3. King TD, Thompson SL, Steiner C, Mills NL. Secundum atrial septal defect. Nonoperative closure during cardiac catheterization. *JAMA.* 1976;235:2506-9.
4. Amanullah MM, Siddiqui MT, Khan MZ, Atiq MA. Surgical rescue of embolized amplatzer devices. *J Card Surg.* 2011;26:254-8.
5. Lysitsas DN, Wrigley B, Banerjee P, Glennon PE, Parmar JM, Shiu MF, Been M. Presentation of an embolised Amplatzer septal occluder to the main pulmonary artery 2 years after implantation. *Int J Cardiol.* 2009;131:e106-7.
6. Boysan E, Cicek OF, Cicek MC, Hamurcu Z, Gurkahraman S. Surgical removal of an atrial septal occluder device embolized to the main pulmonary artery. *Tex Heart Inst J.* 2014;41:91-3.
7. Sahin DY, Koç M, Cakır H, Arık OZ, Elbasan Z, Caylı M. A silent and late embolization of atrial septal defect occluder device into the right pulmonary artery: a case report. *Korean Circ J.* 2012;42:781-3.