Late embolization of an atrial septal defect closure device into the main pulmonary artery

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Introduction

Atrial septal defect (ASD) is known to be the most widespread type of congenital heart disease in adulthood (1). Ostium secundum type is the most common form of ASD (60%-70%). Percutaneous closure of ASD is known as an alternative treatment to surgery. Percutaneous transcatheter closure of ASD provides many benefits compared with surgery, such as decreased surgical morbidity, no scarring, and shortened hospital stay. Nowadays, transcatheter treatment is the first choice in secundum ASD treatment. Various devices have been developed for this purpose, and the Amplatzer septal occluder is the most commonly used. However, this closure method is associated with rare complications observed in the early and late stages. The frequency of device embolization in experienced centers during ASD closure is less than 1%. Embolization usually occurs in the early postoperative period and in the right heart cavities. In the acute period, if embolization into the right ventricular outflow tract or pulmonary artery branches occurs, a snare catheter may be used for the attempted recovery. Late dislocation and embolization of the ASD occluder device are rarely reported in the literature. In this case report, we report silent and late embolization of the ASD occluder device into the main pulmonary artery.

Case Report

A 47-year-old male patient underwent successful transcatheter ASD closure with the 30-mm Amplatzer septal occluder device on January 24, 2012, due to secundum ASD. He did not have any residual shunt, and no problem was detected on echocardiography during the first 4 postoperative years. When the report of the last echocardiography performed in 2017 was examined, the interatrial septum was observed to be intact. When control transthoracic echocardiography was performed on December 20, 2018, 7 years after percutaneous ASD closure, shunt flow from the left atrium to the right atrium was detected. The device was not seen in the interatrial septum and was thought to be embolized (Fig. 1).

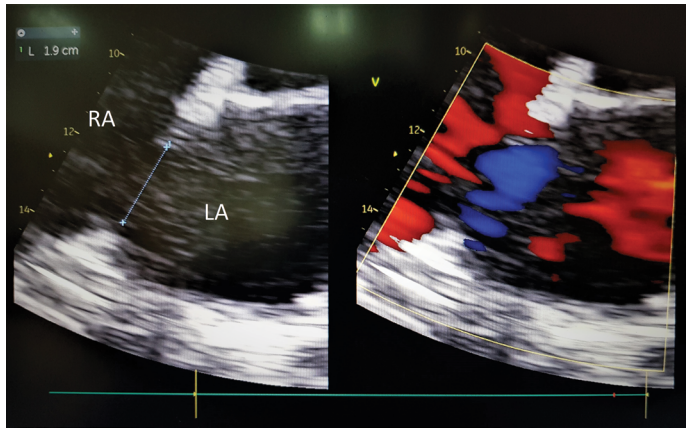

Figure 1. The device was not seen on the interatrial septum and was thought to be embolized

An ASD occluder device with a diameter of 30 mm was observed to be adhered to the inner wall of the artery 2 cm above the pulmonary valve, parallel to the longitudinal axis of the main pulmonary artery (Fig. 2).

Pulmonary flow in this region was slightly turbulent, and the peak gradient was calculated as 16 mm Hg. The patient's right heart chambers were enlarged, and pulmonary artery pressure was increased (46 mm Hg). Pulmonary posteroanterior radiography showed the ASD occluder device in the pulmonary artery (Fig. 3).

The patient had no symptoms, such as dyspnea or chest discomfort, associated with the dislocation of the ASD occluder device. Coronary angiography and right heart catheterization were performed, and the coronary arteries were normal. In addition, the appearance of the occluder during coronary angiography was observed in the main pulmonary artery (Fig. 4).

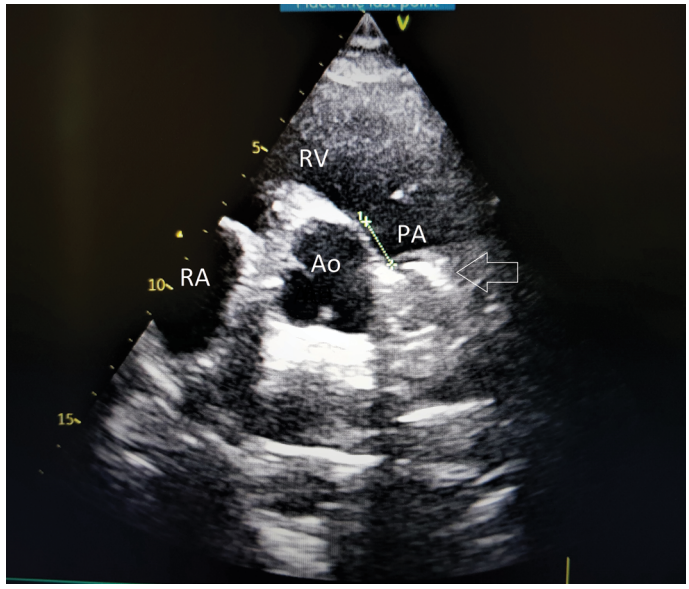

Figure 2. An ASD occluder was observed 2 cm above the pulmonary valve

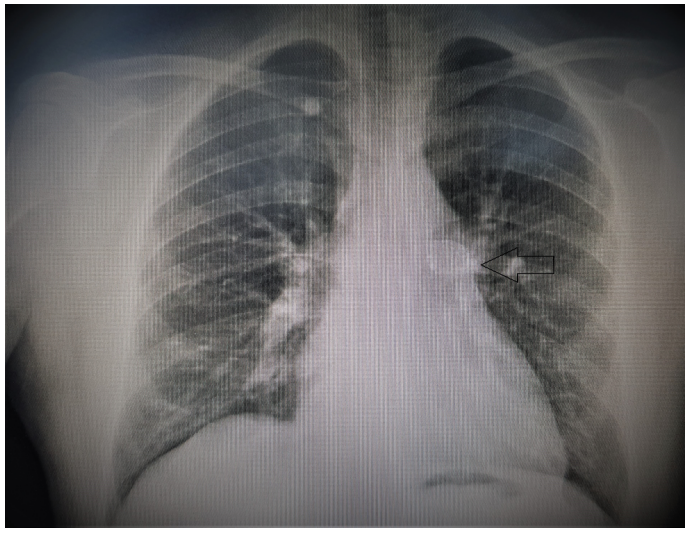

Figure 3. Pulmonary posteroanterior radiography showed the ASD occluder device

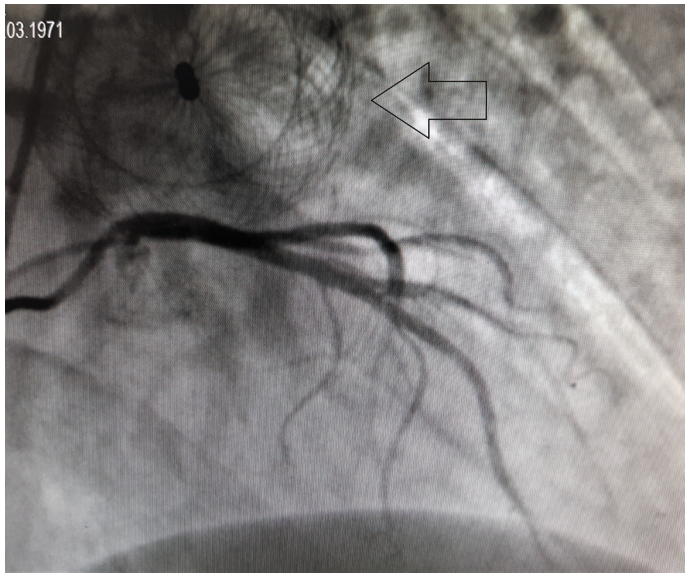

Figure 4. Coronary angiography and right heart catheterization

Right heart catheterization was performed, from which the Qp/Qs ratio was determined to be 1.7. It was decided that an operation should be performed, and the patient subsequently underwent surgical closure of the secundum ASD with a pericardial patch and removal of the ASD occluder device. After surgical removal, the device was observed to be macroscopically intact (Fig. 5). Postoperative follow-up was uneventful. In addition, follow-up echocardiography showed no leakage from the atrial septum.

Discussion

The most preferred treatment in any clinical situation is the surgical closure of ASD. An alternative to surgical closure of ASD is percutaneous closure, which is associated with

Figure 5. After surgical removal, the device was macroscopically intact

reduced morbidity, shorter hospitalization, no scarring, and comparable complication rates (2). On the other hand, percutaneous ASD closure is affiliated with few complications in the early and late stages, including device transfer or embolization, pericardial effusion, arrhythmia, occluder thrombus formation, and vascular damage (3). Device embolization, which has an incidence of 4% to 21%, is the most widespread complication of percutaneous closure of ASD (4). Embolization often develops during the first 24 hours and is rarely seen after this period. Factors associated with device embolization are linked to the type of device used, the width of the defect, a thin atrial tissue rim, increased mobility of the occluder after device implantation, the use of a device that is smaller than the defect, and shortness or absence of the aortic rim (5, 6). As seen in this case, dislocation and embolization can be detected even after years of percutaneous ASD closure.

An aortic rim <5 mm is one of the most important causes of early and late embolization (7). The presence of a large ASD in our patient and an aortic rim of 4 mm might have caused the dislocation in the late period. An acute change in intracardiac pressure as a result of physical strain is another potential cause of late device embolism. An abrupt rise in afterload toward the left heart together with decreased right heart filling might have led to the movement of the device toward the right and afterward into the main pulmonary artery (8). The embolization of the device into the pulmonary circulation and the impediment of pulmonary flow might result in an acute amount of volume and strain overload of the right ventricle. Constitutional symptoms

generally develop depending on the degree of the pulmonary flow impairment (7). Our patient was asymptomatic because the tool was located on the longitudinal axis in the main pulmonary artery and did not affect pulmonary flow. Because of the embolization of the device, percutaneous or surgical removal was indicated (3). In our case, percutaneous removal was not an option because of the chronic embolization of the device in the main pulmonary artery. Thus, we recommended surgery for our patient.

Conclusion

Device embolization, the most common complication of percutaneous closure of ASD, can occur in the late postoperative period. Different clinical presentations are possible depending on the location where the device is embolized. Rarely, patients may be asymptomatic. Individuals with a high risk of embolization should be followed up more frequently and for a longer time using echocardiography.

Informed consent: The informed consent was obtained from the patient.

References

1. Clark EB. Epidemiology of congenital cardiovascular malformations. In: Emmanouilides GC, Riemenschneider TA, Allen HD, et al., editors. Heart disease infants, children, and adolescent including the fetus and young adult. 5th ed. Baltimore: Williams & Wilkins; 1995. pp. 60–70.
2. Knepp MD, Rocchini AP, Lloyd TR, Aiyagari RM. Long-term follow up of secundum atrial septal defect closure with the Amplatzer septal occluder. Congenital Heart Dis 2010; 5: 32–7. [CrossRef]
3. Chessa M, Carminati M, Butera G, Bini RM, Drago M, Rosti L, et al. Early and late complications associated with transcatheter occlusion of secundum atrial septal defect. J Am Coll Cardiol 2002; 39: 1061–5. [CrossRef]
4. Chiappini B, Gregorini R, Di Eusanio M, Ciocca M, Villani C, Minuti U, et al. Embolization of an Amplatzer atrial septal closure device to the pulmonary artery. J Card Surg 2008; 23: 164–7. [CrossRef]
5. Berdat PA, Chatterjee T, Pfammatter JP, Windecker S, Meier B, Carrel T. Surgical management of complications after transcatheter closure of an atrial septal defect or patent foramen ovale. J Thorac Cardiovasc Surg 2000; 120: 1034–9. [CrossRef]
6. Sinha A, Nanda NC, Khanna D, Dod HS, Vengala S, Mehmood F, et al. Morphological assessment of left ventricular thrombus by live three-dimensional transthoracic echocardiography. Echocardiography 2004; 21: 649-55. [CrossRef]
7. Misra M, Sadiq A, Namboodiri N, Karunakaran J. The 'aortic rim' recount: embolization of interatrial septal occluder into the main pulmonary artery bifurcation after atrial septal defect closure. Interact Cardiovasc Thorac Surg 2007; 6: 384–6. [CrossRef]
8. Mashman WE, King SB, Jacobs WC, Ballard WL. Two cases of late embolization of Amplatzer septal occluder devices to the pulmonary artery following closure of secundum atrial septal defects. Catheter Cardiovasc Interv 2005; 65: 588–92. [CrossRef]

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Fatal anaphylactic reaction due to ferric carboxymaltose: A case report

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Introduction

Iron-deficiency anemia is most frequently related with insufficient nutrition. However, it is also encountered in chronic diseases including chronic renal failure (CRF) and congestive heart failure (CHF). Oral or intravenous (IV) iron replacement is recommended for its treatment (1, 2). IV administration is the preferred route in CRF and CHF, where intestinal mucosal edema and diminished gastrointestinal blood flow limit absorption of oral iron. Although IV route may cause hypersensitivity reaction (HSR), it is generally safe. The report of the European Medicines Agency declares that IV-iron treatment has a high benefit/risk ratio (3).

Ferric carboxymaltose (FCM) is a nondextran third-generation IV-iron preparation which has the advantage of normalizing hemoglobin and replenishing iron stores over a short period of time because it can be administered fast and in high doses (4). It has been approved and presented to the market in 2007 in Europe and in 2012 in Turkey (5). Clinical research has shown that it is generally well tolerated and has low serious HSR risk (≥1/10.000 to <1/1.000) (1, 4, 6, 7). Evaluation of safety reports discloses that there are four cases of death related to FCM (5, 8). To our knowledge, this is the first case of death related to FCM in Turkey and the fifth case of fatal anaphylactic reaction (AR) in the literature.

Case Report

A 74-year-old male patient who had a history of diabetes, hypertension, CRF, and coronary artery disease presented to the emergency service of our hospital with shortness of breath. On admission, since he had hypertension (190/110 mm Hg) and lung