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Case Report

Late Management of An Atrial Septal Defect Closure Device Embolized to Left Ventricle Outflow Tract

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Abstract

Atrial septal defect (ASD) which is among most common congenital heart disease diagnosed in adults. It is commonly managed with percutaneous closure successfully. However, percutaneous closure procedure has been associated with some rare complications including embolization. Embolization of the device into to the main pulmonary artery, left atrium, right ventricle, aortic arch, descending aorta, abdominal aorta, iliac bifurcation, and iliac arteries have been reported. Embolizations usually occur during procedure and rarely several hours or a few days after intervention. However, as far as we know, a silent embolization to left ventricle outflow (LVOT) for 13 years has not been reported previously. Here, we report a case of atrial septal defect closure device which is embolized to left ventricle outflow having index procedure 13 years before. The device was successfully retrieved surgically and ASD was repaired.

Keywords

Atrial septal defect, Closure device, Embolization, Left ventricle outflow, Eustachian valv

Article History

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1. Introduction

Atrial septal defect (ASD) is the most common congenital heart disease during adulthood following bicuspid aortic valve. Due to its silent course or subtle clinical symptoms it may be unrecognized till late adulthood. Chronic long term right sided cardiac volume overload may lead to atrial arrhythmias, pulmonary hypertension and right ventricular failure. Patients may become symptomatic at their late decades and the overall survival is decreased. Secundum type ASD is the most common form of ASD. Majority of uncomplicated secundum ASD's are managed with percutaneous device closure. Although the procedure is successfully performed in most patients with a low complication rate sometimes serious complications may occur including pericardial effusion, tamponade, embolization and residual flow [1-3]. Embolized devices usually retrieved percutaneously and, in case of failure, surgical extraction is necessary. Embolization of the device into to the main pulmonary artery, left atrium, right ventricle, aortic arch, descending aorta, abdominal aorta, iliac bifurcation, and iliac arteries have been reported. Embolizations usually occur during procedure and rarely several hours or a few days after intervention. Here, we present a case of ASD closure device embolization to left ventricular outflow tract (LVOT) which remained silent for 13 years after percutaneous closure intervention. Based on our device size measurements and transesophageal echocardiography (TEE) findings we retrospectively speculate that an ASD closure device was tried to be implanted in a large ASD having poor posterior rim and Eustachian valve (EV) might have been misinterpreted as a superior part of atrial septum.

2. Clinical Course

76 years old female, having history of percutaneous ASD closure presented to emergency department with shortness of breath and palpitation on minimal exertion. She described progressively increased dyspnea on exertion for the last 6 months. Blood pressure was 110/50 mmHg and pulse rate was 120 bpm. Electrocardiography showed atrial fibrillation. On physical examination, there were fine crackles in the basal and middle zones of the lungs on auscultation and there was no pretibial edema. Apart from a mild elevation in C-reactive protein level, blood chemistry and hemogram parameters were normal. There was cardiomegaly on chest X-ray. Upon cardiology consultation transthoracic echocardiographic examination revealed normal left ventricular systolic function, biatrial and right ventricular dilatation (parasternal left atrium size was 51 mm), an ASD closure device at LVOT, mild aortic stenosis with 16/10 mmHg gradient, mild aortic and moderate mitral regurgitations and severe tricuspid regurgitation with estimated pulmonary pressure (PAP) of 40 mmHg (Figure 1A).

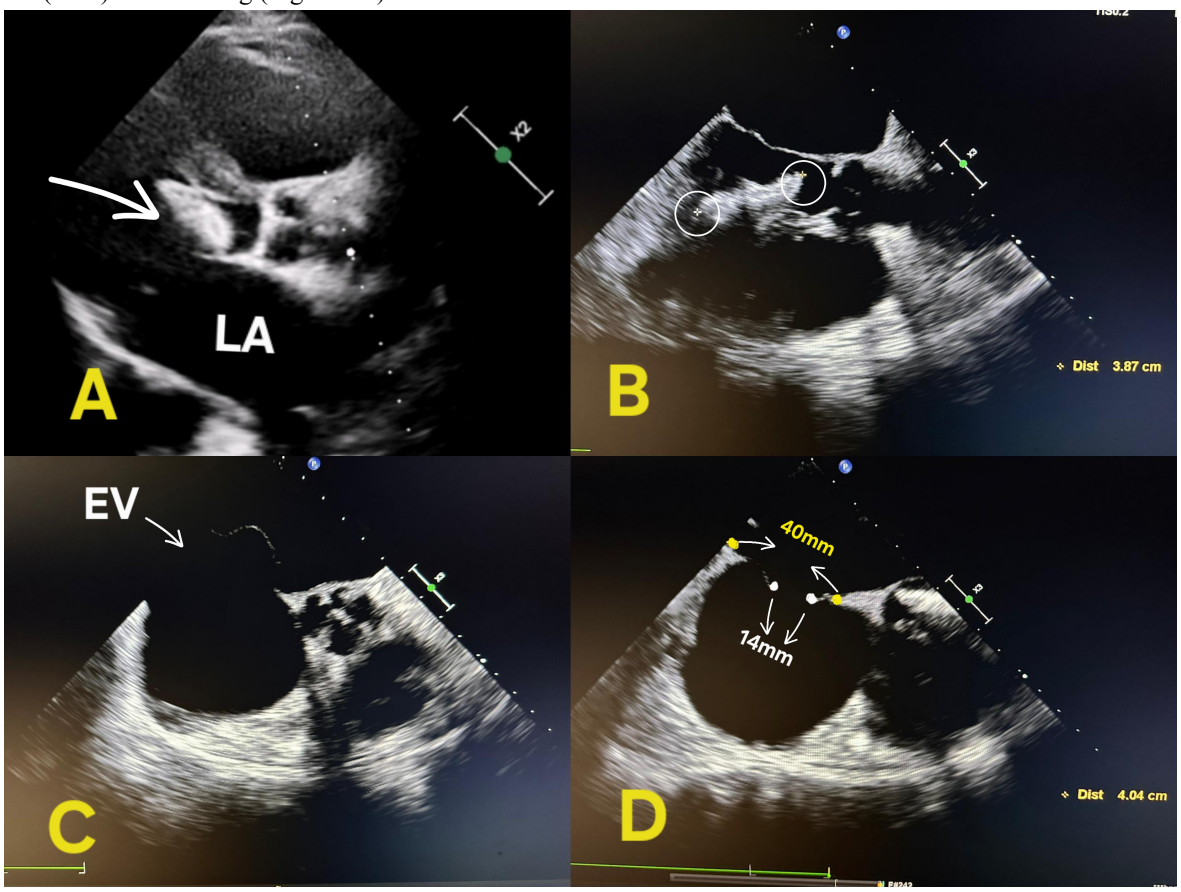

Figure 1. Preoperative transthoracic (A) and transesophageal echocardiographic images of 38 mm measured embolized device (arrow) in left ventricular outflow (B), long flail EV (arrow) bulging to left atrium (C) seems like superior atrial septum that may cause misinterpretation of defect size as 14 mm instead of 40 mm where there's no posterior rim (D). LA = left atrium; RA = right atrium; EV= Eustachian valve.

TEE revealed that the previously embolized ASD closure device was located at LVOT and under anterior mitral valve leaflet. The length of ASD closure device was 38 mm on TEE imaging (Figure 1B). There was a large secundum type ASD accompanied by an aortic rim and absent posterior rim. There was a long flail thread like structure bulging to left atrium and coming back to ASD line which we evaluated as a prominent Eustachian valve (EV). EV might have been potentially evaluated as superior part of aneuyrsmatic interatrial septum and defect may be measured as 14 mm instead of 40 mm (Figure 1C, D).

We learnt that the patient had undergone percutaneous ASD closure in 2012. However, the procedural records and implantation documents were not available. She didn't follow cardiology outpatient clinic since she was asymptomatic. In 2017, following a fall, the patient required an emergent orthopedic surgery. During the preoperative evaluation ASD closure device was detected to be embolized to left ventricle and surgical extraction was offered after orthopedic operation. However, she declined cardiac surgery. Now, the patient accepted the surgical recommendation. Preoperative coronary angiography revealed no significant coronary artery disease (Figure 2).

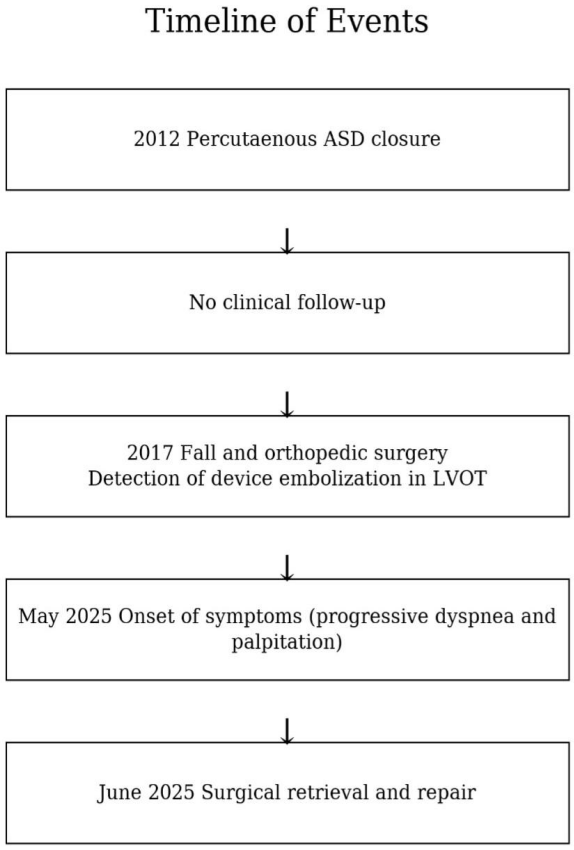

Figure 2. Timeline of events.

The operation was performed under general anesthesia through standard median sternotomy with bicaval and aortic cannulation. Unfractionated heparin at a dose of 300–400 IU/kg was administered intravenously. The pericardium was prepared by treating it with 2% glutaraldehyde. When the targeted activated clotting time value was reached, cardiopulmonary bypass was initiated. After aortic cross clamping and achievement of cardioplegia the right atrium was opened. A 60×40 mm ostium secundum ASD was identified. The migrated ASD occluder device was lodged in the LVOT and was located behind the anterior leaflet of the mitral valve. A transverse aortotomy was performed. The occluder device was still in the LVOT. The occluder device was intertwined with the mitral chordae and was adherent to the interventricular septum and left atrial structures. The device was removed using blunt and sharp dissections without damaging the surrounding tissues (Figure 3). The aortic wall was closed with a simple primary suture. Mitral valve insufficiency was assessed using saline through the ASD and was judged to be severe. Mitral annuloplasty was performed using a s 30 mm sized semi-rigid Carpentier ring. The ASD was closed with the prepared pericardial patch. Cardiopulmonary bypass was terminated after closing the right atrium. The patient remained in the intensive care unit for three days postoperatively and was discharged without any postoperative complications on postoperative day 10.

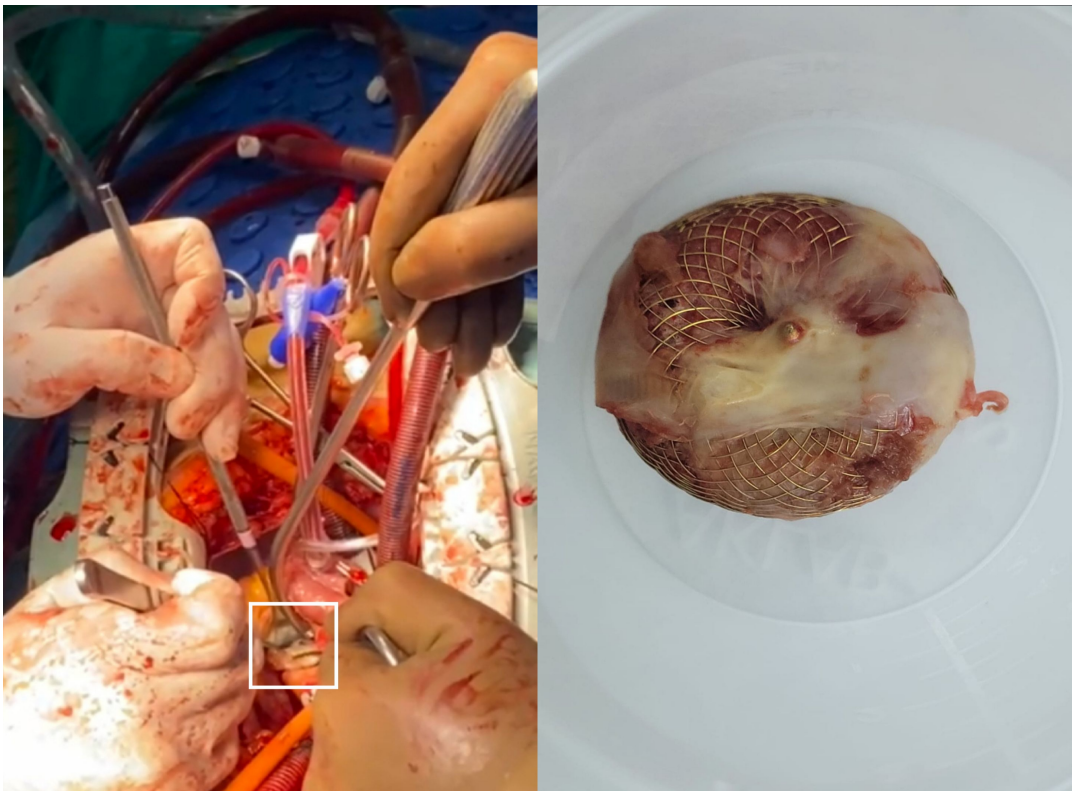

Figure 3. (A) Removal of the device with a transverse incision of ascending aorta. (B) Macroscopic image of the removed device.

One month after operation she was asymptomatic and transthoracic echocardiographic examination showed normal left ventricular systolic function, left atrium size was decreased to 43 mm, there was mild mitral and tricuspid regurgitations with PAP of 20 mmHg. (Figure 4)

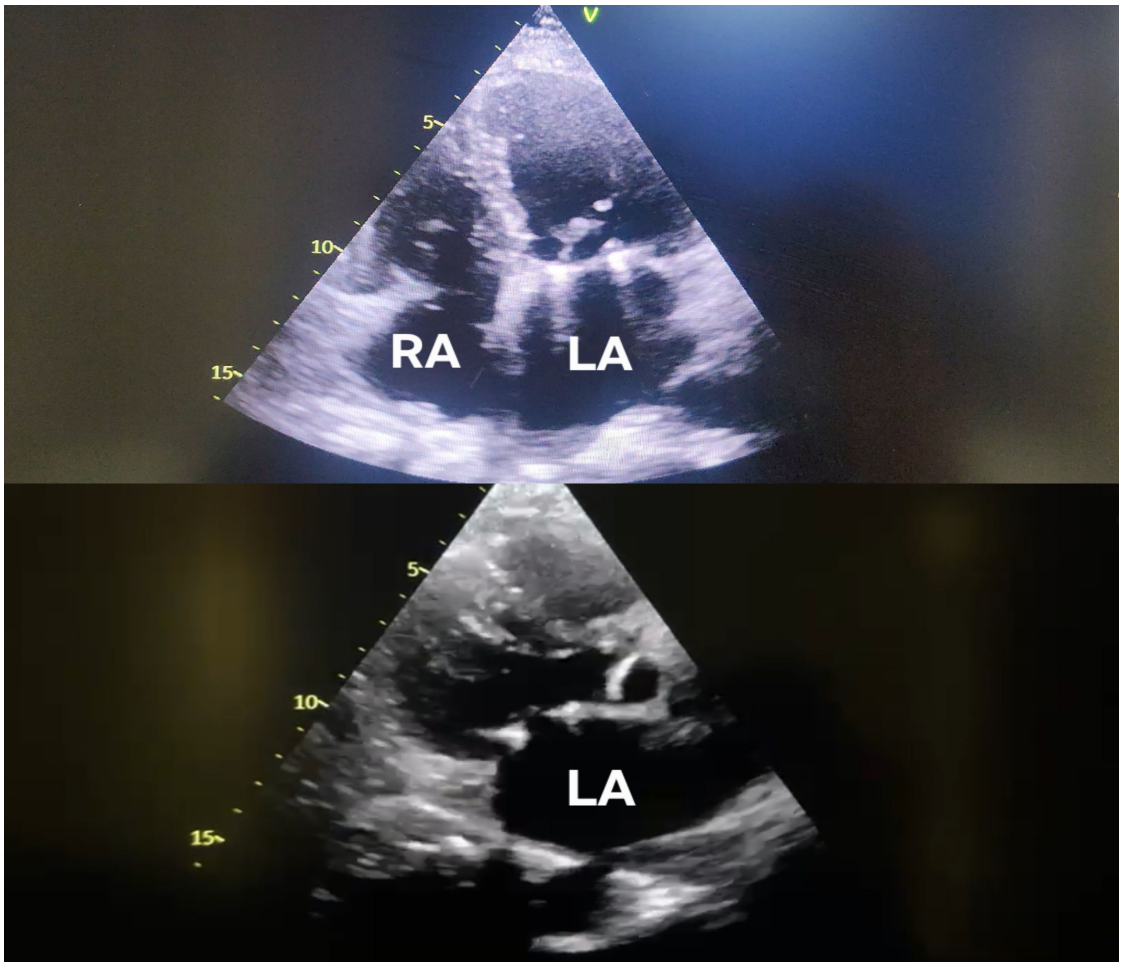

Figure 4. Transthoracic echocardiographic images one month after operation. LA = Left atrium; RA = Right atrium.

3. Discussion

3.1 A Brief Overview of Secundum Type Atrial Septal Defect

Secundum type ASD is among the most commonly encountered congenital heart disease during adulthood. Patients usually remain asymptomatic. Symptoms usually develop after forties including reduced functional capacity, dyspnea on exertion and palpitations. With increasing age, PAP elevates secondary to chronic volume overload and arrhythmias become more prevalent and rarely right heart failure develops.

The complications associated with ASDs include atrial dysrhythmias, pulmonary arterial hypertension, right-sided heart failure and transient ischemic attack or stroke related to atrial fibrillation and rarely to paradoxical emboli, and Eisenmenger syndrome. Prevention of complications associated with ASD is directly related with early diagnosis and management of significant defects.

The course of ASD may be silent or may be associated with subtle symptoms in many patients. Therefore, symptoms may not guide diagnosis and therapy. Advancements in the technology of echocardiography and improvements in availability and accessibility has resulted in earlier diagnosis of ASD and other structural heart diseases. ASDs smaller than 5 mm usually close during first year of life. Defects larger than 10 mm usually require intervention for closure. Adult patients with small defects and no signs of right heart failure should be monitored. An echocardiographic evaluation for right heart function and dilatation and PAP every 2 to 3 years is appropriate. ASD should be suspected in patients with right chamber dilatation. In adults, TEE is of paramount importance for establishment of the diagnosis and for assessment appropriateness for percutaneous closure device selection and avoidance of interventional complications [1-3]. Atrial rims and surrounding structures including EV should be carefully evaluated with various projections during TEE. Agitated saline or contrast aided echocardiographic assessment especially helpful for differential diagnosis. If possible three dimensional TEE has incremental value in evaluation of defect and right sided structures [4,5].

3.2 Functional Capacity and ASD Closure

Surgical or percutaneous ASD closure has been recommended to improve mortality and morbidity, and had a protective effect on functional capacity as well. In their systematic review and meta-analysis of adult patients having surgical or percutaneous closure of ASD, Oster et al. reported that being in NYHA class I was 14 times higher after ASD closure [3].

Increased pulmonary flow will eventually increase pulmonary hypertension and impair left ventricular compliance by time. Therefore, with aging functional capacity deteriorates progressively in ASD patients. In the case we presented here, the patient had remained asymptomatic for decades till seventies but had a rapid progressive decline in functional capacity during last six months. ASD closure has been associated with improved functional capacity even in asymptomatic patients after closure of ASD after > 40 years of age. The functional capacity of the presented case also improved at short term after surgical repair of ASD.

3.3 Timing of ASD Closure

To improve mortality and morbidity surgical or percutaneous ASD closure has been recommended in adults with ventricular volume overload if pulmonary vascular resistance is reasonable. However, compared to age- and sex-matched control population survival may be reduced in even patients with surgically repaired ASDs when surgery is performed after age of 25 years [3]. Long term complications especially atrial arrhythmias including fibrillation that are associated with systemic embolization and stroke may be prevented with closure of ASDs at early ages. Among patients having ASD closure after 40 years of age the prevalence of atrial arrhythmias is around 40-60% [3]. Although the ASD closure after age of 40 years doesn't appear to affect arrhythmia frequency, the symptoms and functional capacity of patients usually improves with ASD closure at any age. Furthermore, as a consequence of decreased volume overload, reductions in cardiac chamber dilatations may be seen in short a time and improvement continues during long term [1-3]. Accordingly, in our case, PAP and atrial volumes were decreased one month after surgery.

In cases with hemodynamically insignificant too small defects closure is not necessary. In cases with severe pulmonary hypertension with increased pulmonary vascular resistance and also in patients with left ventricular systolic dysfunction, closure may be associated with worse outcome since the defect serves as a left to right shunt aiding in avoidance of pulmonary congestion. Therefore, co-associated pathologies like pulmonary venous return anomalies and increased pulmonary vascular resistance should be carefully excluded when closure is planned. In patients with severe pulmonary hypertension, complete defect closure may lead to hemodynamic challenges and fenestrated percutaneous closure devices or fenestration of the patch during surgery may be required. In advanced ages, if ASD is not suitable for percutaneous closure, patients should be individually assessed whether potential benefits outweigh risks of surgical intervention [1-3].

3.4 Surgical vs. Percutaneous Closure of ASD

Surgical repair has low mortality [<1%] and long-term safety. However, percutaneous closure is a more cost-effective and comfortable method compared to surgery. The post procedural complication risk for percutaneous ASD closure is 7.2%, whereas risk for surgical complications is 24% [1]. Therefore, percutaneous device closure has become the first choice of treatment modality due to its safety, simplicity and availability [1-3]. Mortality and success rates of surgical and percutaneous closure of secundum ASD seems similar. However, comfort of patients is higher and hospital stay is shorter with percutaneous closure. Percutaneous closure of secundum type ASD may be performed with local anesthesia or light sedation with guidance of transthoracic echocardiography or with general anesthesia with guidance of trans esophageal echocardiography. Majority of patients are discharged the day after procedure or even the same day. Long-term results have established the safety of procedure.

For surgical repair of secundum ASD, a median sternotomy allows access to harvest autologous pericardium for patch closure, ensuring a tension-free repair. Cardiopulmonary bypass is initiated, and diastolic arrest is achieved with cardioplegia. Surrounding structures like the tricuspid valve, coronary sinus, and atrioventricular node shouldn't be compromised during operation; accurate assessment of localization and correction of defect is mandatory. Minimally invasive techniques using smaller incisions and specialized cannulation techniques, such as transxyphoid, ministernotomy, and transaxillary approaches, aim to reduce surgical trauma and improve cosmetic outcomes. While effective access for defect repair is maintained, risks related with chest closure are minimized. Robot-assisted and thoracoscopic approaches offer even less invasive alternatives [1,6]. Although minimally invasive surgery techniques and video-assisted thoracoscopic ASD closure are becoming more popular, still percutaneous closure is less invasive, has shorter hospital stay and better recovery time compared to surgical repair methods. Therefore, compared to surgical repair, percutaneous device closure is the current treatment of choice for secundum ASD in appropriate cases (i.e. stretched diameter <38 mm, sufficient rim with > 5 mm except towards aorta) [1-3].

3.5 Complications Related with Percutaneous ASD Closure

Percutaneous closure of ASD has been associated with early and late complications including air embolism, clot embolization, device malposition necessitating retrieval, migration or embolization of the device, delayed breakdown of device, vascular complications, pericardial effusion, aortic perforation, atrial arrhythmias, residual shunts, late erosion and thrombus formation on the device and infective endocarditis. The size of the defect and adequacy of the rims are important for device size selection. Large sized devices may be associated with erosion and increased arrhythmia [1-3,7-9].

3.6 ASD Closure Device Embolization

The rate of serious complications related to percutaneous ASD closure is <1% and embolization of ASD device was reported to be about 0.5% [1-3,7-9]. Inadequate or floppy rims, large defects and under- or over-sized devices and incorrect placement of the device may be associated with embolization. The frequency of this complication would be higher in less experienced operators and insufficient cardiac imaging backup and intraprocedural echocardiographic guidance. Embolization may be silent or may be associated with dyspnea, tachycardia, coughing, palpitation with ventricular premature beat or chest discomfort [9].

In their retrospective study Garre et al. have found 30 (1.3%) device embolizations among 2237 patients who had underwent ASD device closure during 12 years [8]. 14 of 30 patients with embolized ASD devices had unfavorable septal morphology emphasizing the paramount importance of favorable septal morphology and adequacy of ASD rims for successful percutaneous ASD device closure. The procedural records were not available in our case. However, based on our device size measurements on TEE imaging we retrospectively speculate that a large sized ASD closure device was tried to be implanted in a large ASD having poor posterior rim. Acute changes in intracardiac pressures secondary to strenuous physical strain or heavy lifting have been also claimed to be potentially responsible for late embolization especially if accompanied by small rims.

Embolization of ASD closure device to atriums, right and left ventricles pulmonary artery and its branches, ascending aorta and descending aorta have been reported [7-9]. Garre et al reported that out of 30 ASD device embolizations, 10 devices were embolized to right side (right atrium and ventricle and main pulmonary artery or branches) and 7 devices embolized to left side (LV, proximal aortic arch, and the descending thoracic aorta) [8].

The timing of device embolization varies widely. Embolization usually occurs during or within following hours of index procedure and sometimes 1-2 days after intervention. However, it may also be seen many months after the intervention. Even, a peripheral embolization, in the left leg, one year after implantation was also reported [9]. The embolization time is not known in our patient since we don't know whether echocardiography was performed in her first control after index procedure or not and she didn't go to cardiology examinations thereafter since she was asymptomatic.

3.7 Management of ASD Device Embolization

Embolized devices are extracted via percutaneous snare or basket catheter and if not possible with surgery [3,7-9]. Attempts during percutaneous retrieval of embolized ASD device may result in damage in some of the cardiac structures, such as the inferior vena cava rim tear. Although the hemodynamic status remains stable in most of the patients having ASD device embolization, emergent retrieval of the device may be necessary in the case of obstruction of blood flow, formation of thrombus on the device, and thromboembolism [8]. Embolized devices can be successfully retrieved percutaneously in 50-70% of the cases and surgery is required in others [3,8,9]. Therefore, although the success rate of percutaneous ASD closure is nearly 99% [2], it's proper to do such interventions in tertiary centers having capability of emergent cardiovascular surgery due to rare complications that may necessitate emergent surgical intervention including device embolization. In the case of unsuccessful percutaneous retrieval and decision of delayed surgery or decline of surgery by the patient, anticoagulation should be preferred to prevent thrombus formation on the device and to minimize stroke or systemic embolization risk. If the embolized device was successfully retrieved percutaneously and the reason for embolization is malposition or small device, the percutaneous management may be reattempted with repositioning of a larger device. In case of conditions like inadequate, floppy rims and too large defects surgery should be advocated. In our case, we didn't try a percutaneous extraction with the expectation of adhesion of device with surrounding tissues.

3.8 Role of Eustachian Valve

Although medical records during the index procedure performed 13 years before were not available, embolization in our case, was probably secondary to large defect size which was measured 40 mm on TEE and at operation, absent posterior rim and misinterpretation of EV as posterior rim. The device waist diameter was probably 24 mm (the size; left disc diameter of extracted device was 38 mm on TEE).

EV is an embryological remnant of sinus venous. Commonly it disappears by time and seems as a thin ridge arising from the anterior rim of the IVC orifice. Sometimes, it may persist and appear as a membranous flap extending from the IVC or right atrial junction to the lower part of the fossa ovalis and even it can be large enough to mimic interatrial septal defects. It may be mistaken as tumor, thrombi or vegetation. Occasionally it may cause misinterpretation as cor triatriatum dexter. It doesn't cause symptoms usually. However, prominent EV has been claimed to be associated with paradoxical embolism and platypnea-orthodeoxia syndrome [10].

A prominent EV may also interfere guidewire management or device placement during percutaneous interventions for ASD and patent foramen ovale. Free end of EV may be confused with superior part of atrial septum and percutaneous ASD closure may be complicated. Misclassifying EV may be associated with device misplacement, residual shunting, and embolization. Even during surgery, EV has been mistaken as part of interatrial septum and incorporated into the patch followed by iatrogenic right to left shunt from inferior vena cava to left atrium [10-13]. Likewise, in our case probably, long flail EV bulging to left atrium was mixed as superior part of atrial septum and a small sized closure device was used to engage aortic rim and misinterpreted EV. Diversion of the vena cava inferior into the left atrium would result in cyanosis, hypoxia or even death. Therefore, if our retrospective assumption is correct, embolization of ASD closure device probably resulted in avoidance of such detrimental complications. Nevertheless, she lived with a migrated device in LVOT for 13 years, a situation which could be complicated with LVOT obstruction, arrhythmias and even sudden death. To avoid such a misleading situation careful assessment of atrial rims and surrounding structures should be made with various projections during TEE and if possible three dimensional echocardiography should be used. A three dimensional TEE with proper image acquisition and image rendering process will be useful in distinctive evaluation of right atrial structures including EV [4].An agitated saline injection may also be useful in some situations. Since EV is a remnant of sinus venous it lies anterior to vena cava inferior. Therefore, agitated saline injected ideally through femoral vein with Valsalva release would be seen from inferior vena cava posterior to the EV and anterior to true septum [13]. Nevertheless, it may be difficult to differentiate this condition with absent posterior rims like in our case.

3.9 Follow-up after ASD Closure

ESC guidelines for the management of adult congenital heart disease suggests regular follow-up patients after ASD closure during adulthood for the first 2 years and then, depending on results, every 3-5 years. Intervals for follow-up depends on characteristics and residual problems of the patients. Assessment of symptoms, functional capacity, 12-lead electrocardiography and evaluation of cardiac chamber sizes and functions, tricuspid regurgitation and PAP by echocardiography and if necessary ambulatory rhythm Holter monitorization is anticipated during these follow-up visits [2].

4. Conclusion

In conclusion, to avoid complications including device embolization TEE imaging is of paramount importance in assessment of ASD. The defect size and adequacy of rims should be carefully evaluated preprocedurally and appropriate device size should be selected accordingly. In addition, misinterpretation of EV like structures should be

avoided. The migrated device may stay silent, even in LVOT, for years. The patients should have routine follow-up visits and echocardiographic examinations to avoid such late potential complications.

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Conflict of Interest

There's no financial or personal relationships to disclose.

Generative AI Statement

The authors declare that no Gen AI was used in the creation of this manuscript.

Abbreviations

ASD: Atrial septal defect

EV: Eustachian valve

LA: left atrium

LVOT: Left ventricular outflow tract

PAP: pulmonary pressure

RA: right atrium

TEE: Transesophageal echocardiography

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