Late complications of transcatheter atrial septal defect closure requiring urgent surgery

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Introduction

Transcatheter atrial septal defect (ASD) closure has become a widely applied procedure – the recommended method of therapy in eligible patients due to the improved learning curve, cosmetic benefits and shorter recovery time [1]. Although the performance and safety of these devices appear to be reliable, certain risks and complications remain.

Case reports

Case 1

A 21-year-old female patient was transferred to our hospital after being diagnosed with massive pulmonary thromboembolism (PTE). Three years prior to admission, she underwent transcatheter closure of the secundum ASD with a 33 mm CardioSEAL-StarFLEX occluder (NMT Medical, Boston, MA, USA). A year ago, she was involved in a car accident and sustained significant blunt chest trauma. Transthoracic echocardiography confirmed the presence of thrombi in the right atrium and the pulmonary artery, with massive dilatation of the right ventricle and the pulmonary artery, along with severe pulmonary hypertension. Also, protrusion or dislodgement of the occluder was suspected. Her deteriorated clinical conditions warranted immediate surgery. The patient was put on a cardiopulmonary bypass (CPB) and the right atrium and the pulmonary artery were opened. Several thrombi were removed, the largest being 2 × 3 cm. The ASD occluder was identified with a thrombus attached to it and evident device-arm fracture (Figure 1). The occluder underwent almost complete healing with full endocardium covering except in the rim area. The device was removed and the ASD was repaired with a patch. Unfortunately, due to right heart failure, the patient could not be successfully weaned from the CPB, not even after an artificially created interatrial shunt, and she expired.

Although one cannot say with absolute certainty that massive PTE developed because of device-related thrombosis, it seems intuitive that blood turbulence around the protruded umbrella and device-arm fracture could have acted as a nidus for repeated thrombus formation with subsequent embolization. The occluder malfunction (fracture) was most likely the result of sustained blunt chest trauma a year prior to admission. We hypothesize that the sudden increase in intrathoracic pressure during trauma as well as direct compression on the heart generated a point of high wall stress around the occluder's septal insertion, which may have led to device fracture and dislodgment.

Case 2

A 32-year-old female patient was referred to our hospital with the diagnosis of acute aortic dissection. She complained of a sharp tearing pain in the chest and back with a loss of consciousness. A year prior to admission, she underwent ASD closure with a 26 mm Amplatzer occluder (St. Jude Medical, Minneapolis, MN, USA) in another hospital. Chest computed tomography was performed showing massive pericardial effusion and no clear origin of contrast extravasation, and no signs of intimal flap throughout the aorta. Due to a cardiac tamponade and deteriorated clinical conditions, the patient was rushed to surgery where she arrested during anesthesia induction and recovered upon pericardial incision and evacuation of 400 ml of blood. She was put on a CPB and the aortic root erosion in the region of non-coronary cusp was identified (Figure 2). No signs of other aortic pathology (dissection) were evident. The tear was sutured with two pledgeted sutures. The right atrium was opened with

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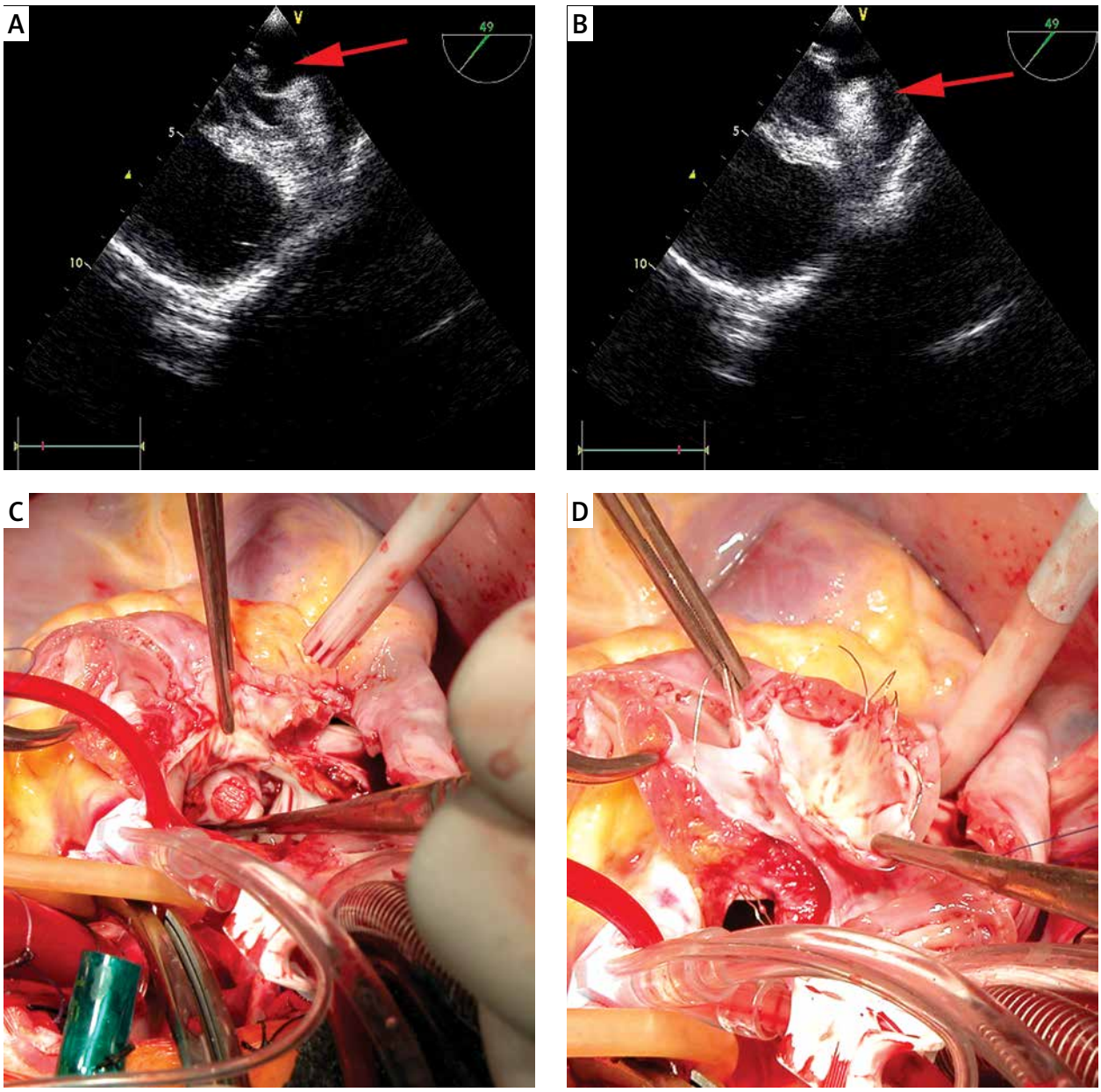

Figure 1. A, B – Trans-esophageal echocardiography demonstrating a mass in the right atrium and detached device, **C, D** – intraoperative finding

no signs of thrombi. The Amplatzer occluder was in close relation to the roof of the atria, and consequently the aortic root, and was undoubtedly the cause of the aortic perforation. It was extracted and the ASD was repaired with a patch. She made an uneventful recovery and was discharged home on the 8th postoperative day.

Discussion

Transcatheter closure of ASD is the recommended method of treatment in patients with suitable defect anatomy (class I) [2, 3]. Due to inherent design properties (size, shape, material, and construction), every device is associated with a specific type of complications, which are potentially life threatening. Complications commonly associated with ASD closure device include residual shunts, embolization, device-related thrombosis, erosion and perforation of the heart, infective endocarditis, and sudden death.

Perforation is the most feared complication described in the literature with the incidence of device erosion in the United States around 0.1% [4]. Perforation is most likely to happen during the first 48 h after the procedure and it is rarely manifested as a late complication. Perforation usually develops on the atrial dome and the adjacent aorta. Patients can present with haemopericardium, pericardial effusion, cardiovascular collapse, and sudden cardiac death. The U.S. Food and Drug Administration

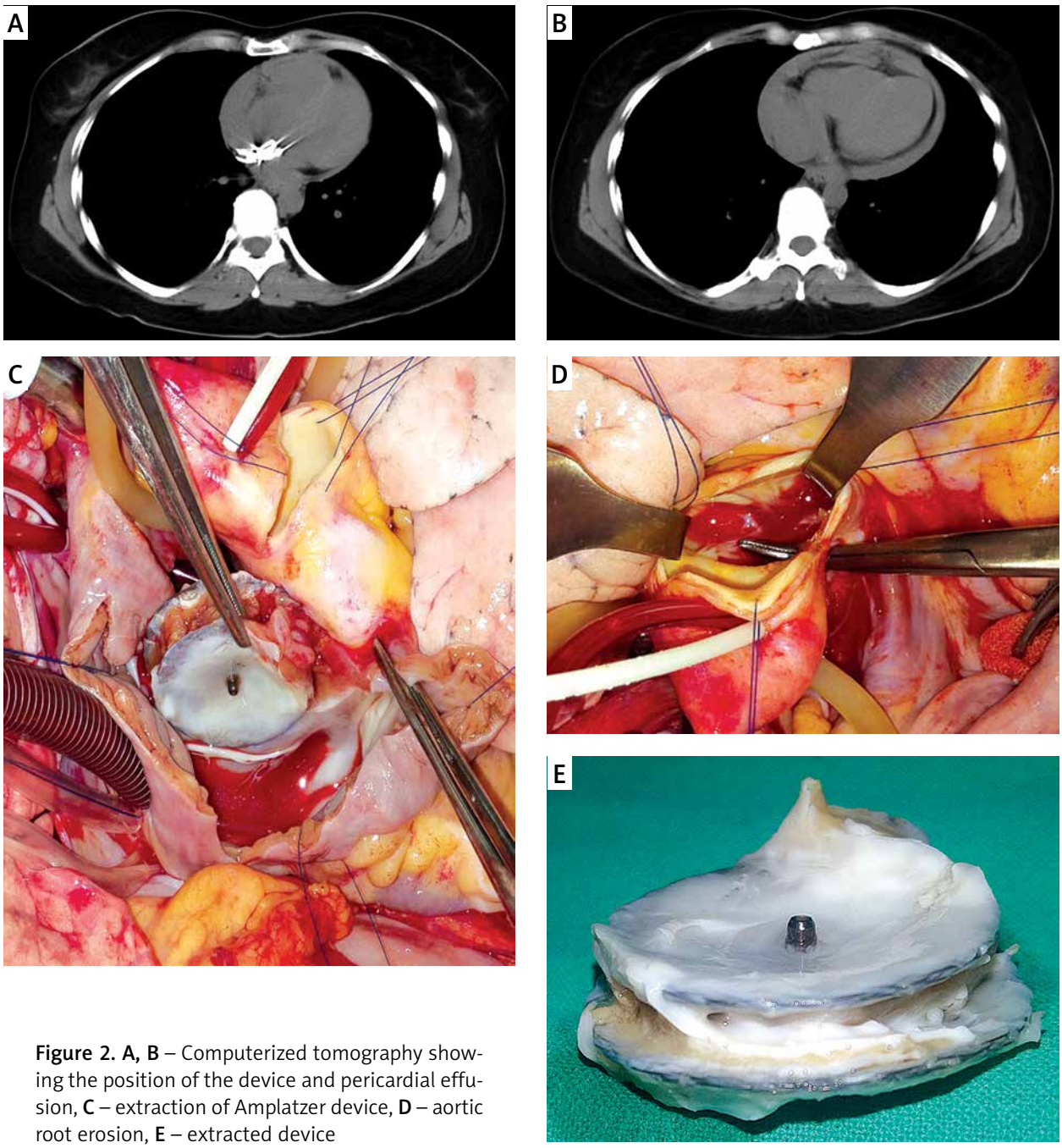

Figure 2. A, B – Computerized tomography showing the position of the device and pericardial effusion, **C** – extraction of Amplatzer device, **D** – aortic root erosion, **E** – extracted device

issued a warning about safety issues encountered with Amplatzer septal occluder devices stating that these devices may cause life-threatening tissue erosion inside the heart requiring immediate surgery. Several mechanisms on how the device may lead to perforation were proposed [4]. Deficient rims in vulnerable areas could increase the chance of contact between the device and the atrial wall in the same manner as the oversizing of a device.

Device-related thrombosis has been reported in many series with the incidence of thrombus formation of 1.2% in ASD patients [5]. The incidence of thrombus formation is highest during the first 4 weeks after device implantation and is extremely rare after 8–12 months. The specific design of the CardioSEAL-StarFLEX device (a metallic framework with Dacron fabric) rendered them prone to thrombosis as well as stress-mediated device arm fractures. The case of late thrombus formation demonstrated in our patient occurred following major blunt chest trauma that might have led to device-arm fracture that, in turn, could have acted as a nidus for repeated thrombus formation. Specifically, the majority of thrombi were found around the rim area of the occluder (fractured wires) – an area with no complete healing and endothelialization. Usually, thrombi resolve spontaneously

after anticoagulation therapy with heparin or warfarin, although some thrombi require surgical intervention, as demonstrated in our case. A longer period of surveillance after device ASD closure might be warranted in order to capture late occurrence of device malfunction, which may be associated with thromboembolic events.

Conflict of interest

The authors declare no conflict of interest.

References

1. Nugent AW, Britt A, Gauvreau K, et al. Device closure rates of simple atrial septal defects optimized by the STARFlex device. J Am Coll Cardiol 2006; 48: 538-44.
2. Kazmi T, Sadiq M, Asif-ur R, et al. Intermediate and long-term outcome of patients after device closure of ASD with special reference to complications. J Ayub Med Coll Abbottabad 2009; 21: 117-21.
3. Chessa M, Carminati M, Butera G, et al. Early and late complications associated with transcatheter occlusion of secundum atrial septal defect. J Am Coll Cardiol 2002; 39: 1061-5.
4. Amin Z, Hijazi ZM, Bass JL, et al. Erosion of Amplatzer septal occluder device after closure of secundum atrial septal defects: review of registry of complications and recommendations to minimize future risk. Catheter Cardiovasc Interv 2004; 63: 496-502.
5. Krumsdorf U, Ostermayer S, Billinger K, et al. Incidence and clinical course of thrombus formation on atrial septal defect and patent foramen ovale closure devices in 1,000 consecutive patients. J Am Coll Cardiol 2004; 43: 302-9.