

Delayed Left Atrial Perforation Associated with Erosion After Device Closure of an Atrial Septal Defect

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A 43-year-old man who had had a history of atrial septal defect (ASD) device closure 31 months previously presented with abrupt chest and back pain along with progressive cardiogenic shock and cardiac arrest. After resuscitation, he was diagnosed with cardiac tamponade. Diagnostic and therapeutic surgical exploration revealed left atrium (LA) perforation due to LA roof erosion from a deficient aortic rim. Device removal, primary repair of the LA perforation site, and ASD patch closure were performed successfully. The post-operative course was uneventful. The patient was discharged after 6 weeks of empirical antibiotic therapy without any other significant complications.

Key words: 1. Atrial septal defects
2. Atrium
3. Septal occluder device
4. Cardiac tamponade
5. Heart atria

Case report

A 43-year-old man visited the emergency room with complaints of abrupt chest and back pain that began 20 minutes earlier. The patient was previously healthy and did not take any medication, although he had a history of transcatheter atrial septal defect (ASD) device (Amplatzer Septal Occluder; AGA Medical Co., Golden Valley, MN, USA) closure performed 31 months ago. The initial electrocardiogram demonstrated normal sinus rhythm without ST elevation. Transthoracic echocardiography (TTE) showed moderate pericardial effusion or hematoma. During evaluation, the patient complained of severe pain with drowsiness and hypotension. Sudden cardiac arrest followed. The return of spontaneous circulation was

achieved within 1 minute with cardiopulmonary resuscitation and intubation. Systemic hypotension was improved after urgent pericardiocentesis. Computed tomography (CT) angiography was then performed. There was no evidence of acute aortic dissection or intramural hematoma. However, the moderate hemopericardium extended into the superior recess. The ASD closure device had not migrated (Fig. 1). After the patient's admission to the intensive care unit, TTE revealed moderate hemopericardium with mild right atrial collapse, suggesting residual tamponade physiology. The ASD closure device was found *in situ* in the interatrial septum, abutting to the aortic root due to the scant aortic rim (Fig. 2). However, there was no evidence of any residual interatrial shunt, valvular dysfunction, or device erosion. Transseso-

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phageal echocardiography showed that the pericardial effusion had resolved, and there were no additional abnormal findings. The patient was extubated 1 day after admission. His left pleural effusion was controlled by closed thoracostomy with a 10-Fr mini-tube. Although the patient's status stabilized after appropriate management, we were not able to identify the exact reason for the hemopericardium.

Clinically, we ruled out left atrium (LA) perforation

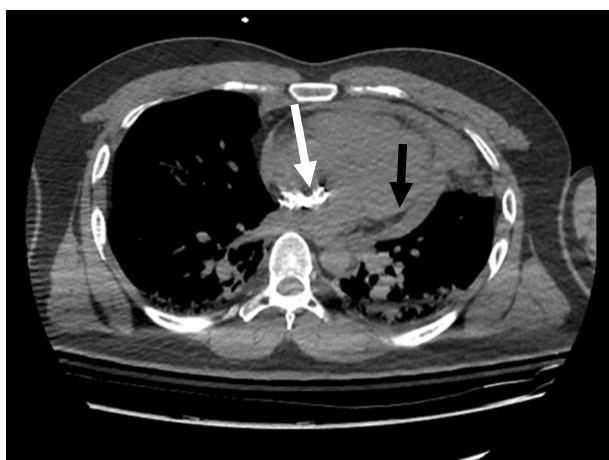


Fig. 1. Computed tomography angiography showing no evidence of acute aortic dissection or intramural hematoma. The white arrow depicts the atrial septum defect closure device *in situ*. The black arrow shows the remaining pericardial effusion, consistent with hemopericardium.

on the basis of the ASD closure device abutting the aortic root and the LA roof. Elective surgical exploration was planned after stabilizing the patient's vital signs. However, it was difficult to decide to perform surgical exploration for resolved hemopericardium without a definite reason or new symptoms. Although the patient exhibited no abnormal laboratory findings or fever and the culture results were negative, empirical antibiotics (vancomycin, gentamicin, and rifampicin) were started in accordance with infective endocarditis on the prosthetic valve. Diagnostic and therapeutic surgical exploration was performed on the eighth day after admission. With full median sternotomy and pericardiotomy, the pericardial space was filled with dark-blood-colored pericardial effusion. Mild adhesion around the LA roof, aortic root, and the right pulmonary artery was found. However, there was no evidence of active bleeding, organized hematoma, abscess formation, or infective tissue. Right atriotomy was then performed under aorto-bicaval cannulation with antegrade cold crystalloid cardioplegic arrest. The ASD closure device was located in the appropriate position. However, the aortic rim was nearly absent. The device was removed with electrocautery and dissection using Metzenbaum scissors. After inspecting the LA cavity via the ASD, we identified the LA perforation site. A 2-mm probe could pass through the perforation site (Fig. 3). This site was repaired primarily with a 4-0 polypropylene pledgetted buttressed suture. The ASD was closed

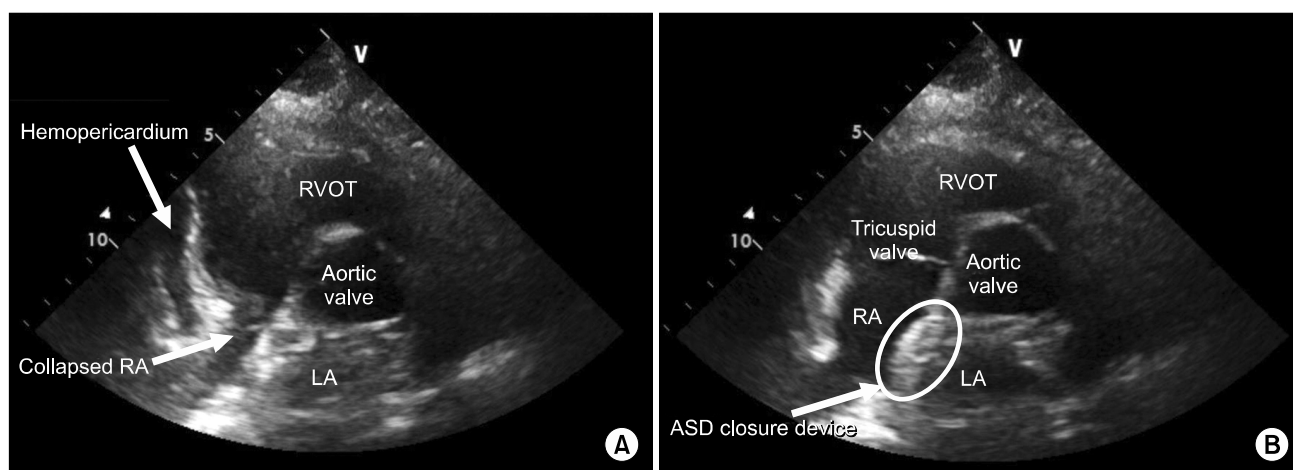


Fig. 2. In the early systolic phase of the parasternal short-axis view, moderate hemopericardium with mild RA collapse is seen, suggesting residual tamponade physiology (A), and after atrial filling, the ASD closure device is seen attached only to the aortic root (B). RVOT, right ventricular outflow tract; LA, left atrium; RA, right atrium; ASD, atrial septum defect.

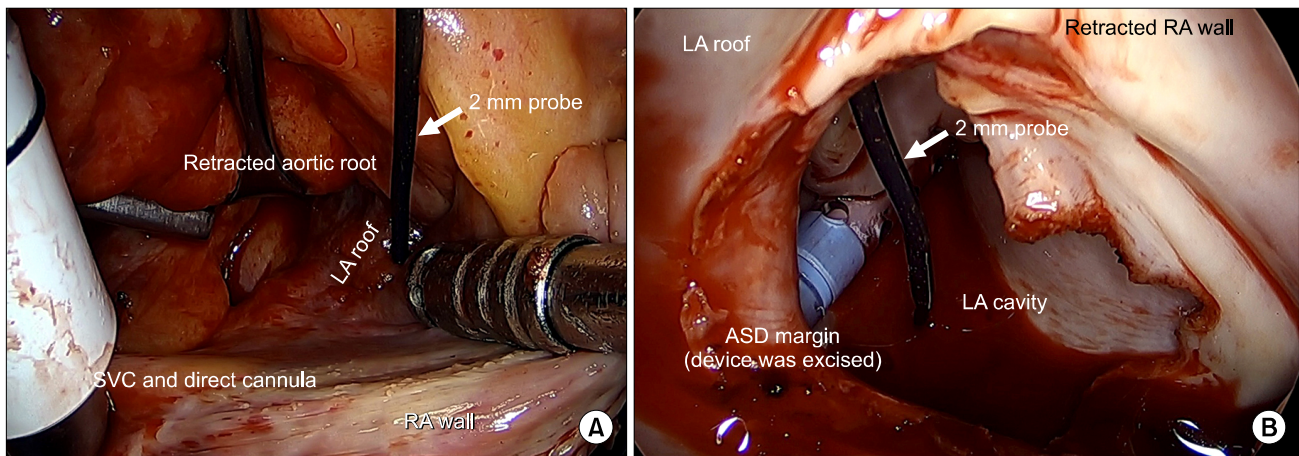


Fig. 3. Intraoperatively, a 2-mm probe (arrow) could pass through the LA roof perforation site (A). Further, with RA wall retraction, the probe was seen in the LA cavity via the ASD (B). LA, left atrium; SVC, superior vena cava; RA, right atrium; ASD, atrial septum defect.

with a glutaraldehyde-fixed autologous pericardial patch. The cardiopulmonary bypass time and the aortic cross clamp time were 108 minutes and 56 minutes, respectively. The patient was extubated 2 hours after surgery and transferred to a general ward on postoperative day 1. Persistent mild sinus tachycardia was controlled with a low dose of oral beta blockers. Postoperative TTE demonstrated normal findings without residual tamponade physiology or pericardial effusion. The rest of the postoperative course was uneventful. The patient was transferred to department of cardiology and maintained with 2 weeks of gentamicin and 6 weeks of vancomycin and rifampicin therapy. He was discharged without any other complications.

Discussion

The closure of secundum ASD without severe irreversible pulmonary artery hypertension either percutaneously or surgically is a preferable therapy for adult patients [1]. Usually, no absolute contraindication exists for the closure of ASD using a device. However, insufficient peridefect rims are considered to be relative contraindications. The Amplatzer Septal Occluder (AGA Medical Co.), which was approved by the US Food and Drug Administration in December 2001, is a safe and effective closure device for children and adult patients. Although the device has been used widely, several early and late complications have been reported, including device malposi-

tion, device embolization, arrhythmia, pericardial effusion, thrombus formation, and iliac vein dissection [2,3].

Among the complications after transcatheter ASD device closure, cardiac erosion with tamponade is very rare but extremely fatal. Most of the complications from erosion occur within 1 year of device closure, but more than 10% of the cases of erosion occurred more than 1 year after device closure [4]. With proper clinical follow-up, early erosion can be treated appropriately [5]. However, as in this case, sudden cardiac tamponade caused by cardiac erosion several years after the procedure is difficult to distinguish from hemopericardium due to other causes. Therefore, if a patient with a history of device closure of ASD presents with chest pain, syncope, or other shock symptoms, device-related cardiac erosion should be suspected and urgent evaluation should be performed for confirmation. Moreover, long-term regular clinical follow-up is recommended [1].

A deficient retroaortic rim has been considered to be a risk factor for erosion. However, O'Byrne et al. [5] have argued that it is not associated with an increased risk of technical failure or early adverse events. On the other hand, McElhinney et al. [4] have evaluated the relative risk factors and revealed that deficiency of any rim, the ratio of the size of the device to the size of the rim, and oversized devices are associated with erosion. A recent study has shown that absence of the aortic rim, poor posterior rim consistency, septal malalignment, and dynamic mor-

phology of the ASD in echocardiographic findings can significantly increase the risk of erosion [6]. In addition, a case report has described device embolization in the left ventricular outflow tract (LVOT) in a left-to-right shunt ASD patient with an aortic rim defect [7].

With a low incidence of early and late complications, interventional cardiologists often do not hesitate in using the device. However, given the severity of complications such as LVOT embolization or device erosion, decision-making regarding device closure for ASD patients with some risk factors should be based on a multidisciplinary team approach.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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