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Late cardiac tamponade after percutaneous closure of a patent foramen ovale

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Abstract We report the case of a 35-year-old man who had a transient ischemic cerebral attack and then underwent a percutaneous closure of the patent foramen ovale (PFO) with a Cardiastar device. One year later, the patient developed a cardiac tamponade due to an important hemorrhagic pericardial effusion. Transoesophageal echocardiography showed that one of the struts had impinged on the aortic root in the region adjacent to the transverse pericardial sinus. Therefore, we speculated that the strut had passed through the aortic wall by slow erosion, leading to the pericardial effusion. Cardiac CT and subsequent surgery confirmed the perforation of the left atrial roof and the aortic root by two struts of the device. This is the first reported case of late cardiac tamponade and underscores the importance of long-term follow-up after PFO closure device implantation.

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Introduction

A 35-year-old man with no previous medical problems was admitted to our hospital in February 2003 because of a transient ischemic cerebral attack. A small left frontal ischemic lesion was detected with a brain CT-scan. Furthermore, we found a patent foramen ovale (PFO), which had a significant right-to-left shunt that was associated with an atrial septal aneurysm, by transthoracic echocardiography (TEE) (Fig. 1A and B). The distances from the PFO to the superior vena cava and the aortic root were 12 mm and 7 mm, respectively (Fig. 1C and D). Following neurological
recovery, we used a Cardiastar device (3/30 mm, third generation) to perform a percutaneous closure of the PFO. Besides fluoroscopy, other imaging techniques were not performed. The patient was discharged after the uneventful percutaneous PFO closure and he returned six months later for a follow-up TEE. This exam did not indicate any pathological mobility or perforation of the intracardiac device, but did show that it was in contact with the aortic root (Fig. 2A). Furthermore, a very small residual right-to-left shunt during the Valsalva manoeuvre was also detected (data not shown).

The patient returned to our hospital one year later due to a sudden onset of left thoracic pain while at rest, which was followed by dizziness and syncope. On examination, his jugular veins were raised and he developed cardiogenic shock that required mechanical ventilation and Adrenaline i.v. Because an important pericardial effusion with compression on the right cardiac chambers was detected by a transthoracic echocardiography (Fig. 2B), we performed an emergency pericardial puncture and removed 250 ml of hemorrhagic fluid. An aortic dissection was ruled out, first by a thoracic CT-scan (data not shown) and then by a TEE. Importantly, the TEE showed no pathological mobility of the Cardiastar device, but it indicated that one of the struts had impinged on the aortic root, in a region adjacent to the transverse pericardial sinus (Fig. 2C). Consequently, we speculated that the left atrium and the proximal aorta had been perforated, which resulted in the pericardial effusion. In fact, in comparison with the first TEE (Fig. 2A), the struts had spread open and pushed against the aortic root (Fig. 2C). By cardiac CT-scan, we confirmed that the Cardiastar’s cranial strut had passed through the

Figure 1  Anatomy of the PFO prior to a Cardiastar device implantation, by transoesophageal echocardiography. An important right-to-left shunt during the Valsalva manoeuvre (A) and an atrial septal aneurysm (B, double arrow) were seen. (C) The distance between the PFO and the superior vena cava was 12 mm in the cranio-caudal direction (double arrow). (D) The distance from the PFO to the aortic root was 7 mm in the posterior–anterior direction (arrow). LA = Left atrium, RA = right atrium, Ao = aortic root, SCV = superior caval vein.
aortic wall (Fig. 3). During removal of the Cardiastar device by open-heart surgery, we detected two nitinol struts from the Cardiastar device, which had perforated the roof of the left atrium (Fig. 4) and one of these two had perforated into the non-coronary sinus of Valsalva of the aortic root. The patient recovered uneventfully from this surgery and was asymptomatic one year later.

Discussion

Based on autopsy reports, the prevalence of PFO is 17–35% in the general population. While numerous studies have suggested an association between PFO and cryptogenic stroke, there are limited explanations on how atrial septal abnormalities are implicated in cryptogenic stroke. In spite of this, percutaneous PFO closure in patients with a paradoxical embolism has emerged as an alternative treatment strategy to lifelong anticoagulation, antiplatelet agents or surgical PFO closure. Several peri-procedural and short-term complications have been recognized in PFO device implantation, but their rate is low. Pericardial effusion after percutaneous PFO closure has been reported as a rare early complication. This complication was described with different devices. In this case, we suspected that slow erosion of the left atrial roof and the aortic wall were responsible for a hemorrhagic pericardial effusion. There was no shunt detected between the aorta and the left atrium because the responsible strut remained in place until surgical removal. We believe that the described complication is due to both the device and its position. In fact, star-like devices have been associated with a higher rate of complications, including early tamponade, when compared to stent-like devices. Furthermore, the PFO in our patient was close to the aortic root, which makes the implantation of a device more difficult, and the use of a star-like device possibly more dangerous.

In conclusion, this case illustrates that TEE or intracardiac echo imaging during device implantation is advisable to evaluate the position of the umbrella with respect to the aortic root. Furthermore, this report shows that pericardial effusion can occur even one year after percutaneous PFO closure, which highlights the importance of long-term follow-up of patients after device implantation.
Figure 3  Gated cardiac CT one year following Cardiastar device implantation. Curved multiplanar reconstruction (MPR) at the level of the coronary ostia (A) oblique sagittal MPR (B) and superior view (C) of volume rendering after removal of the left atrium to visualize the atrial septum and the device. Together, these three views showed that the superior struts of the device cross the left atrial roof and protrude into the aortic wall and the non-coronary sinus of Valsalva (all arrows). PA = Pulmonary artery, RA = right atrium, Ao = ascending aorta, LA = left atrium.

Figure 4  Surgical removal of the Cardiastar device. (A) Two struts from the device (arrows) had passed through the roof of the left atrium. (B) In order to remove the device and close the PFO, the right atrium was opened revealing the right-sided part of the device in situ. LAR = Left atrial roof, AA = ascending aorta, SCV = superior caval vein, IVC = inferior caval vein, IAS = interatrial septum.
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