Case Reports

Left-Atrial-Appendage Occluder Migrates in an Asymptomatic Patient

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Percutaneous closure of the left atrial appendage (LAA) is a new approach to the prevention of cardioembolic events in patients with atrial fibrillation. We implanted an LAA occlusion device (AMPLATZER™ Cardiac Plug) in a 70-year-old woman via a transseptal approach. Upon her discharge from the hospital, a transthoracic echocardiogram showed stable anchoring of the device; 6 months after implantation, a routine transthoracic echocardiogram revealed migration of the occluder into the left ventricular outflow tract, in the absence of symptoms. We surgically removed the device from the mitral subvalvular apparatus and closed the LAA with sutures. This case shows that percutaneous LAA occlusion can result in serious adverse events, including device migration in the absence of signs or symptoms; therefore, careful follow-up monitoring is mandatory. ***(Tex Heart Inst J 2014;41(4):443-4)***

Atrial fibrillation (AF) is present in 0.95% to 1.12% of the total population[1,2] and is responsible for thromboembolic complications in 4.5% of AF patients.[3] In patients with nonvalvular AF, chronic oral anticoagulant therapy reduces the risk of thromboembolism; however, warfarin is contraindicated in many patients. In patients with nonvalvular AF, 90% of stroke-related thrombi form at the left atrial appendage (LAA)[4]; percutaneous exclusion of the LAA might offer non-warfarin candidates an alternative solution to long-term oral anticoagulation.[5,6]

Case Report

A 70-year-old woman with chronic AF, a history of transient ischemic attacks, and a contraindication to anticoagulation due to chronic hepatitis C was identified as a candidate for percutaneous closure of the LAA. The appropriate LAA occlusion device (AMPLATZER™ Cardiac Plug; St. Jude Medical, Inc.; St. Paul, Minn) was selected after echocardiographic measurements of the orifice diameter of her atrial appendage. In March 2010, the device was implanted percutaneously through a transseptal approach. Fluoroscopy, angiography, and intraprocedural echocardiography were used to guide the delivery. The final implant position was confirmed by cineangiography with contrast medium injections distal to the deployed device, and also by transesophageal echocardiographic color-flow Doppler examination of the left atrium.

After LAA closure, the patient was treated with clopidogrel, 75 mg twice daily, for 6 weeks, and with aspirin, 300 mg/d, indefinitely. The postprocedural course was uneventful, with good device placement as seen upon transthoracic echocardiography (TTE); the patient was discharged from the hospital on the day after her procedure.

The patient did not attend her scheduled one-month follow-up visit. Six months after the procedure, asymptomatic and hemodynamically stable, she did undergo a scheduled TTE. This study revealed that the cardiac plug had migrated into the left ventricular outflow tract, where it was trapped in the mitral subvalvular apparatus, producing trivial-to-mild mitral incompetence (Fig. 1). We elected to perform urgent open-heart surgical removal of the device.

Intraoperatively, the device appeared to be attached to the left ventricular outflow wall by fibrous adhesions: we removed the device carefully, with no damage to the mitral apparatus (Fig. 2). (The possibility of performing a Cox maze procedure as an intraoperative remedy for the AF had been discussed with the electrophysiology team, who concluded that it was not indicated.) The LAA was then occluded by means of continuous suture. The patient's postoperative course was uneventful, and

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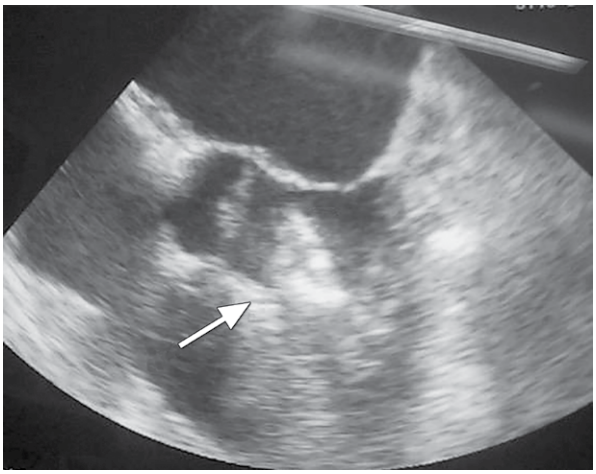

Fig. 1 *This transthoracic echocardiogram shows the occlusion device (arrow) trapped in the mitral subvalvular apparatus.*

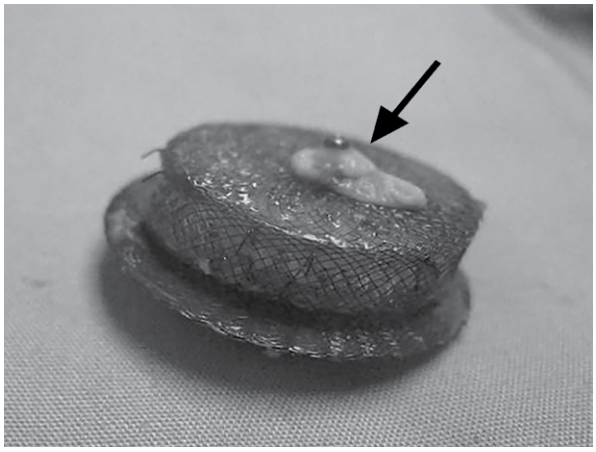

Fig. 2 *Photograph shows the surgically removed occlusion device, free of contiguous cardiac tissue. The arrow points to fibrotic tissue resulting from the device's adhesion to the mitral subvalvular apparatus.*

she was discharged from the hospital in good condition. At her 1- and 3-year follow-up visits, the patient was still asymptomatic and free of cardioembolic events; moreover, her mitral valve displayed good competence when tested by TTE.

Discussion

On the basis of the promising clinical results, transcatheter LAA exclusion is progressively gaining ground as a method to reduce the risk of thromboembolic events in patients who have nonvalvular AF that is unsuitable for chronic oral anticoagulation.[6,7]

Several devices on the market claim to reduce the potential of serious LAA occlusion sequelae, including pericardial effusion requiring pericardial drainage, acute ischemic stroke due to air embolism or solid thromboembolism, embolization of the device, and postimplantation sepsis. We selected the AMPLATZER Cardiac Plug for its design, which is characterized by a crown of retaining hooks at the distal end, its user-friendliness, and its excellent record of low thrombogenicity.[8]

Early dislocation or embolization of an LAA device has been reported, but it has never occurred late after the procedure, as in our patient. Acute displacement is generally an emergency situation that requires an immediate percutaneous or surgical procedure for device removal.[9] In our patient, percutaneous removal was impossible because the device was entrapped in the mitral apparatus.

We conclude that, even in the absence of symptoms, careful early and late echocardiographic examinations (1-month and 6-month follow-ups) are indicated in order to exclude a faulty implant position, device migration, or device encroachment upon surrounding cardiac structures that could result in serious lesions.

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