Case Report

Cryptogenic Stroke After Percutaneous Closure of an Atrial Septal Defect

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We present the case of a patient who underwent a percutaneous secundum atrial septal defect (ASD II) closure with an undersized septal occluder device. One week and one month later she experienced two transient ischemic attacks. Three-dimensional transesophageal echocardiography (TEE) revealed a residual patent foramen ovale (PFO) with a positive Valsalva bubble test. She underwent a second procedure under the 3D TEE guidance and the PFO was successfully closed percutaneously using a PFO occluder device that was attached to the ASD device. Accurate ASD and PFO morphology assessment and appropriate device selection are the key factors in the success of percutaneous closure. 3D TEE is an innovative diagnostic technique, providing a complete description of the cardiac defect and improving spatial orientation. Real-time 3D TEE is the appropriate guidance for successful and accurate positioning of the device.

The incidence of percutaneous closure of a secundum atrial septal defect (ASD) and patent foramen ovale (PFO), which has become an established therapy, is constantly increasing.¹ Transcatheter ASD closure is a safe and effective treatment modality with excellent long-term success rates,² provided that the defect is appropriate for percutaneous closure and the device is deployed successfully.

It is well known that patients who have suffered a cryptogenic stroke, which has been associated with PFO, are at risk of recurrent stroke, despite being on medical treatment.³⁻⁶ The incidence of recurrence of the stroke in these patients varies from 0-15% per year.⁷⁻¹⁰ This risk is particularly increased in patients who have a combination of PFO and atrial septal aneurysm.⁵,¹¹,¹² The likeliest mechanism of stroke in these patients is paradoxical embolization through the PFO.¹³ It appears that percutaneous closure of the PFO is at least as effective as medical treatment in preventing the recurrence of stroke. Moreover, closure appears to be more effective than medical treatment in patients who have suffered more than one event.¹⁴

Amplatzer devices, specifically the Amplatzer Septal Occluder® and Amplatzer PFO Occluder® (AGA, Medical Corporation, Golden Valley MN, USA), have been granted FDA approval for percutaneous closure of ASD and PFO, respectively. At the moment these devices are the most commonly used devices for percutaneous closure of ASDs.¹⁵ The results of percutaneous closure with these particular devices are quite encouraging.¹⁶,¹⁷

We present the case of a 34-year-old woman who experienced two transient ischemic attacks (TIA) one week and one month after a percutaneous ASD closure, as the result of a residual PFO.

Case presentation

A 34-year-old woman underwent uncom-
plicated percutaneous secundum ASD closure because of signs of right heart volume overload. ASD closure was performed with an 18 mm Amplatzer septal occluder device and she was discharged on aspirin (100 mg od). One week later she experienced a TIA with sudden loss of vision in her right eye (amaurosis fugax). A two-dimensional transesophageal echocardiography (2D TEE) examination showed a possibility of thrombus on the left atrium side of the device and she was started on warfarin. One month later, and despite her being fully anticoagulated, she experienced a second TIA. She was fully investigated neurologically and nothing was found. She underwent 2D (Figure 1) and 3D (Figure 2) TEE, which revealed a residual PFO that remained uncovered by the ASD occluder device, with a positive Valsalva bubble test, whereas it did not detect any thrombus in the left atrium. The 3D images, in particular, revealed that, due to device dislodgement, the two disks of the Amplatzer ASD occluder device failed to embrace the septum secundum anteriorly and superiorly and were limited to the septum primum, occluding only the ASD. Due to the weight of the device, the previously undiagnosed PFO was held wide open, facilitating ample passage of bubbles during the Valsalva maneuver. Thus, the residual PFO was the substrate for thrombus formation, as well as the passage to the left atrium and consequently the reason for the TIA.

The patient underwent a second procedure and the PFO was successfully closed percutaneously with a 25 mm Amplatzer PFO occluder device. The procedure was performed under fluoroscopy and 2D/3D TEE guidance. 2D and 3D (Figure 3) TEE images confirmed the complete closure of the PFO (the PFO device was attached to the ASD device). She was discharged on aspirin 325 mg for six months. Her postoperative recovery was uneventful. At one-year follow up, the patient is asymptomatic and off antithrombotic medications for the last 6 months.

Discussion

Transcatheter closure of an ASD is a valid alternative to the surgical approach, with less morbidity than surgical closure. It is currently indicated for the closure of hemodynamically significant (right ventricular volume overload) ASDs with a sufficient rim of tissue around the septal defect, so that the closure device does not impinge upon the superior vena cava (SVC), inferior vena cava (IVC), or the tricuspid or mitral valves. Amplatz devices are clinically safe and effective in ASD closure. Periprocedural complications are rare and late complications even rarer. An increased incidence of transient arrhythmias (usually new-onset atrial fibrillation) has been observed during the post-implantation period, without any clinical consequence. Device embolization, either early or late, is usually the result of suboptimal implantation technique, and mainly due to an undersized device and an insufficient rim at the inferior-posterior defect.

Figure 1. Mid-esophageal long axis view at 90° showing left-to-right linear flow through a patent foramen ovale. The Amplatzer device is visualized inferior to the defect. AD – Amplatzer device; AO – aorta; LA – left atrium; RA – right atrium.

Figure 2. Real-time three-dimensional transesophageal echocardiographic image showing a wide patent foramen ovale (PFO) that extends superiorly and posteriorly. The PFO is held open by the Amplatzer device (AD) that, due to its relative small size, fails to embrace the septum secundum and merely occludes the secundum defect. SVC – superior vena cava. Other abbreviations as in Figure 1.
The incidence of thrombus formation on Amplatzer devices is very low. In a recent study, the incidence of complications requiring emergency surgery (hemopericardium, device embolization, and pericardial tamponade) was only 0.9%. The prevalence of residual shunt is very low, and is associated with the presence of large and multiple defects and the use of multiple devices.

In our case, the undersized Amplatzer septal occluder failed to embrace the septum secundum anteriorly and superiorly and was limited to the septum primum, occluding only the ASD. Due to its weight, the previously undiagnosed PFO was held wide open, facilitating ample passage of micro-bubbles during the Valsalva maneuver. However, the previously unrecognized PFO led to a clinical syndrome (TIAs) due to: a) the changes in local anatomy caused by the first Amplatzer device; and b) the possibility that the anatomical relation of an Amplatzer device with a PFO may became the substrate of thrombus formation.

PFO has been associated with cryptogenic stroke. There is a higher incidence of PFOs in patients with cryptogenic stroke (44-66% in patients with cryptogenic stroke vs. 9-27% in normal controls). As a PFO is usually a tunnel, it has been speculated that the thrombus formation can actually take place within the PFO. The recurrence of strokes has been related with the PFO’s size as well as the presence of an atrial septal aneurysm. The therapeutic options regarding secondary prevention of stroke in patients with PFO include medical treatment (antiplatelet or anticoagulant treatment), and percutaneous or surgical closure of the defect. According to contemporary guidelines, the standard therapy for patients with an ischemic stroke or TIA and a PFO is antiplatelet medication (Class IIa, level of evidence C). PFO closure may be considered for patients with recurrent cryptogenic stroke despite optimal medical therapy (Class IIb, level of evidence C). Despite our patient being on aspirin, and subsequently on full anticoagulation, she experienced two TIAs. Consequently, the silent shunt (PFO) should have been corrected.

At present, pre-interventional evaluation (shunt volume, defect size, relation with adjacent anatomical structures) and post-interventional follow up (device location, thrombus presence, any residual shunt occurrence) of secundum ASDs are routinely performed using 2D TEE, which is considered the gold standard. However, ASDs have a complex geometry that may be elliptical, oblong, fenestrated in shape or may be multiple, precluding accurate and detailed defect assessment by 2D TEE. 3D TEE overcomes these limitations, facilitating the accurate description of ASDs. A recent study showed that 2D TEE returns a smaller long-axis dimension, and a larger short-axis dimension when compared with 3D TEE. In our case, real-time 3D TEE facilitated the diagnosis of PFO and was also used to monitor the implantation of the second Amplatzer device.

In the present case, the undersized occluder device was the reason for the incomplete closure of the secundum ASD. The recognition of the residual PFO was difficult, and was facilitated greatly by the use of 3D TEE. The PFO was clinically silent prior to the ASD occlusion, but led to significant recurrent embolic events after the implantation of the Amplatzer device. Percutaneous closure of the
PFO using a second device was successful and accurate positioning was achieved by careful real-time monitoring with 3D TEE. Accurate ASD morphology assessment and appropriate device selection are the key factors to procedural success. Real-time 3D TEE is a more reliable complementary option to 2D TEE for assessment ASDs and for guidance in device positioning.

References

