Letters to the Editor

Use of a Second Device for the Closure of Patent Foramen Ovale

To the Editor,

The persistence of patent foramen ovale (PFO) in adults has been correlated with an increased incidence of cryptogenic stroke or strokes of an undetermined etiology. Percutaneous PFO closure in high-risk patients or patients with recurrent strokes in spite of medical treatment is a feasible, safe, and efficient technique.1

Here, we present the case of a 52-year-old woman, with a history of migraines with auras, who came to the hospital due to a sudden left hemiparesis and right central facial paralysis with aphasia. The symptoms disappeared a few hours after admission and the patient recovered completely. After performing an echocardiogram and a 24-hour Holter test which showed sinus rhythm, a CT scan and nuclear magnetic resonance imaging showing no lesions, a complete coagulation study with normal results, a Doppler-Ultrasound of the supra-aortic trunks with no alterations, and a transthoracic echocardiogram showing the presence of PFO following bubble injection, the diagnosis of transient ischaemic attack (TIA) of an unknown etiology of the middle left cerebral artery was established.

This being the first episode of TIA, and since the patient had PFO with high-risk criteria, medical treatment with acetylsalicylic acid at 100 mg/24 h was initiated. At 5 months, the patient suffered another TIA, and we decided to close the PFO.

A 33 mm CardioSeal device (Figure 1) was implanted as usual, with no complications. However, the transthoracic echocardiogram taken before discharge (24 hrs after implantation) showed the presence of a moderate residual shunt (Figure 2). In the
Echocardiograms taken at one month and 6 months, we confirmed the persistence of the shunt, which had not reduced in size, and also observed the passage of bubbles using Valsalva maneuvers (Figure 2), and so we decided to perform a percutaneous closure of the residual shunt. A transeptal puncture was performed in the area above the device, previously implanted under echocardiographic control, and an Amplatzer ASD 18 mm device was implanted (Figure 1). A complete closure was confirmed along with an absence of passage of bubbles one month and 6 months after the procedure. After 2 years of follow-up, the patient still has not had any recurrences, and the migraines have disappeared.

The presence of at least a moderate residual shunt after a percutaneous PFO closure has been related to increased risk of recurrent stroke during follow-up. Complete PFO closure is achieved in 95% of cases, but the presence of moderate or severe shunts has been described in 2%-3%. An incomplete closure can be related to an under-sized device, an inadequate design of the device given the morphology of the PFO, or multiple septal fenestration. The treatment for this subgroup of patients is not clear: medical treatment can be continued, surgical closure can be considered, and recently, percutaneous closure of the residual shunt has been described.

In the case we have presented, the second device implantation was performed with no complications using the transeptal puncture technique, which facilitates the placement of a second device in the area of the shunt. During the follow-up period of this high-risk patient, no recurrent cerebral strokes have been recorded.

Studies are required that include a greater number of patients in order to confirm the safety and efficacy of this technique.

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Figure 1. A: CardioSeal implant device (33 mm) for closing a patent foramen ovale. B: implant of a second 18 mm Amplatzer ASD device to close the residual shunt.

Figure 2. A: Echocardiogram following the implantation of the first device showing the persistence of the residual shunt through a Doppler color image. B: Echocardiogram following implantation of the first device showing the passage of bubbles through the patent foramen ovale during the Valsalva maneuvers.
To the Editor,

A 42-year-old male patient was admitted to the hospital with fever and chest pain complaints. The patient had a history of human immunodeficiency virus (HIV) infection, which was diagnosed 15 years before the hospital admission. The CD4+ T cell count at admission was 874 cells/µL and viral load was undetectable (<50 copies/mL). Nadir of CD4+ T cells was 414 cells/µL in 1996 and patient was in treatment with tenofovir + didanosine + atazanavir + ritonavir since 2003 with an undetectable level of viral load the last 6 years. He had no relevant prior cardiac history.

He began having fever, diarrhea, poor general condition and myalgia over the course of one week. In the last two days prior to admission he developed progressive intense stabbing chest pain at rest, with radiation to the back that worsened with recumbent position and deep breaths. Chest pain intensity diminished following intravenous analgesics but not sublingual nitroglycerin.

On physical examination the vital signs were normal, other than a body temperature of 38ºC. He did not have signs of systemic or pulmonary congestion, neither low output signs nor respiratory distress. The heart sounds were normal.

Laboratory data revealed leukocytosis with lymphocytosis, C-reactive protein was 19 mg/mL and peak cTnI reached 30 ng/mL. Chest radiography was unremarkable. ECG showed normal sinus rhythm with 1 mm ST-segment elevation in leads DI, aVL, and V4-V6 (Figure. 1). Nasal swab, blood, feces and urine were taken for culture and real-time reverse transcription polymerase chain reaction (RT-PCR) assays.

Despite the initial clinical suspicion of myocarditis a coronary angiography was performed that ruled out coronary artery disease.

The 2D echocardiography obtained in the emergency department showed a normal sized non-hypertrophic left ventricle with inferior wall hypokinesis and normal global systolic function, with no pericardial effusion.

The patient was admitted with a diagnosis of acute myocarditis. The RT-PCR was positive for Pandemic Influenza A (H1N1) infection.

A conventional delayed-enhanced Cardiac Magnetic Resonance study conducted 3 days after admission shows typical intramyocardial latero-apical and epicardial inferior late enhancement, as well as high signal intensity at T2-weighted imaging, consistent with acute multifocal myocarditis (Figure 2).

The patient was treated with oseltamivir with good clinical response. He did not develop any complications during the hospitalization. When treatment was completed, a second RT-PCR assay for H1N1 was negative. He was discharged with the diagnosis of acute viral myocarditis secondary to H1N1 infection.

As of April 14, 2010, more than 208 countries and overseas territories or communities have reported...