

## Short Communication

# Risk of Stroke and Thrombus Formation From Delay Incontinence of a PLAATO-Device in Friedreich Ataxia

Claudia Stöllberger, MD; Josef Finsterer, MD; Marion Avanzini, MD;  
Günther Mölzer, MD; Franz Weidinger, MD

Second Medical Department, Krankenanstalt Rudolfstiftung, (Stöllberger, Avanzini, and Weidinger); Krankenanstalt Rudolfstiftung (Finsterer); Department of Radiology, Krankenanstalt Rudolfstiftung (Mölzer) Vienna, Austria

## ABSTRACT

We present the case of a 76-year-old female with suspected Friedreich ataxia, in whom leakage and thrombosis of a percutaneously implanted left atrial appendage occluder (PLAATO) was observed 2 years after implantation. Because of late developing leakages and thrombi, regular transesophageal echocardiographic examinations should be carried out in patients with occluded left atrial appendages. In view of these complications and the potential important hemodynamic role of the left atrial appendage, the benefit of its occlusion is questionable, and the indications of PLAATO should be strongly reconsidered.

The percutaneous left atrial appendage transcatheter occlusion device (PLAATO) is considered a safe and feasible alternative to oral anticoagulation (OAC) in atrial fibrillation (AF) patients with contraindications to OAC.<sup>1</sup>

In March 2005, a 74-year-old female with paroxysmal AF, arterial hypertension, and two ischemic strokes with left hemiparesis (in 1989 and 1990), underwent left atrial appendage (LAA) occlusion by a PLAATO system. This procedure had to be performed since the patient did not tolerate OAC, which resulted in hair loss as well as her risk for stroke/embolism being considered as high. LAA occlusion was successful, as documented by transesophageal echocardiography (TEE) and dye injection into the left atrium during the procedure. Postprocedural cranial computed tomography showed no signs of recent ischemia. The patient was released with a therapy of 100 mg aspirin, 75 mg clopidogrel, 20 mg pantoprazole, 4 mg tizanidine, 5 mg bisoprolol, and 10 mg enalapril. After 2 months, TEE showed complete sealing of the LAA without any thrombus on the device. Clopidogrel was discontinued and aspirin 100 mg/day continued.

In March 2007, the patient was readmitted because of chest pain. ECG showed AF but no signs of ischemia. Neither blood tests nor perfusion scintigraphy showed signs of myocardial ischemia, why her chest pain was assumed to be musculoskeletal. There were no signs of infection as per the patient's history, clinically, or through laboratory investigations. Chest pain decreased after analgetic drugs

and physiotherapy. AF converted spontaneously to sinus rhythm. Clinical neurologic investigation showed postural tremor, intention tremor, dysdiadochokinesia, weakness for hip flexion (M5-) and foot extension (M4-), bilateral foot deformity (Friedreich foot), reduced Achilles tendon reflexes, and atactic stance. Cerebral magnetic resonance imaging (MRI) showed spotted small hyperintense lesions subcortically and in the frontal and parietal lobes, referring to microangiopathy. Friedreich ataxia and polyneuropathy were suspected.

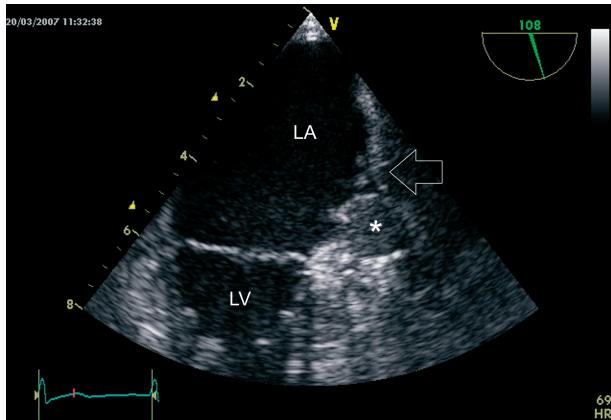
TEE showed a structure measuring 6 × 2 mm in the niche between the LAA wall and the PLAATO device, highly suggestive of a thrombus (Figure 1). Additionally, by color-Doppler, a small jet between the LAA wall and the PLAATO device was detected (Figure 2). Cardiac MRI failed to visualize the jet and the suspected thrombus, due to artifacts deriving from the PLAATO device and the small size of the thrombus (Figure 3). The patient consented for OAC treatment and the antithrombotic therapy was changed from aspirin to phenprocoumon.

These images show that thrombi in AF may develop outside the occluded LAA despite aspirin therapy. Furthermore, leakage of an initially sealed LAA may occur even after 24 months. Cardiac MRI is not an adequate tool to exclude these complications because of the complex anatomy of the LAA and artifacts arising from the device.<sup>2</sup>

It is unknown if there is a common pathogenesis of leakage and thrombus formation. The frequency of asymptomatic late leakage and atrial thrombi in PLAATO

### Address for correspondence:

Claudia Stöllberger, MD  
Steingasse 31/18  
A-1030 Wien, Austria  
claudia.stoellberger@chello.at



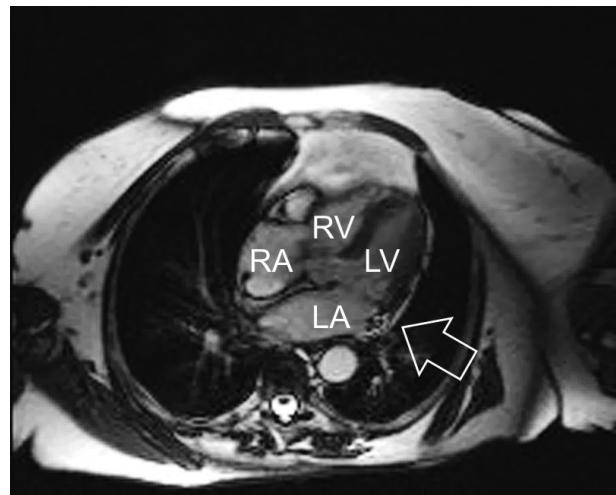
**Figure 1.** Transesophageal picture of the left atrium (LA), left ventricle (LV) showing the percutaneous left atrial appendage transcatheter occlusion (PLAATO) device within the left atrial appendage (asterisk) and a small thrombus at the junction between the left atrium and the left atrial appendage (arrow).



**Figure 2.** Transesophageal picture of the left atrial appendage and the percutaneous left atrial appendage transcatheter occlusion (PLAATO) device showing a small jet by color-Doppler sonography (arrow) between the PLAATO device and the left atrial appendage wall.

is unknown, since TEEs have been carried out only up to 6 months after implantation.<sup>1</sup> Incomplete LAA occlusion, however, is not only a problem of percutaneous techniques. Incomplete LAA occlusion has been found by TEE in 38% of patients who underwent endocardial suture ligation of the LAA during mitral valve surgery.<sup>3</sup>

To our knowledge, chest pain has not been described as a clinical manifestation of incomplete LAA closure. Chest pain in the presented patient was most probably due to musculoskeletal causes. Leakages can be explained by



**Figure 3.** Cardiac magnetic resonance four-chamber view showing the percutaneous left atrial appendage transcatheter occlusion device (arrow) in the left atrial appendage.

properties of the LAA, which may impede long-term patency of occlusion. The LAA myocardium has a higher distensibility than the left atrial myocardium, and progressive dilation of the LAA occurs over time in AF.<sup>2</sup> Dilation of the LAA may lead to splitting of struts or sutures from the LAA wall.

The neurological deficits of our patient were rather related to Friedreich ataxia than to thromboembolism due to the left atrial thrombus. Cardiac involvement in patients with Friedreich ataxia manifests as left ventricular hypertrophy and arrhythmia.<sup>4</sup> Whether AF in the presented patient was caused by long-standing hypertension or by cardiac involvement in Friedreich ataxia is uncertain.

Because of late-developing leakages and thrombi, regular TEE examinations should be carried out in patients with occluded LAA. In view of these complications and the potentially important hemodynamic role of the LAA, the benefit of LAA occlusion is questionable, and the indications of PLAATO should be strongly reconsidered.<sup>2</sup>

## References

1. El-Chami MF, Grow P, Eilen D, Lerakis S, Block PC. Clinical outcomes three years after PLAATO implantation. *Catheter Cardiovasc Interv.* 2007;69:704–707.
2. Stöllberger C, Schneider B, Finsterer J. Elimination of the left atrial appendage to prevent stroke or embolism? Anatomic, physiologic, and pathophysiologic considerations. *Chest.* 2003;124:2356–2362.
3. Katz ES, Tsiamtsiouris T, Applebaum RF, et al. Surgical left atrial appendage ligation is frequently incomplete: A transesophageal echocardiographic study. *J Am Coll Cardiol.* 2000;36:468–471.
4. Ribai P, Pousset F, Tangy ML, et al. Neurological, cardiological, and oculomotor progression in 104 patients with Friedreich ataxia during long-term follow-up. *Arch Neurol.* 2007;64:558–564.