Editorial comment

Percutaneous left atrial appendage transcatheter occlusion as an alternative to oral anticoagulation in patients with atrial fibrillation. Is it the time?

Antonio Di Chiara, Guglielmo Bernardi*

*Coronary Care Unit, *Catheterization Laboratory, S. Maria della Misericordia Hospital, Udine, Italy*

(Ital Heart J 2005; 6 (5): 418-419)

The opinions expressed in this editorial comment are not necessarily those of the Editors of the Italian Heart Journal.

Address:

Dr. Antonio Di Chiara

U.O. di Cardiologia
A.O. S. Maria
della Misericordia
Piazzale S. Maria
della Misericordia, 15
33100 Udine
E-mail:
dichiara.antonio@
aoud.sanita.fvg.it

Atrial fibrillation (AF) is the cause of a significant proportion of strokes, particularly in the elderly. Several randomized clinical trials demonstrated that oral anticoagulants are effective in preventing stroke in patients with AF and that the benefit is higher in the presence of risk factors (clinical and morphological) favoring cardioembolic strokes[1]. Despite the proven efficacy, the effectiveness of oral anticoagulation is hampered by the need of continuing monitoring, the scarce adherence to therapy in terms of correct INR, and by the underprescription due to the fear of serious bleeding. As a result a significant proportion of elderly patients who might benefit most are also those at higher risk of hemorrhagic complications[2].

In the present issue of the Journal, Ramondo et al.[3] present an attractive non-pharmacological alternative to reduce the risk of cardioembolic stroke in patients with non-rheumatic AF, not candidate for oral anticoagulation therapy: the occlusion of the left atrial appendage by means of the percutaneous insertion of an occluder (Fig. 1). In the setting of a multicenter randomized trial (PLAATO), the implant of the left atrial appendage occluder was performed in 4 patients with non-rheumatic AF at high risk of cardioembolic events and a clear contraindication to oral anticoagulants. In this experienced Center, the procedure was uncomplicated, without device-related sequelae at the short-term follow-up.

The rationale of closing left atrial appendage has a robust background, since it excludes the primary source of cardiac embolism. However, although most ischemic strokes and systemic arterial occlusions in AF patients are generally attributed to thromboembolism originating from the left atrium, its pathogenesis remains complex, and up to 25% of AF-associated strokes may be due to intrinsic cerebrovascular disease, other cardiac sources of embolism, or atheromatous pathology in the proximal aorta. Moreover, in a significant proportion the pathogenesis is unclear[1].

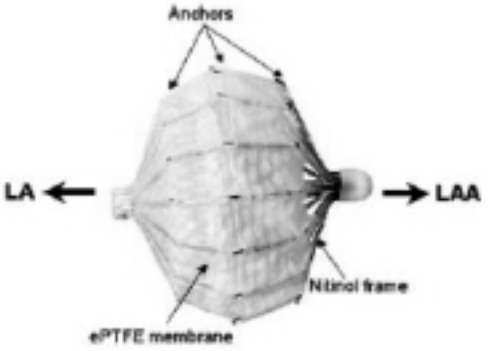

Figure 1. *The implant consists of a nitinol frame covered with an implant occlusion membrane made of laminated expanded polytetrafluoroethylene (ePTFE). Small anchors along the frame and passing through the occlusion membrane assist with device anchoring. LA = left atrium; LAA = left atrial appendage. From Sievert et al.[4], modified.*

In the experience of Ramondo et al., the PLAATO device was implanted in patients with a definite and clear contraindication to oral anticoagulants. However, in the first series of Sievert et al.[4], 11 patients (out of 15) were considered not suitable for oral anticoagulants due to previous gastrointestinal bleeding or unstable INR, in our

opinion reasons not configuring an absolute and permanent contraindication, and nonetheless all patients were placed on aspirin 300 mg indefinitely and clopidogrel 75 mg for 6 months.

With reference to safety and feasibility, in the latter study the procedure was complicated by a hemopericardium in 2/31 patients, and the device was removed and exchanged for re-sizing in 4/16 patients.

This new technological opportunity should be considered as intriguing for its pathological hypothesis, and potentially useful for those patients not otherwise treatable. Before the introduction into clinical practice, we deem mandatory to wait the results of the ongoing clinical trial (PLAATO) in terms of safety and efficacy. Moreover, the correct selection of patients is of utmost importance, due to the complexity of stroke mechanism, especially in high-risk patients, in order to avoid the risk of depriving a patient from a therapy with proven efficacy. It would also be advisable that these patients be enrolled in a randomized study, since even if a procedure is based on a strong rationale, it might turn out to be ineffective. This recently happened with distal protection and thrombus removal during angioplasty in the setting of acute myocardial infarction[5], which should not be any longer performed, at least on a routine basis.

References

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