Letters to the Editor

Silent Embolism After Electrical Cardioversion of Atrial Fibrillation: What Does Brain Magnetic Resonance Imaging Provide?

Embolias silentes tras cardioversión eléctrica de fibrilación auricular: ¿qué aporta la resonancia magnética cerebral?

To the Editor,

Maintaining sinus rhythm (SR) in patients in atrial fibrillation (AF) with electric cardioversion is very common practice applicable to a broad range of patients.¹ In a small percentage of patients, reverting from AF to SR is associated with embolic events both due to mobilization of a pre-existing atrial thrombus or formation of a de novo thrombus. Anticoagulation therapy is therefore usually given to patients not only before but after cardioversion. Thus, a period of prior anticoagulation therapy of 3 to 4 weeks followed by 4 weeks of anticoagulation therapy after cardioversion reduces the risk of clinical embolism to less than 1%.² One aspect that has not been assessed in any detail but that nevertheless has long-term prognostic implications is the production of nonclinical, silent cerebral embolism after reverting to SR. Magnetic resonance imaging techniques are sensitive for identifying small cerebral lesions and also, through studies before and after the intervention, such techniques can establish a causal relationship between the onset of cerebral lesions and the intervention. This aspect is topical, particularly regarding procedures of AF ablation, in which a significant incidence of silent cerebral lesions has been observed after the procedure, although this incidence varies according to the techniques used.³ These lesions largely disappear in the first few weeks,⁴ especially small lesions, but the long-term clinical significance of these findings is as yet unknown.

In the study by Vázquez et al,⁵ the authors assessed the possible production of silent cerebral embolism after electric cardioversion in a series of 62 patients with persistent AF. They performed a magnetic resonance imaging study immediately before and 24 h after the procedure. In their series, the authors state that electric cardioversion can be performed safely, as they did not observe new-onset silent embolisms. The authors claimed that this was due in part to appropriate anticoagulation therapy, although they only provide a single mean international normalized ratio value, which is presented in the abstract. However, this study did not take into account an essential aspect: most embolisms after cardioversion do not occur within the first 24 h but rather in the first few days after reversion to SR. There is no direct casual relationship with reversion to SR itself, but rather an association with the moment when the recovery of atrial contractile function observed after cardioversion occurs (this is when there is greatest propensity to mobilize pre-existing thrombi and the end of the phase when formation of new thrombi can occur, favored by the mechanical dysfunction and subsequently mobilized by the recovery of contractile function). The duration of atrial contractile dysfunction is in part dependent on the duration of AF; thus in patients whose state of AF lasts less than 48 h, proper atrial function is usually recovered in the first 24 h after cardioversion, but in patients with longer standing AF, such as those studied in the aforementioned article, atrial contractile dysfunction and the precoagulation state can extend as long as one month after cardioversion.^{6,7} For these reasons, an appropriate assessment of the potential production of silent embolisms after electric cardioversion should include an imaging study to assess the period of greatest embolic risk, and not just the first 24 h, before being able to affirm that this technique is not associated with silent embolisms, whose prognostic significance, should they occur, would be worthy of subsequent studies.

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