Ablating Brain Embolism in Atrial Fibrillation

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Atrial fibrillation (AF) is widely recognized as the most common cause of cardioembolic stroke in those aged >55 years. Less well appreciated are silent embolic infarcts and their impact on cognitive decline and vascular dementia. Hence, the prevention of both clinical and silent brain infarcts is the primary goal of effective AF treatment. Indeed, entire industries have been spawned to prevent stroke in AF.

Warfarin is established as the benchmark stroke prevention treatment, although its risks and limitations are well known, not the least of which is underuse by physicians in patients with AF. Now physicians will have more effective, safer, and more convenient antithrombotic choices, including the direct thrombin inhibitor dabigatran and (probably) soon factor Xa inhibitors, including rivaroxaban and apixaban. Nonpharmacological means to prevent brain embolism in patients unable or unwilling to use antithrombotic agents including left atrial appendage devices and embolism diversion devices are also under investigation.

Perhaps paradoxically, the role of converting AF to sinus rhythm is controversial. Traditionally patients were often converted to sinus rhythm pharmacologically or electrically. However, when the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial indicated that rate control was as effective as rhythm control for preventing stroke in AF, there became less emphasis on converting patients to sinus rhythm. As well, the AFFIRM stroke substudy indicated that patients with AF should remain on warfarin indefinitely even when apparently converting to sinus rhythm because they often cannot tell when they are fibrillating.

As indicated in the timely review by Haeusler et al, the role of catheter ablation in stroke prevention in AF is even less clear than traditional cardioversion. Catheter ablation is rarely the first option for stroke prevention, although it may be considered in the occasional patient unable to use antithrombotic therapy. Ablation itself carries an approximately 1% risk of periprocedural stroke and may be associated with an even higher risk of silent brain embolism. Strategies for reducing periablation embolism should therefore be a major goal, but the risk varies by patient selection, procedural anticoagulation regimen, and ablation technique, none of which have been standardized. Although not addressed by the authors, intra-arterial thrombolysis has been safely used in patients with periablation brain embolism and most periablation embolic strokes appear to be relatively mild in severity. Finally, permanent procedural success rates and the need for long-term antithrombotic therapy remain uncertain. Hopefully the ongoing randomized trials will provide more insight into these important issues. Until such data are available, cardiac interventionalists and stroke neurologists will find the review of Haeseler et al useful in the management of patients undergoing catheter ablation for AF.

Disclosures

None.

References


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