Silent Cerebral Embolism After Catheter Ablation for Atrial Fibrillation
– Unresolved Issue or Too Much Concern? –

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The role of radiofrequency catheter ablation in the management of atrial fibrillation (AF) has been continuously increasing as a reflection of the fact that the effect of antiarrhythmic medication is relatively poor and that the procedure can not only eliminate symptoms but also improve the mortality rate of the patients. Although the results of a large prospective study have not been revealed yet, it is generally believed that AF catheter ablation will improve the quantity and quality of the patients’ lives.

It is well known that AF catheter ablation is infrequently associated with the occurrence of thromboembolism (TE), with an incidence of 0.94% reported in a 2005 worldwide survey. Despite several methodological developments, including the continuation of vitamin K antagonist (VKA) oral anticoagulation all through the ablation process, keeping the activated clotting time >300 s with periodic measurement every 20–30 min etc, the incidence of clinically symptomatic TE has been constant at approximately 1.0%.

Recently, there has been increasing awareness of silent or asymptomatic cerebral embolism (ACE) following catheter ablation treatment of AF, detected by brain diffusion-weighted magnetic resonance imaging (MRI). In the pioneering work by Gaita et al, the incidence of clinically apparent stroke and ACE was reported as 0.4% and 14%, respectively. The accumulated data so far demonstrate that the incidence of ACE ranges from 1.7 to 38%. This greater incidence of ACE compared with symptomatic stroke seems to suggest a hidden risk of overt TE accident and the interest of ablationists has been focused on reducing the incidence of ACE following AF catheter ablation.

The predictors of ACE have been reported to include the presence of SEC (spontaneous echo contrast), procedure duration before heparin injection, older age, and ablation protocol (CFAE ablation). On the other hand, it has been suggested that silent cerebral ischemia is not a specific finding in AF ablation but a relatively common or unavoidable events in various invasive procedures. A more aggressive anticoagulation protocol to reduce ACE in AF ablation might raise another risk of major bleeding. It is unclear to what extent we should be concerned with this issue.

In this issue of the Journal, Nakamura et al report on the predictors of silent cerebral ischemic lesions (SCILs) among patients treated with 5 types of oral anticoagulants (warfarin and 4 NOACs [non-VKA oral anticoagulants]) and undergoing catheter ablation for AF. Their retrospective study demonstrated a total of 45 SCILs in 40 cases (14% of 286 cases), which were independently associated with both long-standing AF and dabigatran use. Although factors relating to SCILs have been previously reported, this is the first report showing the difference in risk for SCILs among the anticoagulation regimens. Selection bias, as the authors describe, can affect this type of observational study; however, the unique action of direct thrombin inhibitors compared with the other 3 factor Xa inhibitors might be related to the risk if SCILs and further clinical studies are strongly required.

Another outstanding feature of this study by Nakamura et al is the results of follow-up MRI. Among 49 SCILs, 92% disappeared and 8% developed into chronic cerebral infarcts, which was shown to have a larger in diameter than other transient lesions. It will be of interest which clinical factors are associated with the development of chronic infarcts and future investigations with larger numbers of cases are needed.

Lastly, I wonder what is the ideal anticoagulation regimen for the ablation of AF. Procedures under a therapeutic INR level with VKA have now been established as the gold standard, but procedures with the use of interrupted NOAC

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<th>Table. Incidence of ACE in Various Procedures Detected by Diffusion-Weighted Magnetic Resonance Imaging</th>
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<td>Procedure</td>
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<td>Coronary angiography (femoral)</td>
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ACE, asymptomatic cerebral embolism. (Quoted from Bendszus M, et al.)
treatment are rapidly increasing, showing similar efficacy and safety as for those under VKA treatment. As for the continuous use of NOACs during the procedure, we should be prudent in considering whether it is really necessary, because rapid interruption and reinstating of therapy is the unique advantage of NOACs compared with VKA.

Disclosures

References