# Direct oral anticoagulants as thromboembolic prophylaxis after catheter ablation of ventricular tachycardia: Not only safe and effective

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**Editorial**

**TITLE**

Direct oral anticoagulants as thromboembolic prophylaxis after catheter ablation of ventricular tachycardia: Not only safe and effective

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Recently, warfarin has been supplanted by direct oral anticoagulants (DOACs) for many indications. DOACs are preferred because of their ease of use, lack of need for monitoring, and greatly reduced concern about drug-drug interactions. However, there is clear evidence from patients with mechanical heart valves, rheumatic valve disease, and antiphospholipid antibody syndrome that warfarin is preferred to DOACs because of its improved safety and/or efficacy.

After left ventricular endocardial ablation for ventricular tachycardia (VT), especially after extensive lesions, patients are at high risk for thromboembolism. The options for prophylactic anticoagulation therapy are antiplatelet or anticoagulant therapy including warfarin or DOACs (Table). However, the 2019 American College of College/Heart Rhythm Society VT consensus document only mentions antiplatelet agents and warfarin. [1] There are insufficient data to determine the superiority of these drugs in terms of the safety and efficacy when used as thromboprophylaxis after left ventricular ablation.

Siontis et al. reported the safety of warfarin and low-dose heparin bridging for thromboembolic prophylaxis after left ventricular ablation.[2] Further, the patients enrolled in the Thermocool VT ablation study, which was not a randomized trial, received either warfarin (if ablation was performed in a >3-cm area) or aspirin 325 mg, and there were no thromboembolic events in either group.[3]

On the other hand, the STROKE-VT (Safety and Efficacy of Direct Oral Anticoagulant Versus Aspirin for Reduction of Risk of Cerebrovascular Events in Patients Undergoing Ventricular Tachycardia Ablation) trial showed that the use of DOACs (including dabigatran, rivaroxaban, and apixaban) was associated with a lower incidence of a postprocedural stroke, transient ischemic attack, and asymptomatic brain magnetic resonance imaging lesions as compared to aspirin (81 mg).[4] However, no study has directly compared warfarin and DOACs as anticoagulants after VT ablation.

In this issue of the Journal of Cardiovascular Electrophysiology, Deshmukh et al. compare the safety and efficacy of warfarin and DOACs as anticoagulants after VT ablation in a single-center retrospective cohort study.[5] This study included 80 consecutive patients with structural heart disease who underwent left ventricular endocardial ablation for VT between 2016 and 2021, including 38 consecutive patients who received post-procedure anticoagulation with warfarin and 42 consecutive patients who received treatment with DOACs. In the warfarin group, anticoagulation was bridged with intravenous unfractionated heparin during hospitalization and low-molecular-weight heparin after discharge until the therapeutic INR range (2-3) was achieved. In the DOAC group, treatment with the DOACs was initiated as early as 6 hours after hemostasis, provided there was no inguinal hematoma. The mean age was 66.2 ± 11.7 years, 91% were male, 64% had ischemic cardiomyopathy, and the mean left ventricular ejection fraction was 32 ± 14%. The baseline characteristics were similar between the two groups. The majority of DOACs used in this study were apixaban (88%). The majority of the left ventricular access for mapping and ablation was via a retrograde aortic access (91%). The duration of the radiofrequency energy delivery (108±50 minutes vs. 113±48 minutes, p>0.05) and procedure times (412±100 minutes vs. 472±110 minutes, p>0.05) were similar between the patients treated with DOACs and warfarin, and arterial closure devices were generally used only in patients scheduled to receive DOACs after ablation. Thrombotic events occurred one day after ablation in one patient in the DOAC group, which was a right branch retinal artery occlusion, and one day after ablation in one patient in the warfarin group, which were iliac deep vein and right atrial thrombi. All bleeding complications were vascular access-related hematomas and were comparable between the two groups (occurred in 2 [4.8%] patients in the DOAC group and in 6 [15.8%] in the warfarin group, p=0.2). The post-ablation hospital stay was shorter in the DOAC group than in the warfarin group (3.3±1.8 vs. 5.0±2.5 days, p=0.001).

Although this study is not a randomized trial, it is significant in that it shows that the use of DOACs after VT ablation is as safe as a protocol with a slowly escalating regimen of unfractionated heparin followed by 3 months of therapeutic anticoagulation with warfarin. Because of the rapid onset of therapeutic anticoagulation with DOACs as compared to warfarin, there was concern that DOACs would increase bleeding complications, but the use of vascular closure devices in the DOAC group successfully reduced the incidence of vascular access-related hematomas. If vascular closure devices were also used in the warfarin group, it is likely that the incidence of vascular access-related hematomas would have been as low as in the DOAC group.

This study also showed that anticoagulation with DOACs after catheter ablation of ventricular tachycardia was not only as safe and effective as warfarin for thromboembolic prophylaxis, but also resulted in shorter length of the hospital stay after ablation than warfarin. Reduced bleeding events and avoidance of anticoagulation titration in patients treated with DOACs allowed for shorter hospital stays. The additional cost associated with the use of vascular closure devices in the DOAC group can be considered a necessary expense to reduce the length of the hospital stay.

This new evidence for the choice of a DOAC as a means of preventing thrombosis after VT ablation is clinically beneficial.

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