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EDITORIAL

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Editorial to "The differences of atrial thrombus locations and variable response to anticoagulation in non-valvular atrial fibrillation with ventricular cardiomyopathy"

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in adults. It contributes to cardioembolic stroke that in turn causes disability and great economic burden. During AF, the atrial cavities become static and contract irregularly. This may lead to cardiogenic thromboembolism. The left atrial appendage (LAA) is the remnant of the embryonic left atrium and is the most common site for blood clot formation. This is usually manifested by spontaneous echocardiographic contrast, decreased LAA peak flow velocity and LAA fibrosis.[1] In the era of nonvitamin K-dependent oral anticoagulation (NOAC) usage for the prevention of AF-related thromboembolism, the prevalence of LAA thrombus formation can be up to 4%. This is similar to the use of vitamin K anticoagulants (VKA).[2] In patients with embolic stroke of undetermined source, atrial cardiopathy was hypothesized to be the cause of embolization in the absence of AF. Thus, the interplay between LAA, AF, and atrial cardiopathy has been considered as the principal factor leading to cardioembolic events.

Many studies have investigated the predictors of LAA thrombus formation, including CHA2SD2-VASc score, severe impaired systolic function, type of AF, LAA morphology, and LA size.[2] However, only few studies examined the efficacy in resolving the LAA thrombus. Adam et al consecutively reviewed 2106 transesophageal echocardiogram (TEE) cases, and found that 21% of these cases had a detectable thrombus despite being appropriately anticoagulated.[3] There was no statistically significant difference in the rate of LAA thrombus resolution with regard to the use of VKA or NOAC. However, the history of diabetes mellitus (DM) was the only predictor of LAA thrombus persistence in this study.[3] Dominik et al examined the clinical and echocardiographic determinants of LAA thrombus resolution in 78 patients and found no differences in left ventricular ejection fraction (LVEF), LAA velocity, thrombus diameter, and LA diameter between responders and nonresponders.[4] There was also no difference between the use of VKA and NOACs in the proportion of thrombus resolution. Interestingly, in the NOACs group, the time taken to reach full resolution was shorter compared to VKA (81 ± 38 days vs 129 ± 46 days, $P = .03$).[4]

Zhang et al retrospectively examined the locations of atrial thrombus and the determinants of thrombus resolution in response to anticoagulant therapy in 191 patients with AF and atrial thrombosis.[5] These patients were further divided into ventricular cardiomyopathy (VCM) and non-VCM subgroups.[5] The mean CHA2DS2-VAS score was 2.7 in the non-VCM and 3.0 in the VCM group. Although 57% of non-VCM patients and 28% of VCM patients in this study were being anticoagulated, the presence of atrial thrombi could still be detected, indicating that there still remains an unignorable residual risk of thromboembolism in AF patients undergoing anticoagulant therapy. After atrial thrombosis was confirmed, all patients were administered anticoagulants; 55% of them took VKA. Before propensity matching, LVEF was significantly lower in the VCM group (70% ischemic cardiomyopathy) compared to the non-VCM group (49% vs 60%). After propensity score matching, LVEF became similar in both groups (57% vs 56%). However, the composition of VCM was not mentioned in the article. Therefore, the influences of different types of VCM on thrombus resolution remain unclear. Interestingly, the authors found that VCM, not LA size, LVEF, or DM, is an independent predictor of thrombus persistence in multivariate analysis. This finding adds new information to our current understanding of anticoagulant therapy and atrial thrombus resolution. They also reported the presence of 30% extra-LAA thrombi in VCM, independent of LA diameter in the subgroup analysis. However, only 28% of VCM patients were receiving anticoagulants before thrombus detection, which may have contributed to the formation of extra-LAA thrombus. Additionally, the thrombus resolution time was 20 and 30 weeks in the non-VCM and VCM groups, respectively. This was much longer than the resolution times reported in previous studies (11 weeks in the NOAC group and 17 weeks in the VKA group).[4] In the current study, Zhang et al[5] used multiple modalities including TEE, cardiac computed tomographic angiography, cardiovascular magnetic resonance imaging, or transthoracic echocardiography instead of using a unique tool, to detect the presence or resolution of atrial thrombus, which may also render the comparisons across the different subgroups of patients difficult. Nevertheless, the results suggest that the interaction of VCM and atrial remodeling plays an important role in the formation of the thrombus. While the study was limited by the design and background, the findings remind us that atrial thrombosis does not only result from an atrial disease but also occurs because of ventricular disease. It is therefore imperative to carry out more studies to examine the hypothesis.

CONFLICT OF INTEREST

The authors declare no conflict of interests for this article.

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