LETTERS TO THE EDITOR

doi:10.1093/ehjci/jes231
Online publish-ahead-of-print 6 November 2012

Left ventricular mass as a discriminator of left atrial appendage thrombus in persistent atrial fibrillation: promise or over-enthusiasm?

We have read with enthusiasm the paper by Boyd et al.1 concerning a left ventricular (LV) mass as a predictor of left atrial appendage thrombus. These authors have shown in a population of 156 patients with persistent atrial fibrillation undergoing transoesophageal echocardiogram (TOE) that an indexed LV mass had a highly discriminative capability for the prediction of left atrial appendage thrombus: receiver operator characteristic curves for the prediction of thrombus displayed values of the area under the curve (AUC) for the LV mass of ~0.98. This was far better than what was observed with spontaneous echocardiographic contrast (AUC: 0.73–0.75), a parameter that has long been known for its association with the presence of a thrombotic milieu in the left atrium. In multiple logistic regression analysis, an indexed LV mass was also the only independent predictor of thrombus.

The usefulness of transthoracic echocardiography-derived parameters in thromboembolic risk assessment has been overlooked for long. However, the left ventricle systolic function has been included in the CHA2DS2-VASc score and recent evidence has supported the additive value of left atrial dimensions (area and volume)2 in the prediction of surrogate TEE endpoints and the identification of those patients who could potentially be spared TEE before cardioversion3 or percutaneous ablation of atrial fibrillation.

Left ventricular hypertrophy, a marker of an increased LV mass, has also been proposed in the past as a possible risk factor for stroke in AF patients.4 Still, in subsequent systematic reviews (comprising studies conducted in the 1980 and 1990’s), the only transthoracic echocardiogram parameter that was considered useful for the prediction of cerebrovascular events was the presence of moderate-to-severe left ventricle dysfunction.5,6

Nonetheless, despite very promising, the results of Boyd et al. are preliminary and must not be looked with over enthusiasm as there are some caveats that must be clarified with further investigations.

There may be a selection bias in this population, since the 35 consecutive patients with thrombus were collected during a 7-year period (between August 1999 and April 2005) and the population of the control group was gathered only until June 2003. Furthermore, despite the absence of data concerning the average CHADS2 and CHA2DS2-VASc scores of the population, the fact that only 40–51% of subjects had a CHADS2 score ≥2 and the average left atrial volume was ~41.7 ± 11.3–43.5 ± 16.6 mL/m² shows that these patients had a low risk of thromboembolic events (almost 67% of subjects in the RE-LY trial7 and 100% of patients in the ROCKET-AF trial8 had a CHADS2 score ≥2) and did not present very diseased atria. Conversely, the average LV ejection fraction is ~42–43%, which seems very low for such a low-risk population, with not very dilated left atria.

We think that the lack of association in this study between parameters that have previously shown across studies to be discriminative of the presence of thrombus, like left atrial volume and LV ejection fraction, or the moderate association of spontaneous echocardiographic contrast, may be due to the lack of statistical power of the small study sample.

Additionally, the authors claim that LV hypertrophy is not present in all hypertensive patients. Furthermore, they say that an increased LV mass results in LV diastolic dysfunction and left atrial enlargement. Therefore, since hypertension was present in only 54–63% of patients (and assuming, as the authors state that not all will develop LV hypertrophy) and left atria were not significantly enlarged, it is difficult to understand how the LV mass could so accurately discriminate the presence of left atrial appendage thrombus.

The ongoing ENGAGE-AF TIMI 48 trial9 echocardiographic substudy will probably shed a light into the role of echocardiographic parameters derived from transthoracic echocardiogram in the risk stratification of atrial fibrillation.

Conflict of interest: none declared.

References


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