The role of echocardiography in stroke risk assessment in patients with atrial fibrillation: is it additive or does it simply echo clinical risk factors?

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This editorial refers to ‘Possible refinement of clinical thromboembolism assessment in patients with atrial fibrillation using echocardiographic parameters’ by R. Providência et al., on page 36

Stroke risk assessment is key when determining the appropriate long-term antithrombotic therapy in patients with atrial fibrillation (AF). Over time many stroke risk stratification schema have been developed, initially with the objective to better define ‘high-risk’ patients who could be subjected to an inconvenient (and potentially dangerous) drug, warfarin.1

Current European guidelines recommend the simple and commonly used CHADS2 as an initial assessment, which is then refined with a newer and more comprehensive assessment, the CHA2DS2-VASc score.1,2 The latter includes additional common clinical risk factors, and has shown to be better than the CHADS2 score in identifying ‘truly low-risk’ patients (who do not need any antithrombotic therapy) and is at least as good as—and possibly better than—the CHADS2 score in identifying ‘high-risk’ patients.3 Still, there may be sufficient room for improvement, given that some patients may have associated stroke risk factor on imaging, and indeed, echocardiography may even refine stroke risk stratification.4

In this issue of Europace, Providência et al.5 explore the potential benefit of adding echocardiography to the contemporary clinical stroke risk schema, CHADS2 and CHA2DS2-VASc. Their cross-sectional study consisted of 405 consecutive AF patients who underwent both transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) without a significant time delay. After excluding patients with mitral valve stenosis or a prosthetic heart valve the remaining study population predominantly (352/376, 94%) consisted of patients undergoing echocardiographic evaluation as part of standard care prior to direct current cardioversion of AF. Transesophageal echocardiography was used to identify surrogate endpoints being markers associated with thromboembolism4: left atrial dense spontaneous echo contrast (SEC) and the presence of left atrial thrombus (LAT) or low flow velocities (LFV) in the left atrial appendage. Transesophageal echocardiographic features of interest were moderate-to-severe left ventricular dysfunction and a dilated left atrium as determined by left atrial area measurement using the apical four-chamber view. Importantly, the authors observed that in contrast to those with a CHADS2 score = 0, not a single patient with a CHA2DS2-VASc score = 0 demonstrated any of the TEE markers of increased risk of stroke. When it comes to predicting the presence of these left atrial abnormalities on TEE both clinical schema perform only modestly (AUC 0.54–0.63), but this was only a relatively small-sized cohort for this sort of analysis. However, the addition of above-mentioned TTE parameters improved the predicative ability for the surrogate endpoints in both cases.

The findings by Providência et al.5 reinforce the important message that CHA2DS2-VASc is superior to CHADS2 in identifying those at truly low risk of stroke. When using CHADS2 a ‘normal’ TTE is required in addition to a score of 0 before the conclusion of a ‘truly low stroke risk’ classification can be made. Anyone unconvinced of the benefit of CHA2DS2-VASc from this perspective (i.e. to identify ‘truly low-risk’ patients) should consider that their patients could possibly be spared from an inconvenient time-consuming procedure by simply applying the CHA2DS2-VASc score.

The observation that both clinical schemas are poor in predicting the presence of left atrial abnormalities on TEE reflects certain limitations. Firstly, imaging is all about timing and thereby provides a ‘snap shot’. The lifespan, so to speak, of a left atrial (appendage) thrombus is unknown. However, if the thrombus ‘disappears’ on its
own it is unlikely that the patients risk of stroke also ‘disappears’ or decreases. This limitation also holds true, but perhaps to a lesser extent, for the dense SEC and LPV in the left atrial appendage as they can differ in time too.

Furthermore, given all presented echocardiographic data by Providência et al.5 are strictly derived during AF, it would be interesting to know whether its significance changes if measured during sinus rhythm. Perhaps the difference between sinus rhythm and AF is of even greater importance.

Secondly, it is important to realize that the increased stroke rate in AF is not fully attributable to cardio-embolism and that other mechanisms are at play.4,6 Hence, AF should really be considered a ‘vascular’ disease and this approach also helps us to understand why stroke risk refinement with CHA2DS2-VASc is successful, considering that commonly encountered cardiovascular risk factors are taken into account with CHA2DS2-VASc compared with CHADS2. Although these clinical risk factors improve the overall stroke risk stratification of AF patients, many of them do not (fully) relate to true cardio-embolic stroke. Therefore, the poor predictive power of the presence of localized—cardiac/left atrial—abnormalities for both clinical schema should not come as a big surprise as, for this matter, they are surrogates of what is going on in the left atrium.

The observation of a beneficial effect of adding TTE parameters—that is, moderately to severely depressed left ventricular function and a dilated left atrium—to both clinical schema is therefore plausible and less surprising. In fact, it exemplifies the power of echocardiography—or any imaging modality for that matter—which is to visualize the scene of calamity: in this case, the left atrium.

This raises the question: ‘What and how to visualize best?’ Whether left ventricular dysfunction, as suggested by the authors, will prove to be the most important (transhilaric) echocardiographic parameter, needs to be seen. Perhaps this is also a surrogate marker of the concomitant occurrence of left atrial pressure and/or volume overload that potentially triggers (pro-thrombotic) atrial changes. Increased atrial dimensions or more specifically increased atrial volume over time is bad news in general and associated with increases risk of stroke in particular.7 However, it is important to realize that most of the available evidence concerns ‘simple’ quantification of the left atrium. Such measurements are outdated and there is a lot to be gained if they are at least expanded, as the authors did, with left atrial area measurement using the apical four-chamber view and, preferably, volumes.

Perhaps cardiac magnetic resonance imaging (MRI) will become the way forward once atrial inflammation and fibrosis can be reliably quantified.9 Going even one step further, we could even consider performing a cerebral MRI in patients with ‘score 0 or 1’ to reveal clinically silent cerebral infarctions or small vessel disease, and thereby re-classifying them from low- to high-stroke-risk patients. This also creates the possibility to potentially fine-tune existing bleeding risk schema10 by concomitant screening of, for instance, cerebral micro-bleeds and small vessel disease.

In conclusion, the study by Providência et al.5 underlines that we are on the right track with CHA2DS2-VASc when it comes to stroke risk classification. Indeed, it also illustrates the potential added value of imaging modalities, here echocardiography, especially when used for what they do best: obtaining information from the scene of calamity. However, the reported echocardiographic findings by Providência et al.5 need to be validated in larger prospective studies using solid, clinical end points that matter (i.e. stroke, thromboembolism) and include contemporary imaging measurements, as well as net clinical benefit assessments balancing ischaemic stroke against major bleeding (especially intracranial haemorrhage).11 Finally, given the vascular nature of AF, it is also important to note that a lot can be gained by continued efforts to further break down the already-incorporated clinical risk factors, such as diabetes mellitus, and to investigate other potentially relevant ones, such as renal impairment.12

Conflict of interest: G.Y.H.L. has served as a consultant for Bayer, Astellas, Merck, AstraZeneca, Sanofi, BMS/Pfizer, Biotronik, Portola, and Boehringer Ingelheim and has been on the speakers bureau for Bayer, BMS/Pfizer, Boehringer Ingelheim, and Sanofi-Aventis.

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