Letter to the Editor

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Letter by Kosiuk et al Regarding Article, “Implication of Left Ventricular Diastolic Dysfunction in Cryptogenic Ischemic Stroke”
To the Editor:

We read with great interest the article by Seo et al1 that adds new evidence to the discussion on the link between left ventricular diastolic dysfunction (LVDD) and ischemic stroke. In their work on patients with strokes of different origin, including strokes with and without atrial fibrillation (AF), as well as cryptogenic stroke, the authors analyzed the complex relationships among LVDD, cerebral ischemic events, and AF. We were pleased to identify some parallels between their study and our previous work, which we would like to comment on in this letter on thereby further highlighting some important findings.

First, the authors observed virtually an identical LVDD prevalence of 37% in patients with AF when compared with what we have previously described.2 Furthermore, although not reaching statistical significance, the prevalence of severe LVDD in patients with documented AF was higher than in patients with cryptogenic stroke (37.1% versus 27.3%; P=0.104). Considering our previous findings that LVDD is more frequently found in patients with persistent AF,3 we fully agree with the interpretation of the authors that the somewhat lower prevalence of LVDD in patients with cryptogenic stroke might suggest the coexistence of paroxysmal AF, which was not detected by their implemented 24-hour Holter-ECG.

Second, the reported higher prevalence of LVDD in patients with cardioembolism-mimic versus noncardioembolism-mimic cryptogenic stroke (41.4% versus 11.5%; P=0.013) stresses the role of LVDD as a mediator of thromboembolic risk in those patients. Given the fact that LVDD leads to structural and functional remodeling of the left atrium, which is the most frequent source of cardioembolism and also correlates with new onset of AF,3 this hypothesis is most convincing and was also discussed in our previous work.4

Last but not least, LVDD grade II and III were independently related to stroke with AF, emphasizing also the potential prognostic value of LVDD for assessment of individual thromboembolic risk. We have reported results that are in line with the current study: there was an association between LVDD with increased thromboembolic risk expressed by the CHA2DS2-VASc (congestive heart failure, hypertension, age >75, diabetes mellitus, stroke/transient ischemic attack, vascular disease, age >65, sex category) score (R=0.392; P<0.001), and E/e′ as a single echocardiographic surrogate parameter of LVDD was closely linked with individual stroke risk in patients with AF (area under curve, 0.864; P<0.001).4 Those results that are mirrored by this study as the LVDD grade II and III are characterized by the highest E/e′ values.

Finally, a word of caution is needed. Echocardiographic assessment of LVDD in clinical practice may differ from that under study conditions. This is illustrated by a long list of exclusion criteria in this and our previous work, the load-dependency of LVDD and—probably most importantly for this cohort—the actual or previous presence of AF that substantially affects LVDD.

In conclusion, the authors should be applauded for their contribution to this still poorly studied topic. We strongly think that the increasing evidence will alert the medical community, and this problem will gain further clinical importance and will be given an adequate scientific prominence.5

Disclosures

None.

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