Left ventricular wall motion abnormalities (LVWMAs) are commonly observed in a variety of medical conditions, including coronary artery disease, congestive heart failure, stress-induced cardiomyopathy, myocarditis, chronic renal disease, and stroke.1-4 Their underlying disease mechanisms and their potential causative role for stroke remain inadequately elucidated. LVWMAs may directly increase stroke risk through thrombus formation in the left ventricle3 or may merely indicate the burden of systemic atherosclerosis underlying stroke, and not the primum movens.

In this issue of Neurology®, Choi et al.5 report on the association between LVWMAs and long-term stroke recurrence in a cohort of 4,316 Korean patients presenting with acute stroke or TIA. The data originate from the Korea University Stroke Registry, which contains an extensive set of clinical, biochemical, and imaging measures. To evaluate the stroke risk related to LVWMAs independent of cardiac comorbidity, they only included regional or global hypokinetic, akinetic, or dyskinetic left ventricular wall motion abnormalities detected on transthoracic echocardiography, unrelated to acute or recent myocardial infarction, dilated cardiomyopathy, or heart failure with an ejection fraction less than 30%. Using these criteria, 10% of the stroke patients (n = 430) were diagnosed with LVWMAs.

In univariate analysis, LVWMAs associated with older age, arterial hypertension, diabetes mellitus, coronary artery disease, cardiac arrhythmias, cerebrovascular artery stenosis or occlusion, severe stroke, and elevated plasma levels of C-reactive protein, all of which may increase risk of stroke recurrence.6 Yet after adjustment for these possible confounders, patients with LVWMAs had 1.7 times higher risk of recurrent stroke in the first years following the index event compared to those without LVWMAs. Moreover, diverse subgroup analyses demonstrated consistent results, indicating that LVWMAs hold potential as an independent risk factor for stroke recurrence.

The retrospective observational design warrants caution given the risk of selection bias, reporting bias, and incomplete data, for instance with regard to stroke events, paroxysmal atrial fibrillation, or patient adherence to secondary stroke prevention regimens. Still, given the considerable study population and the extensive set of possible confounding factors taken into account, the results may serve as a starting point for more detailed exploration of the pathophysiology underlying LVWMAs in stroke patients. Should LVWMAs be regarded as an indication of unrecognized coronary artery disease, stress-induced cardiomyopathy, or autonomic dysfunction in acute stroke? In this context, LVWMAs may warrant prospective investigation as a temporary phenomenon during the (sub)acute phase of stroke, seeking to establish a relation between LVWMAs and heart rate variability or baroreceptor sensitivity.6 Along the same lines, correlations with concomitant medication and infarct location may be of interest.

Regarding future optimization of secondary cerebrovascular prevention, the remarkably high relative risk of stroke recurrence in patients with LVWMAs who did not receive statins at hospital discharge requires confirmation. Similarly, the effects of antiplatelet treatment and anticoagulation on stroke recurrence (both ischemic and hemorrhagic) is of interest, especially given the possible role that LVWMAs may play in context of the more recently introduced concept of embolic stroke of undetermined source.7

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**DISCLOSURE**

Raf Brouns reports no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

**REFERENCES**


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