Prevalence of Intracranial Atherosclerosis and Coexistent Conditions in Ethnic South Asians

To the Editor:

Although the objectives of their study were not stated, De Silva et al. used clinical data on 200 acute ischemic stroke patients admitted in a hospital in Singapore to determine the prevalence of intracranial large-artery disease (ICLAD) and its risk factors. The study subjects were selected on the basis of their origin of the Indian subcontinent (ethnic South Asians). The authors declared that “ICLAD is the most common vascular lesion in stroke worldwide”. To justify their declaration, they cited a reference (Reference 2 in their article) to an article published in *Int J Stroke*. This publication was not found in PubMed Central. The authors found that 54% of stroke patients had ICLAD and they pronounced it as “high burden of ICLAD among ethnic South Asians” without comparing this prevalence with that among non–South Asian stroke patients. Based on the 54% prevalence, the authors concluded that “intracranial large arteries are the predominant site of disease (atherosclerosis)” and that “these data may be extrapolated to ethnic South Asians living in the urban regions of South Asia and developed countries such as the United Kingdom and the United States”. Large artery disease in 54% of patients can hardly be described as predominantly intracranial. How can the results based on selected 200 stroke patients in a Singapore hospital be generalized to 1 billion urban South Asians in South Asia and elsewhere?

The descriptive results are confusing. Of the 200 patients, 102 had ICLAD and 86 did not have ICLAD. The ICLAD status of the remaining 12 patients was not given. There were some errors in the descriptive data such as the mean age of the study subjects and the proportion of patients who were men. The authors failed to describe the type of regression analysis and the software used in their study. Their analysis found 2 (high erythrocyte sedimentation rate [ESR] and hypertension) of the 5 potential risk factors to be associated with having ICLAD. The authors claimed that the association between hypertension and ICLAD was documented in “previous literature”. However, they cited only 1 reference (Reference 8) to a study of 425 Japanese patients. The authors also cited 1 reference (Reference 9) in support of their finding of high ESR being associated with having ICLAD. It was interpreted as the role of inflammation in atherosclerosis pathogenesis. However, Reference 9 is about C-reactive protein, not ESR. The authors did not provide any explanation for their findings. Why do hypertension and high ESR coexist more frequently with ICLAD? The ESR is raised in acute inflammatory conditions and chronic infections.

In their discussion, the authors stated that “although ICLAD was most prevalent in large-vessel stroke, it was also common among other stroke subtypes” and “ICLAD was either asymptomatic or concurrent with small-vessel stroke”, but their study data cannot be the foundation of these speculative statements. It appears that the medical records of the patients admitted in the hospital with ischemic stroke were the source of the study data. Consequently, the data on hypertension, ESR and other potential risk factors were collected at the same time as the data on ICLAD. Inferences about predictive associations cannot be drawn from such cross-sectional data. For example, the authors cannot assert that hypertension increased or may have increased the risk of ICLAD in ischemic stroke patients because the data were not collected prospectively—measurement of blood pressure and ESR in ICLAD-free persons. The alleged “association” of hypertension and high ESR with ICLAD found in this study has to be mere coexistence of hypertension and high ESR with ICLAD without any causal or effect modification assumption. Consequently, the results of this cross-sectional study are not useful. At best, these results can be used to generate hypotheses for future prospective studies.

Disclosures

None.

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