A Precise Stroke Classification for Evaluation of Ischemic Stroke Subtypes and Their Relation With Diabetes: Is TOAST the Best?

To the Editor:

Regarding the study by Turin et al1 performed to estimate the secular time trends in incidence using a population-based stroke registry in Japan, we discuss some issues of potential interest. First, have the authors classified stroke subtypes according to TOAST classification or Oxfordshire classification? No mention has been made in the text concerning the classification methodology of ischemic stroke. It is conceivable that applying the TOAST classification2 of ischemic stroke (a classification with clinical, pathophysiological, anatomic, and instrumental basis that is easily applicable and widely validated) could provide a more precise selection of patients with lacunar stroke. On this basis, by failing to specify how they classified ischemic stroke subtype, in our opinion, the authors could not exclude an overestimation of lacunar stroke.

Second, authors affirm that a stroke was classified as ischemic stroke when brain imaging revealed acute infarction or showed no evidence of hemorrhage. They also claim that ischemic stroke was further classified as lacunar stroke in patients who present focal neurological symptoms and signs, even if the imaging result was negative. This appears in contrast to the TOAST classification, for which a stroke in the presence of symptoms suggestive of lacunar syndrome should be considered as lacunar, even without infarction signs on imaging. Perhaps the reviewer suggested that the authors had mentioned this study in the text and in references.

Third, similarly, no mention has been made in the text concerning the criteria used to evaluate diabetes prevalence. Previous studies analyzed diabetes prevalence according to World Health Organization criteria because American Diabetes Association criteria have been published in 1997. If they had classified diabetes in accordance with American Diabetes Association (1997) criteria (fasting plasma glucose ≥126 mg/dL), then a higher number of subjects with diabetes would have been detected. The authors do not distinguish among known diabetes, newly diagnosed diabetes, and stress-induced hyperglycemia.

Fourth, authors affirm that cardioembolic infarction required the same criteria as ischemic stroke plus evidence of a possible source of embolus, such as valvular heart disease, atrial fibrillation, or history of acute myocardial infarction. They should not exclude other possible cardioembolic clinical conditions, and, in our opinion, these criteria of classification of cardioembolism (cardioembolic ischemic strokes), lacking a clear reference to specific classification system, do not appear exhaustive.

Fifth, why do the authors refer to atherothrombotic or large artery atherosclerosis strokes with the term nonlacunar? Generally, the term nonlacunar refers to atherothrombotic, cardioembolic, and cryptogenetic strokes.

Sixth, our group,3 even in a case-control study of comparison between subjects with and without diabetes with acute ischemic stroke, previously reported that diabetes is associated with lacunar subtype with a history of hypertension. This finding is suggestive of the anatomopathological background of the diabetic cerebral infarction, as shown by Kane and Aronson,4 who reported a higher incidence of lacunar infarcts detected at autopsy in diabetic subjects who died after an ischemic stroke.

Seventh, the data reported by Turin et al1 in a Japanese population are not completely original findings. Kubo et al5 have already analyzed secular trends in the incidence of ischemic stroke subtypes established in the Japanese community of Hiyama. In this study, age-standardized incidence of lacunar infarction significantly declined by 59% for men and by 28% for women from the first to the second cohort, and it continued to decline by 41% for men. On this basis, it would be interesting if the authors had mentioned this study in the text and in references.

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Stroke. 2011;42:e10; originally published online December 9, 2010;
doi: 10.1161/STROKEAHA.110.602508

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