Letters to the Editor

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Transient Ischemic Attack Etiologic Subtype and Early Risk of Stroke

To the Editor:

Although clinical features help stratify stroke risk after transient ischemic attack (TIA), the novel study by Purroy et al1 on predictors of early recurrent ischemia according to etiologic subtype of the index TIA emphasizes additional variables we must consider in our prognostication algorithms. We wish to discuss some issues raised in the article which we felt merited further exploration.

The study found a very low recurrent event rate in the small-vessel disease (SVD) group (1.5%, or 1 event/68 patients). Transient lacunar events due to SVD are commonly thought to have a relatively benign prognosis, and these results support such an assertion. However, in our experience, lacunar events can often progress after a seemingly transient onset, and others have shown that lacunar TIsAs predict subsequent lacunar strokes.2 Perhaps the variability in prognosis stems from how a lacunar TIA is defined. CT alone would presumably make identification of a causative lesion in the SVD group uncommon, in which case reliance on a clinical “lacunar syndrome” might misclassify the etiology in over 40% of patients.3 This misclassification rate might be even higher when assessing patients with only transient or unobserved symptoms. It has been shown that diffusion-weighted imaging (DWI)-MRI can improve TOAST classification of ischemic strokes, and in particular, events due to SVD.3 Most strokes initially classified as being due to an unknown cause were reclassified following DWI-MRI as being secondary to SVD.3 It is therefore possible that in the study by Purroy et al some patients classified as “undetermined” may in fact have had SVD.

It is also possible that without MRI, an SVD determination relied primarily on the absence of large-vessel disease and a cardioembolic source as opposed to positive imaging findings. Thus, some patients with transient neurological symptoms of nonischemic origin (ie, migraine) may have been labeled SVD, thereby further diluting the stroke risk in this group. Baseline MRI might also be useful because patients with transient symptoms who are DWI-negative appear to have a low risk of subsequent stroke.4

We also found it interesting that clustering of TIsAs within 1 week of the index event did not appear to have any value in predicting the risk of early recurrent ischemia. So-called “ascending TIsAs” are classically cited as an ominous sign of impending stroke, particularly among SVD patients with the “capsular warning syndrome”. Such patients frequently progress within 24 hours, however, and may therefore not have been classified as TIsAs by Purroy et al. This suggests that transient lacunar events are particularly benign if patients are clinically normal by 24 hours. For the acute management of such patients, though, it would be interesting to know the prognostic value of persistent neurological symptoms versus resolved symptoms at the time of first evaluation, which should ideally be well before 24 hours after onset.

Another important finding in this study is the possible ceiling effect of the ABCD2 score. The same proportion of patients in all TOAST groups had an ABCD2 score of ≥5 (P=0.786). Despite this, a differential stroke risk was still found among etiologic subtypes, bolstering the notion that risk stratification after TIA should be further refined by determining TIA etiology. Early and accurate ascertainment of stroke etiology is possible using DWI-MRI and should be similarly feasible in DWI-positive TIsAs.

Purroy et al are to be commended for their study, which underscores the concept that stroke risk after TIA may depend in large part on the underlying TIA mechanism. Accurate early etiologic classification, however, might require imaging more sensitive to acute ischemia than CT, such as DWI-MRI. Future TIA prognostication scores should incorporate both clinical and imaging information and take into account the predictive importance of TIA etiology.

Disclosures

None.

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