Recurrent Events in Transient Ischemic Attack and Minor Stroke

To the Editor:

We have read with great interest the article entitled “Recurrent events in transient ischemic attack and minor stroke. What events are happening and to which patients?,”1 which appeared in Stroke. On the one hand, this study suggests that a high rate of recurrent events after a first minor stroke (MS) or transient ischemic attack (TIA) may be due to symptomatic infarct growth, and not to stroke recurrence; on the other hand, it indicates that MRI may be useful to stratify patients who are at higher risk for recurrence. Although Coutts et al have reached some interesting conclusions, we have few comments on their study. Firstly, it should be considered that all patients were attended and evaluated by a stroke neurologist at the emergency department and that treatment for secondary prevention of stroke was then initiated. All patients were treated acutely with aspirin, and most commenced on statins before discharge. This means that all patients had optimal regimen for secondary prevention of ischemic stroke. Some recent studies have shown both the advantage of early treatment to reduce recurrence after a first MS or TIA,2,3 and the efficacy of statin therapy for secondary prevention of ischemic stroke.4 Therefore, it could be assumed that the study by Coutts et al was going to present a low recurrence rate, with similar values to those obtained in both FASTER and EXPRESS. That is, it is indeed probable that symptomatic infarct growth plays an important role in early impairment of patients with MS or TIA, but only after the best possible treatment to avoid recurrence has been applied. For this reason, we disagree with the idea that “therapies targeting the mechanism of stroke, such as antithrombotic agents, may be less effective than drugs targeting reduction of final infarct volume in reducing the risk of stroke recurrence in the first few days after TIA or minor stroke.” It could be somehow affirmed that once the best available measures targeting the mechanism of stroke are established, the next step to reduce early recurrence after MS or TIA would be acting on final infarct volume. In other words, these two measures should be considered as complementary to each other because they have an influence on different mechanisms contributing to early functional impairment after a MS or TIA. Obviously, there is need for prospective, randomized and well-designed studies to determine the use of drugs acting on final infarct volume. Another noticeable contribution by Coutts et al is the necessity to restore the classic definition of TIA. It is evident that this entity is defined arbitrarily, and also that it has gradually lost clinical and prognostic efficacy with the development of neuroimaging techniques. In this sense, we coincide with the authors in affirming that it is probable that the MRI plays a key role both in the acute stage of the disease—as it establishes the ischemic penumbral area that could be potentially recovered through reperfusion techniques—and in the implementation of adequate strategies for secondary prevention—as it helps to determine the group of patients at higher risk for recurrence. However, as Coutts et al indicate, “it remains unclear whether MR is an appropriate surrogate outcome because the correlation with 90-day clinical outcomes is less than convincing.” Thus, it is necessary to perform further, well-designed studies to define which parameters related to RM techniques could predict a higher risk for recurrence and to what extent they are fully reliable. In conclusion, we think that this is an interesting study which adds some relevant data on what occurs after a MS or TIA once prevention measures have been established.

Disclosures

None.

Ayoze González-Hernández, MD
Oscar Fabre-Pi, MD
Juan Carlos López-Fernández, MD
Department of Neurology
Hospital Universitario de Gran Canaria Dr. Negrín
Las Palmas, Spain

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Ayoze González-Hernández, Óscar Fabre-Pi and Juan Carlos López-Fernández

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