Association Between Diabetes and Stroke Subtype on Survival and Functional Outcome 3 Months After Stroke: Data From the European BIOMED Stroke Project

To the Editor:

Regarding the study by Megherbi et al. performed to evaluate stroke features, prognosis, and functional outcome in patients with diabetes compared with patients without diabetes, we would like to discuss some issues of potential interest.

First, the authors analyzed diabetic patients classified in accordance with WHO diagnostic criteria for diabetes used in 1993 (fasting plasma glucose >140 mg/dL), so it is probably an underestimation of the number of diabetic subjects in the 4537 consecutive patients with ischemic stroke enrolled. Perhaps if they could have classified diabetic patients in accordance with American Diabetes Association (1997) criteria (fasting plasma glucose >126 mg/dL), a higher number of diabetic subjects would have been detected. Indeed the authors do not distinguish among known diabetes, newly diagnosed diabetes, and stress hyperglycemia.

Second, in this study clinical subtypes of ischemic stroke were rated according to the Oxfordshire Community Stroke Project criteria, but it is conceivable that applying the TOAST classification of ischemic stroke—a classification with anatomical, physiopathological, clinical, and instrumental basis that is easily applicable and extensively validated—would offer a more precise selection of patients with lacunar strokes.

Third, Megherbi et al evaluated as outcome indicators vital status, handicap (Rankin score), and disability (Barthel index), but they have not evaluated acute neurological deficit using the Scandinavian Stroke Scale (SSS) so as to evaluate some difference between diabetic and nondiabetic subjects.

Fourth, our group (reference 4 and unpublished data), even in a case-control study of comparison between diabetic and nondiabetic including 102 diabetic patients and 204 nondiabetic controls matched by age and sex with acute ischemic stroke, evaluated the relationship between diabetes and stroke subtype, prior to the article by Megherbi et al. In our study, diabetes results associated with lacunar ischemic stroke subtype, with a history of hypertension, and with a higher SSS score at admission and association of diabetes with lacunar subtype remain significant also after adjustment for hypertension. These data are suggestive of the peculiar anatomopathological findings of the diabetic cerebral infarction as showed by Aronson and Kane and Peress and Kane that reported a higher, autopsically detected incidence of lacunar infarcts in diabetic subjects who died after an ischemic stroke. Indeed the higher SSS score of our diabetic patients converges with the higher frequency of lacunar stroke subtype among diabetics, because lacunar stroke could lead to a lower neurological deficit grade at admission owing to the smaller brain infarct size.

We agree with the authors’ conclusion to recall the role of diabetes in the development of cerebral microangiopathy and lacunar infarction. This role may explain the lack of high mortality in the diabetic group in the acute stage. However, the likelihood of handicap and disability are high.

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Response

We thank Licata at al to the interest they show in the difficult question of diabetes and stroke.

For the first remark, we classified the patients in accordance with the WHO diagnostic criteria for diabetes used in 1993 for a study that took place at this time.

It was not possible to translate the data observed in 1993 with diagnostic criteria defined in 1997. We enrolled patients with repeated fasting plasma glucose level performed in all centers >7.8 mmol/L, so patients with transient stress response hyperglycemia were not enrolled. With our criteria, we enrolled 937 diabetes patients representing 21% of the stroke group, which is the usual data in the literature.

The second remark is interesting. The TOAST classification had not yet been published at the time we were planning our study. The Oxfordshire Community Stroke Project Classification is extensively validated and offers large value for multicenter epidemiological studies such as ours. Finally, it is not possible to translate from a classification used as in a large study as ours, to another one.

In response to the third remark, we did not use the Scandinavian Stroke Scale in the acute stage, because our aim was to study handicap (Rankin Score) and disability (Barthel index) at 3 months, compared with all clinical symptoms and deficits analyzed one after another.

With regard to the fourth remark, we are happy to see that the authors have found similar results with the large place of lacunar infarct, but these results were not easily available or had been only submitted.

We agree with the authors’ conclusion to recall the role of diabetes in the development of cerebral microangiopathy and lacunar infarction. This role may explain the lack of high mortality in the diabetic group in the acute stage. However, the likelihood of handicap and disability are high.

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