The Glycemia in Acute Stroke Study

To the Editor:

We read with interest the recent publication by Fuentes and colleagues concerning the prognostic value of capillary blood glucose levels in determining prognosis after acute stroke. Admission hyperglycemia is a common finding after acute stroke, and studies by our own and other groups have confirmed its association with poor clinical outcomes. The Glycemia in Acute Stroke (GIAS) study reported by Fuentes et al was an observational study in which 476 patients presenting with acute ischemic stroke had capillary blood glucose measurements taken on admission and then 3 times daily for the first 48 hours in hospital. Maximum capillary blood glucose >155 mg/dL (8.6 mmol/L) at any time during the first 2 days was shown to have predictive value for poor outcome at 3 months. However, hyperglycemia or indeed maximum blood glucose during this time period may have been influenced by several factors including administration of intravenous fluids, reinitiation of oral feeding or initiation of enteral feeding, all of which may be associated with stroke severity and thereby outcome. The issue of feeding is of particular relevance because all patients in the GIAS study received oral or tube nutrition (where required) at 24 hours after admission and the timing of capillary blood glucose measurements in relation to mealtimes or feeds is not specified. Furthermore, glucose-lowering therapy was not standardized, being left to the discretion of the supervising clinician, and this could also have influenced the maximum glucose level seen in individual patients. Moreover, some patients likely to have a poor prognosis due to stroke severity or premorbid disability were also excluded from the study. The GIAS study does not provide any information on the previous glycemic status of included patients, such as admission glycohemoglobin (HbA1c) levels. Our group has shown that patients with known and therefore treated type 2 diabetes mellitus may have a better clinical outcome after ischemic stroke than individuals with previously unrecognized diabetes or impaired glucose tolerance in whom the diagnosis is made on admission to hospital.

We would also question the interpretation of the receiver operating characteristic curves that suggest capillary blood glucose >155 mg/dL as the optimal cut-off value for prediction of poor outcome. The reported sensitivities and specificities for admission glucose (34.6%, 80.3%) and maximum glucose (53%, 73%) are likely to be of limited clinical value.

In referencing our study, the Glucose Insulin in Stroke Trial (GIST-UK), we are disappointed to see this misquoted by Fuentes and colleagues because patients with primary intracerebral hemorrhage were eligible for inclusion. We are surprised by their statement that most participants in GIST had a baseline glucose level <150 mg/dL and were ‘thus without hyperglycemia,’ when the Glias cut-off level for poor outcome is only 5 mg/dL greater than this and therefore not a clinically significant difference. Furthermore, to suggest that the THIS trial data from only 46 patients with baseline glucose >150 mg/dL supports the GIAS threshold for poor outcome is probably beyond extrapolation.

We would also dispute the final conclusion from Fuentes and colleagues concerning the use of the GIAS cut-off level for capillary blood glucose as a reference marker for future interventional studies; a cut-off level with such poor sensitivity and specificity has to be of limited value.

Disclosures

None.

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