The Hidden Burden of Glucose Pathology in Acute Stroke Remains Hidden

To the Editor:

Using the ECASS-II cohort, Drs Yong and Kaste postulate that hyperglycaemia persisting over the first 24 hours after onset of stroke (found in 62.1% of their cohort) is a marker of deleterious outcome. The authors are to be congratulated for focusing on the underinvestigated role of glucose pathology and unfavourable outcome of acute stroke. This relationship is not well understood but bears a vast potential for treatment as well as prevention.

We hold that persisting high-glucose blood levels seen in acute stroke patients should firstly be seen as a marker of undiagnosed diabetes and reflects a worse outcome because of the additional risk known from diabetics.

We have investigated a cohort of 238 acute stroke patients and found a high rate of previously undiagnosed diabetes in such patients: 16.4% had newly diagnosed diabetes adding up to a total of 36% diabetic patients. An additional 24% had impaired glucose tolerance or impaired fasting glucose. We therefore argue that also in the ECASS-II cohort such a large and undetected rate of diabetes might have been prevalent, and that the high rates of blood glucose persisting in such patients rather reflects the high rate of diabetics prone to stroke and thus prone to worse outcome.

The criteria for defining diabetes in acute stroke trials usually are based on history taking. In our view the presence of “history of known diabetes” and/or “intake of antidiabetic drugs or insulin” are not sufficient to define the large and hidden burden of glucose pathology in a cohort of stroke patients and should include serial measurements as well as oral glucose tolerance tests where applicable.

Therefore, we hold that the results of this study reflect rather the influence of the high rate of previously undiagnosed diabetics than the dynamics of stress-related hyperglycemia in acute stroke. This does not contradict the clinically most pertinent reasoning by Yong and Kaste—that the negative influence of high glucose metabolism rates in the peri-infarct area especially in thrombolysed patients invariably causes worse outcome; rather, it only shows that it is not a transitory phenomenon occurring only in acute stroke.

Although using data sets from clinical trial studies to investigate other research questions that are unrelated to the trial aims is an interesting and cost-saving approach, it must be conceded that the weakness of such data sets stems from the fact that these were never collected to specifically address such questions and might therefore contain an immeasurable bias, especially in view of such studies as ours that show that glucose pathology in acute stroke has a high prevalence comparable only to traditionally known risk factors with high prevalence such as arterial hypertension.

Disclosures

None.

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