LETTER TO THE EDITOR

Hyperglycaemia and the outcome of stroke

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We read with great interest the article by Uyttenboogaart and co-workers on the association of serum glucose level with outcome in acute ischaemic stroke. In their multivariate analysis hyperglycaemia (glucose >8 mmol/l) was associated with worse functional outcome in a concentration-dependent manner in non-lacunar stroke, whereas in lacunar stroke hyperglycaemia was associated with favourable outcome, this beneficial effect diminishing with more severe hyperglycaemia (>12 mmol/l). Although Uyttenboogart and colleagues creditably discuss the potential neuroprotective effect of lactate produced by astrocytes, they fail in their conclusions stating that ‘hyperglycaemia has a detrimental effect in non-lacunar stroke, but moderate hyperglycaemia may be beneficial in lacunar stroke’. We would like to point out that the association of hyperglycaemia with worse clinical outcome in non-lacunar stroke does not automatically prove causality between these matters.

We know from previous studies that the increase in serum glucose level in acute stroke is a dynamic (Christensen and Boysen, 2002), stress-related reaction displaying a significant positive correlation between serum cortisol and glucose levels (Murros et al., 1993; O’Neill et al., 1991). Furthermore, it was revealed already over 30 years ago that in non-lacunar stroke hyperglycaemia is associated with worse outcome (Melamed, 1976). This association was later linked also with the size of the stroke (Baird et al., 2003; Murros et al., 1992). The problem in evaluating the significance of serum glucose level in the acute phase is that the preceding glucose level is rarely known. Studies evaluating the usefulness of glycosylated haemoglobin are scarce, but they suggest a lack of correlation with outcome and the size of the brain infarction (Baird et al., 2003; Murros et al., 1992).

We emphasize that it may be dangerous to conclude that hyperglycaemia has a detrimental effect in acute stroke. Based on the existing data modest hyperglycaemia may even have a neuroprotective effect. This view is partly supported by a recent interventional GIST-UK study (Gray et al., 2007) reporting the outcome of 933 patients with acute stroke that were randomized to receive either saline infusion or glucose/potassium/insulin infusion to achieve target blood glucose levels between 4 and 7 mmol/l. The baseline clinical and stroke characteristics were similar in both groups. There was no statistically significant difference in the outcome between the groups, as measured by mortality and severe disability at 90 days, but the patients who died actually had the greatest reduction in blood glucose levels.

Whether hyperglycaemia should be treated actively in the acute phase of non-lacunar stroke thus remains to be elucidated (Diringer, 2005). Current American guidelines (Adams et al., 2003) recommend lowering of the blood glucose to reach a level below 16.6 mmol/l in acute stroke, while European guidelines advise to treat hyperglycaemia already exceeding 10 mmol/l (European Stroke Initiative Recommendations for Stroke Management—Update 2003). For today, we think that clinicians should have a critical attitude towards the present guidelines and remember the principle primum non nocere.

References