Plasma Cortisol as a Measure of Stress Response in Acute Stroke

To the Editor:

Several studies have suggested that elevated blood glucose levels in acute stroke are related to clinical outcome. This could be a cause-and-effect relationship since there is evidence of aggravated tissue lactacidosis and a poor outcome in hyperglycemic animals subjected to acute cerebral ischemia.1 The hyperglycemia could also be a reflection of a stress response only coincidentally related to prognosis.2 Support for the concept of stress changes being linked with outcome results from studies of blood cortisol levels.

In the double-blind randomized trial of dexamethasone carried out in Nottingham, UK,3 blood cortisol levels were recorded on admission. The relationship between cortisol levels and survival is shown in Figure 1. There was also a relationship between plasma cortisol and the level of consciousness. Thus the 12 deeply unconscious patients had a mean plasma cortisol level of 1,750 nmol/l, in contrast to a mean level of 800 nmol/l in 36 alert subjects.

These data show that patients with severe strokes who are in coma on admission and destined to die within 1 month show a high plasma cortisol, providing evidence of an increased stress response in such patients. Two previous studies also showed evidence of a greater stress response in stroke patients with poor outcome. Oka4 showed a higher plasma cortisol in stroke patients who died and a return to normal levels by the second week in those who recovered. Feibel et al5 found a higher mortality in stroke patients with high 24-hour urinary catecholamine levels, and their cortisols were also elevated when compared with those with low catecholamine excretion rates. Woo et al2 also noted that fructosamine and glycosylated hemoglobin levels, which reflect prestroke glucose levels, did not correlate with outcome.

Only interventional studies will reveal whether a cyclical process could occur with stress hyperglycemia due to severe ischemic insult that aggravates the tissue metabolic damage, thereby increasing tissue destruction.1,2 In the absence of such information, epidemiological evidence that hyperglycemia is linked to outcome should not yet be interpreted as proof of a cause-and-effect relationship.

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