

Hyperglycemia during acute cerebral infarction

Bruno et al. studied patients with acute cerebral infarction. Admission hyperglycemia was associated with worse clinical outcomes, including a greater chance for intracerebral hemorrhage. This association was independent of rt-PA use or other factors that contribute to stroke outcomes, such as age, stroke severity, and history of diabetes mellitus.

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Commentary

by Roger Simon, MD

In 1977 Meyers and Yamaguchi reported that glucose administration prior to experimental global cerebral ischemia in primates worsened neurologic outcome. A meta-analysis of retrospective studies has supported a correlation to this observation in clinical stroke morbidity and mortality.¹ Large prospective trials now provide additional insight into this association. From the 1,259 patients in the TOAST trial of heparinoid treatment, admission hyperglycemia was associated with worse outcome in nonlacunar stroke but was not associated with hemorrhagic transformation.²

The Bruno et al. study analyzes data from the NINDS rt-PA trial, which again shows an association between admission hyper-

glycemia and stroke outcome, with hypertension as a covariable. Importantly, in this study hyperglycemic patients were at higher risk for symptomatic hemorrhage.

Thus, normalization of glucose might be a reasonable component of acute stroke management if the risks of treatment induced hypoglycemia could be avoided.³

Curiously, 25 years after Meyers and Yamaguchi, the mechanism of toxicity of hyperglycemia remains elusive; the initial assumption that lactate and acidosis were the culprits have not withstood careful scrutiny.⁴ Solving the puzzle as to *why* may provide the ultimate therapeutic target(s) for treatment of hyperglycemia in acute stroke patients.

References

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Cognitive performance 5 years after coronary artery bypass surgery (CABG)

“Late cognitive decline is not an inevitable outcome after CABG . . . cognitive decline may be preventable.”

In contrast to a recently published US study reporting a marked long-term cognitive decline after CABG, Müllges et al. could not demonstrate a decline in their 52 patients. The authors propose that stringent vascular risk factor control contributed to the more favorable long-term outcome.

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The accompanying editorial by Selnes and McKhann notes that the optimistic outcome of patients in the Müllges et al. study contrasts with earlier studies. However, there is evidence that CABG patients are at high risk for new strokes, which may in fact be a major reason for decline in cognition during follow-up.

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Multiple sclerosis, depression, suicidal intent, and β -interferon

Feinstein studied 140 patients with MS. Lifetime suicidal intent was present in 28.6% of patients. Independent predictors of suicidal intent were major depression, the severity of the depression, living alone, and alcohol abuse. A third of patients with suicidal intent and a third with major depression had not received psychological help.

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Patten and Metz obtained depression ratings from 365 MS patients in a placebo-controlled clinical trial of interferon β -1a. In 36 months of follow-up, depression occurred frequently, but depression ratings did not differ significantly between placebo and IFN-treated patients.

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“Clinicians have the opportunity to intervene in . . . the lives of their patients, reducing avoidable deaths and . . . suffering.”

The Caine and Schwid editorial accompanying these two papers points out that reasons for the high prevalence of depression in MS patients remain unclear. They emphasize the need to look beyond depression to hopelessness, social support, and alcohol abuse, which may be independent contributors to suicidal risk. Depression and the risk of suicide are not adequately treated in MS. Because neurologists see these patients often, modest changes in practice may reduce suffering and save lives.

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Interferon β -1a in secondary progressive MS

The Cohen et al. Phase III study of IFN β -1a in secondary progressive MS employed the MS Functional Composite as the primary outcome measure. They found benefit on disease progression, relapses, MRI, and quality of life.

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Methylprednisolone (IVMP) effect on MRI brain atrophy in RRMS

Rao et al. studied serial MRI in 26 RRMS patients during baseline period and while receiving IFN β who were treated with IVMP for exacerbations by serial MRI. Following IVMP, there was significant decrease in the brain volume for 1 month. MRI should be delayed for >1 month following IVMP when studying brain atrophy in RRMS patients.

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Blood pressure and brain atrophy in healthy elderly

Studying normal subjects with hypertension Goldstein et al. found that slight elevation of ambulatory 24-hour blood pressure correlated with brain atrophy in subjects aged 56 to 80 years. Blood pressure level and its variability were related to brain atrophy; the combination of both was even more strongly correlated with atrophy.

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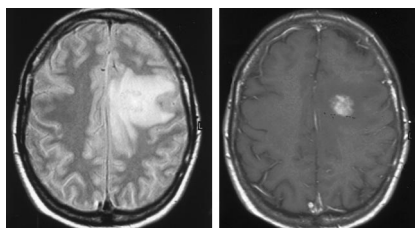
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SOD1 mouse model of ALS: Survival unrelated to glutathione peroxidase

Cudkowicz et al. report that survival in transgenic mice expressing mutant human SOD1 does not vary with CNS glutathione peroxidase activity. This argues against the importance of enhanced peroxidative activity in motor neuron death in this ALS model.

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Is a “vanishing tumor” always a lymphoma?



Bromberg et al. followed 12 patients with a spontaneous or steroid-induced regression of a contrast-enhancing cerebral mass lesion. In only 6/12 was primary cerebral non-Hodgkin lymphoma present.

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Steroid-responsive neuropathy and myelin protein zero gene mutation

Watanabe et al. studied a case of hereditary neuropathy that presented with asymmetric distal weakness. The patient responded twice to corticosteroids and symptoms recurred after corticosteroid tapering.

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Transient global amnesia after sildenafil (Viagra) use

Savitz and Caplan report a 51-year-old man with a history of migraine who developed transient global amnesia after taking sildenafil. Use of sildenafil by migraineurs may trigger posterior circulation vasomotor instability and impair hippocampal function.

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Somatic mosaicism in Sturge-Weber syndrome (SWS)

SWS is not considered a hereditary disease. Here, Huq et al. report evidence for mosaicism in affected tissues of two (of four) patients with SWS.

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Neurology[®]

September 10 Highlights
Neurology 2002;59;657-659
DOI 10.1212/WNL.59.5.657

This information is current as of September 10, 2002

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