Serum Albumin Level as a Predictor of Ischemic Stroke Outcome

To the Editor:

We read with great interest the recent article by Dr Dziedzic et al.1 on the relationship between serum albumin level and ischemic stroke outcome. Even though low serum albumin has been associated with an increased incidence of stroke in epidemiologic studies,2 a high serum albumin level in acute stroke patients was described, for the first time, to decrease the risk of poor outcome among hospitalized patients. The neuroprotective effects of endogenous albumin on the capillary microcirculation in the early reperfusion phase were proposed to explain the cellular mechanism of this association, and the role of exogenous albumin therapy in stroke recovery was briefly reviewed.

It is important to recognize the role of serum albumin as a marker of clinical outcomes in vascular disease. Apart from stroke, serum albumin has been associated with adverse vascular events in patients with cardiac3 and renal4 diseases. Among hospitalized patients, hypoalbuminemia was found to be associated with frequent hospitalizations, higher mortality, and readmission,5 and an independent prognostic factor for all deaths among healthy middle-aged individuals in population studies.6 Serum albumin is regulated by factors influencing protein synthesis, breakdown, leakage to the extravascular space, and food intake. In clinical practice, serum albumin is often considered a marker of nutritional status and a negative phase protein that decreases in concentration during injury and sepsis.7

There has been conflicting evidence in the literature on albumin therapy in treating patients with hypoalbuminemia from an underlying vascular disease. Albumin has a molecular weight of about 66 kDa, thus preventing it from passing through the blood–brain barrier by diffusion or by carrier systems through these membranes. Local redistribution, crystalloid dilution, and changes in the metabolism of albumin, which result in ineffective delivery and concentration within the central nervous system, frustrate therapeutic interventions. In septic patients, albumin therapy was not associated with a rise in serum albumin. Instead, a fall in serum albumin was observed and this was hypothesized to be secondary to capillary leakage.8

Until these important questions on albumin therapy are answered in randomized controlled studies, therapeutic options in patients with hypoalbuminemia should be directed toward treating the underlying cause, avoiding or treating salt and water overload, instituting prompt medical and surgical treatment of inflammation and sepsis, and providing appropriate nutritional support to enhance recovery in patients with ischemic strokes.

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Stroke. published online October 7, 2004;
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/early/2004/10/07/01.STR.0000145487.89910.12.citation

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