Early neurological deterioration in acute ischemic stroke

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We have greatly enjoyed reading the recently published article by Huang and colleagues.[1] The authors aimed to investigate whether endovascular therapy (EVT) was one of the factors influencing the incidence of early neurological deterioration (END) in patients with acute ischemic stroke as compared with intravenous thrombolysis alone. They found that hyperglycemia, hyperuricemia, and EVT may be independently associated with END in acute ischemic stroke.

Finding predictors of END is crucial because early treatment can help to prevent this serious complication and several studies have focused on the search for predictors of END which is usually defined as the clinical worsening or recurrence during the first 72 hours after ischemic stroke. Several biomarkers have been investigated for this purpose previously. Ryu et al.[2] found that low high-density lipoprotein-cholesterol levels and high apoB/apoA-I ratios were independently associated with END in patients with ischemic stroke. Bhatia et al.[3] previously showed that the use of blood urea nitrogen/creatinine ratio >15 as a marker of relative dehydration can be helpful in detecting patients with dehydration early and thus plays a role in preventing END. Kwon et al.[4] showed that patients with acute stroke with elevated serum homocysteine levels are at an increased risk for END. Brain natriuretic peptide (BNP) is frequently elevated after an acute stroke and has been shown to be an independent predictor of mortality[5] and a biological marker to distinguish cardioembolic from other stroke subtypes.[6] We have previously showed that plasma BNP level of >235 pg/mL can predict 1-year mortality and >155 pg/mL can differentiate cardioembolic from non–cardioembolic stroke in patients with first-ever acute ischemic stroke.[6] Therefore, it would be useful to have data about troponin and BNP levels on admission and their relationship with the END and EVT in patients with acute ischemic stroke.

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