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Serum tenascin-C predicts severity and outcome of acute intracerebral hemorrhage☆

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ABSTRACT

Background: Tenascin-C is a matricellular protein related to brain injury. We studied serum tenascin-C in acute intracerebral hemorrhage (ICH) and examined the associations with severity and outcome following the acute event.

Methods: Tenascin-C samples were obtained from 162 patients with acute hemorrhagic stroke and 162 healthy controls. Poor 90-day functional outcome was defined as modified Rankin Scale score > 2. Early neurological deterioration (END) and hematoma growth (HG) were recorded at 24 h.

Results: Patients had higher tenascin-C levels than controls. Tenascin-C levels were positively correlated with hematoma volume or National Institutes of Health Stroke Scale score at baseline. Elevated tenascin-C levels were independently associated with END, HG, 90-day mortality and poor functional outcome. Moreover, tenascin-C levels significantly predicted END, HG and 90-day outcomes under receiver operating characteristic curves.

Conclusions: An increase in serum tenascin-C level is associated with an adverse outcome in ICH patients, supporting the potential role of serum tenascin-C as a prognostic biomarker for hemorrhagic stroke.

1. Introduction

Spontaneous intracerebral hemorrhage (ICH) is a catastrophic event. It accounts for about 10% of strokes and is associated with high mortality and morbidity. Despite its devastating effects and social burden, no proven treatment has been consistently demonstrated to be effective in ameliorating ICH consequences [1–5]. Clearly, both baseline hematoma volume and National Institutes of Health Stroke Scale (NIHSS) score are predictors of outcome in ICH patients [6–8]. Alternatively, Hematoma growth (HG) and early neurological deterioration (END) have been found to be the independent determinants of death and disability after ICH [9,10]. Therefore, predicting HG and END is beneficial to assess outcome following ICH.

Tenascin-C (TNC), a large extracellular matrix glycoprotein, belongs to the damage-associated molecular patterns family and plays important roles in cell proliferation, migration, differentiation and apoptosis [11–14]. Increased circulating TNC levels have been previously demonstrated to be associated with severity and outcome of some diseases related to tissue injury and inflammation, e.g. acute myocardial infarction, pneumonitis, inflammatory bowel disease and rheumatoid arthritis [15–19]. Intriguingly, mechanical injury induces boundary astrocytes to produce and release TNC that promotes cell proliferation and migration [20]. Moreover, neurons can express tenascin-C mRNA [21]. Still, TNC induces neuronal apoptosis and is implicated in early brain injury after subarachnoid hemorrhage in rats [22–24]. Similarly, deficiency of endogenous TNC prevented neurological impairments, brain edema formation, and blood-brain barrier disruption following subarachnoid hemorrhage in mice [25,26]. Of Note, elevated cerebrospinal fluid TNC levels were recently found to be correlated with worse admission World Federation of Neurosurgical Societies grade and cerebral vasospasm after aneurysmal subarachnoid hemorrhage in humans [27]; and enhanced serum TNC levels were associated with severity and independently predicted outcome in patients with traumatic brain injury [28]. Thus, we hypothesized that the TNC levels might be useful for evaluating severity and predicting outcome after ICH. To date, no researchers have reported circulating TNC levels in ICH patients. We aimed to determine the relationship between TNC levels and severity in patients with acute ICH and their impact on clinical outcome.

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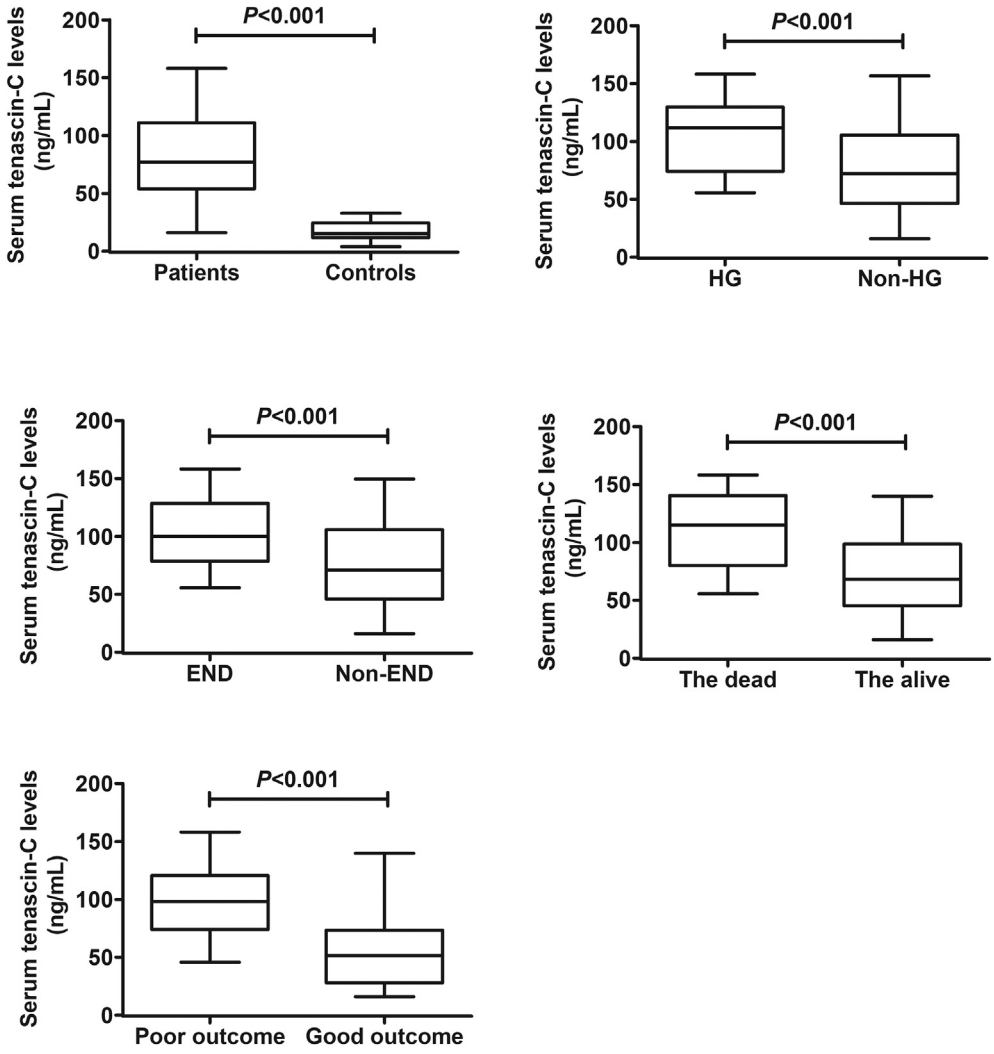

Fig. 1. Comparison of serum tenascin-C levels between different subgroups after acute intracerebral hemorrhage in terms of hematoma growth (HG), early neurologic deterioration (END), 90-day death and 90-day poor outcome. Serum tenascin-C levels are presented as the medians and interquartile ranges.

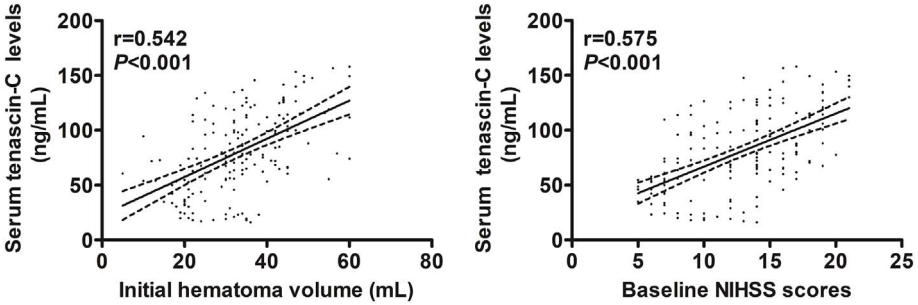

Fig. 2. Correlation of serum tenascin-C levels with hematoma volume and National Institutes of Health Stroke Scale (NIHSS) scores after acute intracerebral hemorrhage.

2. Methods

2.1. Study population

We prospectively evaluated consecutive patients with acute primary basal ganglia hemorrhage admitted to our emergency room within 6 h from symptoms onset between July 2014 and December 2016. We excluded those patients who were under.

antiplatelet or anticoagulant treatment, those who underwent a surgical procedure, those who had such comorbidities as autoimmune diseases, uremia, liver cirrhosis, malignancy and chronic heart or lung disease and those previously suffered from neurological diseases e.g. ischemic or hemorrhagic stroke and severe head trauma. Control group were composed of healthy individuals who underwent regular physical examinations at our hospital. All aspects of the study were approved by the institutional review board at our Hospital. Written informed consent was obtained from controls and family members of patients.

2.2. Clinical and radiological assessment

Upon entry into emergency department, we collected the following data: gender, age, body mass index, cigarette smoking, alcohol