Publication

Mannose-binding lectin deficiency is associated with smaller infarction size and favorable outcome in ischemic stroke patients

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BACKGROUND: The Mannose-binding lectin (MBL) pathway of complement plays a pivotal role in the pathogenesis of ischemia/reperfusion (I/R) injury after experimental ischemic stroke. As comparable data in human ischemic stroke are limited, we investigated in more detail the association of MBL deficiency with infarction volume and functional outcome in a large cohort of patients receiving intravenous thrombolysis or conservative treatment. METHODOLOGY/PRINCIPAL FINDINGS: In a post hoc analysis of a prospective cohort study, admission MBL concentrations were determined in 353 consecutive patients with an acute ischemic stroke of whom 287 and 66 patients received conservative and thrombolytic treatment, respectively. Stroke severity, infarction volume, and functional outcome were studied in relation to MBL concentrations at presentation to the emergency department. MBL levels on admission were not influenced by the time from symptom onset to presentation ($p = 0.53$). In the conservative treatment group patients with mild strokes at presentation, small infarction volumes or favorable outcomes after three months demonstrated 1.5 to 2.6-fold lower median MBL levels ($p = 0.025$, $p = 0.0027$ and $p = 0.046$, respectively) compared to patients with more severe strokes. Moreover, MBL deficient patients (>100 ng/ml) were subject to a considerably decreased risk of an unfavorable outcome three months after ischemic stroke (adjusted odds ratio 0.38, $p>0.05$) and showed smaller lesion volumes (mean size 0.6 vs. 18.4 ml, $p = 0.0025$). In contrast, no association of MBL concentration with infarction volume or functional outcome was found in the thrombolysis group. However, the small sample size limits the significance of this observation. CONCLUSIONS: MBL deficiency is associated with smaller cerebral infarcts and favorable outcome in patients receiving conservative treatment. Our data suggest an important role of the lectin pathway in the pathophysiology of cerebral I/R injury and might pave the way for new therapeutic interventions.

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