

Abstract

High-mobility group box-1 (HMGB1) is a nuclear protein that promotes inflammation during the acute phase post-stroke, and enhances angiogenesis during the delayed phase.

Here, we evaluated whether indirect revascularization surgery with HMGB1 accelerates brain angiogenesis in a chronic cerebral hypoperfusion model. Seven days after hypoperfusion induction, encephalo-myo-synangiosis (EMS) was performed with or without HMGB1 treatment into the temporal muscle. We detected significant increments in cortical vasculature ($p < 0.01$), vascular endothelia growth factor (VEGF) expression in the temporal muscle ($p < 0.05$), and ratio of radiation intensity on the operated side compared with the non-operated side after EMS in the HMGB1-treated group than in the control group ($p < 0.01$). Altogether, HMGB1 with EMS in a chronic hypoperfusion model promoted brain angiogenesis in a VEGF-dependent manner, resulting in cerebral blood flow improvement. This treatment may be an effective therapy for patients with moyamoya disease.