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Microglial activation induced by traumatic brain injury is suppressed by postinjury treatment with hyperbaric oxygen therapy.

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Source

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Abstract

BACKGROUND:

The mechanisms underlying the protective effects of hyperbaric oxygen (HBO) therapy on traumatic brain injury (TBI) are unclear. TBI initiates a neuroinflammatory cascade characterized by activation of microglia and increased production of proinflammatory cytokines. In this study, we attempted to ascertain whether the occurrence of neuroinflammation exhibited during TBI can be reduced by HBO.

METHODS:

TBI was produced by the fluid percussion technique in rats. HBO (100% O₂ at 2.0 absolute atmospheres) was then used at 1 h (HBO I) or 8 h (HBO II) after TBI. Neurobehavior was evaluated by the inclined plane test on the 72 h after TBI and then the rats were killed. The infarction area was evaluated by Triphenyltetrazolium chloride. Immunofluorescence staining was used to evaluate neuronal apoptosis (TUNEL + NeuN), microglial cell aggregation count (OX42 + DAPI), and tumor necrosis factor-alpha (TNF- α) expression in microglia cell (OX42 + TNF- α).

RESULTS:

The maximum grasp angle in the inclined plane test and cerebral infarction of the rats after TBI were significantly attenuated by HBO therapy regardless of whether the rats were treated with HBO 1 or 8 h after TBI compared with the controls. TBI-induced microglial activation, TNF- α expression, and neuronal apoptosis were also significantly reduced by HBO therapy.

CONCLUSIONS:

Our results demonstrate that treatment of TBI during the acute phase of injury can attenuate microgliosis and proinflammatory cytokine TNF- α expression resulting in a neuroprotective effect. Even treating TBI with HBO after 8 h had a therapeutic effect.

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