J Trauma Acute Care Surg. 2012 Mar;72(3):650-9. doi: 10.1097/TA.0b013e31823c575f.

Attenuating inflammation but stimulating both angiogenesis and neurogenesis using hyperbaric oxygen in rats with traumatic brain injury.

Lin KC, Niu KC, Tsai KJ, Kuo JR, Wang LC, Chio CC, Chang CP. J Trauma Acute Care Surg. 2012 Jul;73(1):295-6.

Abstract

BACKGROUND:

Inflammation, angiogenesis, neurogenesis, and gliosis are involved in traumatic brain injury (TBI). Several studies provide evidence supporting the neuroprotective effect of hyperbaric oxygen (HBO2) therapy in TBI. The aim of this study was to ascertain whether inflammation, angiogenesis, neurogenesis, and gliosis during TBI are affected by HBO2 therapy.

METHODS:

Rats were randomly divided into three groups: TBI + NBA (normobaric air: 21% O2 at 1 absolute atmospheres), TBI + HBO2, and Sham operation + NBA. TBI + HBO2 rats received 100% O2 at 2.0 absolute atmospheres for 1 hr/d for three consecutive days. Behavioral tests and biochemical and histologic evaluations were done 4 days after TBI onset.

RESULTS:

TBI + NBA rats displayed: (1) motor and cognitive dysfunction; (2) cerebral infarction and apoptosis; (3) activated inflammation (evidenced by increased brain myeloperoxidase activity and higher serum levels of tumor necrosis factor-α); (4) neuronal loss (evidenced by fewer NeuN-positive cells); and (5) gliosis (evidenced by more glial fibrillary protein-positive cells). In TBI + HBO2 rats, HBO2 therapy significantly reduced TBI-induced motor and cognitive dysfunction, cerebral infarction and apoptosis, activated inflammation, neuronal loss, and gliosis. In addition, HBO2 therapy stimulated angiogenesis (evidenced by more bromodeoxyuridine-positive endothelial and vascular endothelial growth factor-positive cells), neurogenesis (evidenced by more bromodeoxyuridine-NeuN double-positive and glial cells-derived neurotrophic factor-positive cells), and overproduction of interleukin-10 (an anti-inflammatory cytokine).

CONCLUSIONS:

Collectively, these results suggest that HBO2 therapy may improve outcomes of TBI in rats by inhibiting activated inflammation and gliosis while stimulating both angiogenesis and neurogenesis in the early stage.