Inhibition of apoptosis by Hyperbaric Oxygen in a rat focal cerebral ischemic model.

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The hypothesis was tested that hyperbaric oxygen therapy (HBO) reduced brain infarction by preventing apoptotic death in ischemic cortex in a rat model of focal cerebral ischemia. Male Sprague-Dawley rats were subjected to middle cerebral artery occlusion/reperfusion (MCAO/R) and subsequently were exposed to HBO (2.5 atmospheres absolute) for 2 h, at 6 h after reperfusion. Rats were killed and brain samples were collected at 24, 48, 72 h, and 7 days after reperfusion. Neurologic deficits, infarction area, and apoptotic changes were evaluated by clinical scores, 2,3,7-triphenyltetrazolium chloride staining, caspase-3 expression, DNA fragmentation assay, and terminal deoxynucleotidyl transferase-mediated 2'-deoxyuridine 5'-triphosphate-biotin nick end labeling (TUNEL)-hematoxylin and eosin (H&E) costaining. In MCAO/R without HBO treatment animals, DNA fragmentation was observed in injured cortex at 24, 48, and 72 h but not in samples at 7 days after reperfusion. Double labeling of brain slides with NeuN and caspase-3 demonstrated neurons in the injured cortex labeled with caspase-3. TUNEL+H&E costaining revealed morphologic apoptotic changes at 24, 48, and 72 h after reperfusion. Hyperbaric oxygen therapy abolished DNA fragmentation and reduced the number of TUNEL-positive cells.

Hyperbaric oxygen therapy reduced infarct area and improved neurologic scores at 7 days after reperfusion. One of the molecular mechanisms of HBO-induced brain protection is to prevent apoptosis, and this effect of HBO might preserve more brain tissues and promote neurologic functional recovery.

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