Induced Transient Cerebral Ischemia

Lasers Surg Med. *2002;31(4):283-8.*

Treatment of experimentally induced transient cerebral ischemia with low energy laser inhibits nitric oxide synthase activity and up-regulates the expression of transforming growth factor-beta 1.

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Background and Objectives:

Nitric oxide (NO) has been shown to be neurotoxic while transforming growth factor-beta 1 (TGF-beta1) is neuroprotective in the stroke model. The present study investigates the effects of low energy laser on nitric oxide synthase (NOS) and TGF-beta1 activities after cerebral ischemia and reperfusion injury.

Study Design/Materials and Methods:

Cerebral ischemia was induced for 1 hour in male adult Sprague-Dawley (S.D.) rats with unilateral occlusion of middle cerebral artery (MCAO). Low energy laser irradiation was then applied to the cerebrum at different durations (1, 5, or 10 minutes). The activity of NOS and the expression of TGF-beta1 were evaluated in groups with different durations of laser irradiation.

Results:

After ischemia, the activity of NOS was gradually increased from day 3, became significantly higher from day 4 to 6 ($P < 0.001$), but returned to the normal level after day 7. The activity and expression of the three isoforms of NOS were significantly suppressed ($P < 0.001$) to different extents after laser irradiation. In addition, laser irradiation was shown to trigger the expression of TGF-beta1 ($P < 0.001$).

Conclusions:

Low energy laser could suppress the activity of NOS and up-regulate the expression of TGF-beta1 after stroke in rats.

PMID: 12355575 [PubMed - indexed for MEDLINE]