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[Effects of hydrogen-rich water on the expression of aquaporin 1 in the cerebral cortex of rat with traumatic brain injury]

[Article in Chinese]

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Abstract

Objective: To investigate the effect of hydrogen-rich water on cerebral edema and aquaporin 1 (AQP1) expression in rats with traumatic brain injury (TBI).

Methods: Ninety male Sprague-Dawley (SD) rats were randomly divided into sham operation group, TBI model group, hydrogen-rich water treatment group (H group),with 30 rats in each group. TBI model was reproduced by weight dropping method. The skulls of rats in sham operation group underwent only craniotomy without direct hit and with bone wax sealed suture.5 mL/kg of hydrogen-rich water injection was given intraperitoneally after model reproduction in H group, and equal amount of normal saline was given in sham and TBI groups, once a day for both groups for 5 days. Six rats from each group were sacrificed at 6,12,24,48 hours and 5 days after evaluating neurological severity scores (NSS).The cerebral cortex was harvested, and the pathological changes in morphology of brain tissue were observed with light microscope. The positive expression of AQP1 in cerebral cortex was observed with immunohistochemistry by light microscopy, the AQP1 mRNA expression in cerebral cortex was determined by real-time fluorescent quantization reverse transcription-polymerase chain reaction (RT-PCR),and the AQP1 protein expression in cerebral cortex was determined by Western Blot.

Results: ① All rats in sham operation group had a NSS of zero at each time point. NSS of TBI group was obviously raised with time prolongation, and peaked at 24 hours followed by a lower tendency, while the score in H group was significantly lower than that of TBI group, and the difference was the most obvious at 24 hours as compared with TBI group (9.83 ± 2.78 vs.13.50± 2.42,$P < 0.05$).② It was shown by light microscope that in the TBI group there were pathological changes in cerebral cortex, including obvious irregular arrangement of nerve cells, cerebral edema, obvious bleeding, especially at 24 hours, then the cerebral edema became vanished gradually; and the positive expression of AQP1 in the pia mater at all the time points in the TBI group was significantly increased, and it was most obvious at 24 hours. Compared with TBI group, the pathological changes at time points of 12 hours to 5 days in H group was significantly lessened, and the positive expression of AQP1 in the cerebral pia mater was reduced obviously.③ Compared with sham operation group, the mRNA and protein expressions of AQP1 in cerebral cortex in TBI group were significantly elevated, peaked at 24 hours [AQP1 mRNA ($2^{-\triangle\triangle}$Ct):7.50±0.26 vs.1,AQP1 protein (gray value):1.986±0.110 vs.0.336±0.034, both $P < 0.05$], then they gradually declined. The mRNA and protein expressions of AQP1 in cerebral cortex were significantly decreased after hydrogen-rich water treatment [24-hour AQP1 mRNA ($2^{-\triangle\triangle}$Ct):5.40±0.21 vs.7.50±0.26, 24-hour AQP1 protein (gray value): 1.246±0.137 vs.1.986±0.110, both $P < 0.05$].

Conclusions: The up-regulation of AQP1 mRNA and protein in rats' cerebral cortex after TBI perhaps participates in edema formation which might be involved in the pathophysiology of cerebral edema in TBI. Early treatment with an intraperitoneally injection of hydrogen-rich water is capable of attenuating the extent of TBI-induced up-regulation of AQP1 mRNA and protein, alleviating cerebral edema, and achieving its protective effects.

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