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Hydrogen-rich saline alleviates early brain injury via reducing oxidative stress and brain edema following experimental subarachnoid hemorrhage in rabbits

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Abstract

Background: Increasing experimental and clinical data indicate that early brain injury (EBI) after subarachnoid hemorrhage (SAH) largely contributes to unfavorable outcomes, and it has been proved that EBI following SAH is closely associated with oxidative stress and brain edema. The present study aimed to examine the effect of hydrogen, a mild and selective cytotoxic oxygen radical scavenger, on oxidative stress injury, brain edema and neurology outcome following experimental SAH in rabbits.

Results: The level of MDA, caspase-12/3 and brain water content increased significantly at 72 hours after experimental SAH. Correspondingly, obvious brain injury was found in the SAH group by terminal deoxynucleotidyl transferase-mediated uridine 5'-triphosphate-biotin nick end-labeling (TUNEL) and Nissl staining. Similar results were found in the SAH+saline group. In contrast, the upregulated level of MDA, caspase-12/3 and brain edema was attenuated and the brain injury was substantially alleviated in the hydrogen treated rabbits, but the improvement of neurology outcome was not obvious.

Conclusion: The results suggest that treatment with hydrogen in experimental SAH rabbits could alleviate brain injury via decreasing the oxidative stress injury and brain edema. Hence, we conclude that hydrogen possesses the potential to be a novel therapeutic agent for EBI after SAH.

Figures

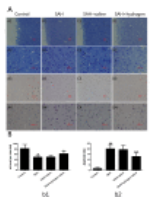

Figure 1 Nissl and TUNEL staining (A)...

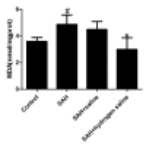

Figure 2 Content of MDA. MDA increased...

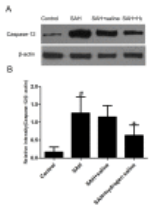

Figure 3 Representative Western blots of caspase-12...

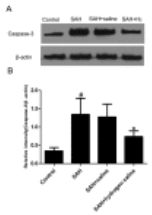

Figure 4 Representative Western blots of caspase-3...

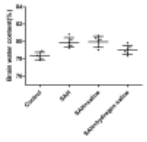

Figure 5 Brain water content measurement. As...

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