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Nuclear factor-κB/Bcl-XL pathway is involved in the protective effect of hydrogen-rich saline on the brain following experimental subarachnoid hemorrhage in rabbits

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Abstract

Early brain injury (EBI), a significant contributor to poor outcome after subarachnoid hemorrhage (SAH), is intimately associated with neuronal apoptosis. Recently, the protective role of hydrogen (H2) in the brain has been widely studied, but the underlying mechanism remains elusive. Numerous studies have shown nuclear factor-κB (NF-κB) as a crucial survival pathway in neurons. Here we investigated the role of H2 in EBI following SAH, focusing on the NF-κB pathway. A double blood injection model was used to produce experimental SAH, and H2 -rich saline was injected intraperitoneally. NF-κB activity within the occipital cortex was measured. Immunofluorescence was performed to demonstrate the activation of NF-κB; Bcl-xL and cleaved caspase-3 were determined via Western blot. Gene expression of Bcl-xL was detected by real-time PCR, and TUNEL and Nissl staining were performed to illustrate brain injury in the occipital cortex. SAH induced a significant increase of cleaved caspase-3. Correspondingly, TUNEL staining demonstrated obvious neuronal apoptosis following SAH. In contrast, H2 treatment markedly increased NF-κB activity and the expression of Bcl-xL and decreased the level of cleaved caspase-3. Additionally, H2 treatment significantly reduced post-SAH neuronal apoptosis. The current study shows that H2 treatment alleviates EBI in the rabbits following SAH and that NF-κB/Bcl-xL pathway is involved in the protective role of H2 .

Keywords: NF-κB/Bcl-xL; apoptosis; early brain injury; hydrogen; subarachnoid hemorrhage.

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