Acute intracranial hypertension

The benefits of controlled decompression (CDC) for patients with acute intracranial hypertension especially in terms of alleviating the complications caused by rapid decompression (RDC) have been confirmed by clinical studies. This study is aimed at evaluating the therapeutic potency of CDC with ubiquitin C-terminal hydrolase-L1 (UCH-L1) and glial fibrillary acidic protein (GFAP) by investigating the potential molecular mechanism in the acute intracranial hypertension (AICH) rabbit model.

Methods: Male New Zealand white rabbits were randomly subdivided into the sham-operated (SH) group, CDC group, and RDC group. Blood plasma samples and brain tissue were collected 2 days before operation (baseline) and at 3, 6, 24, and 72 hours after operation to measure the levels of UCH-L1, GFAP, oxidative stress indicators, and inflammatory cytokines by performing ELISA or Western blot. The neurological score of the rabbits and brain water content was graded 24 h after surgery. qPCR, immunofluorescence, and FJ-C staining were conducted.

Results: CDC improved neurological function, lowered brain water content, ameliorated neuronal degeneration, attenuated oxidative damage, and inflammatory responses to a greater extent than RDC. Plasma UCH-L1 level was significantly lower in the CDC group at 3 h postoperatively than in the RDC group. CDC reduced plasma GFAP levels to various degrees at 3 h, 6 h, and 24 h postoperatively compared with RDC. Immunofluorescence confirmed that the expression of UCH-L1 and GFAP in the cortex of the CDC group was lower than that of the RDC group.

Conclusions: Our data collectively demonstrate that CDC could attenuate oxidative damage and inflammatory responses, downregulate UCH-L1 and GFAP levels, and contribute to an improved neuroprotective effect compared with RDC¹.

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Zhang C, Qian X, Zheng J, Ai P, Cao X, Pan X, Chen T, Wang Y. Controlled Decompression Alleviates Brain Injury via Attenuating Oxidative Damage and Neuroinflammation in Acute Intracranial Hypertension. Biomed Res Int. 2022 Feb 9;2022:1936691. doi: 10.1155/2022/1936691. PMID: 35187159; PMCID: PMC8850036.

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