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[Hydrogen-rich saline attenuates hyperalgesia and reduces cytokines in rats with post-herpetic neuralgia via activating autophagy]

[Article in Chinese]

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Abstract

Objective: To investigate the role of autophagy in hydrogen-rich saline attenuating post-herpetic neuralgia (PHN) in rats.

Methods: A total of 100 male SD rats were randomly divided into the five groups (n = 20) : control group, PHN group, PHN group treated with hydrogen-rich saline (PHN-H2 group), PHN group treated with hydrogen-rich saline and 3-MA (PHN-H2-3-MA group), PHN group treated with hydrogen-rich saline and rapamycin (PHN-H2-Rap group). PHN models were established by varicella-zoster virus (VZV) inoculation. After modeling, 15 mg / kg 3-MA or 10 mg / kg rapamycin were intraperitoneally injected in corresponding rats with PHN once two days for 3 times. Hydrogen-rich saline (10 mL / kg) was injected intraperitoneally twice a day for 7 consecutive days in PHN-H2 group, PHN-H2-3-MA group and PHN-H2-Rap group after VZV injection. The paw withdrawal thresholds (PWT) of 50 rats were detected at 3, 7, 14 and 21 days after modeling. Spinal cord enlargements of the other 50 rats were collected to examine tumor necrosis factor α (TNF- α), interleukin 1 β (IL-1 β) and IL-6 by ELISA and autophagy protein microtubule-associated protein 1 light chain 3 (LC3), beclin 1 and P62 by Western blotting.

Results: Compared with the control group, the rats in the PHN group presented with decreased PWT, increased levels of TNF- α , IL-1 β , IL-6, LC3II and beclin 1, and down-regulated P62 expression. Compared with PHN group, the rats in the PHN-H2 group and PHN-H2-Rap group showed increased PWT, decreased levels of TNF- α , IL-1 β and IL-6, further up-regulated expressions of LC3 and beclin 1 as well as P62 expression. Compared with PHN-H2 group, the rats in the PHN-H2-3-MA group had reduced PWT, elevated expressions of TNF- α , IL-1 β and IL-6, suppressed expressions of LC3 and beclin 1, and enhanced p62 expression.

Conclusion: Hydrogen-rich saline attenuated PWT and inhibited the release of cytokines TNF- α , IL-1 β , IL-6 in rats with PHN via activating autophagy.

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