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Mechanism of demyelination and protective effect of EA on neural myelin sheaths after compressed spinal cord injury

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Compressed spinal cord injury (CSCI) has become a global issue because of its high incidence and complexity etiology. Demyelination is one of the most important pathological factors of CSCI and oligodendrocyte apoptosis is involved in triggering of demyelination. However, fewer reports on pathogenic mechanism and effective treatment of demyelination have been presented from CSCI. To determine whether or not oligodendrocyte apoptosis and demyelination occur after CSCI and to investigate the protective effect of EA- stimulation of Zusanli and Taixi acupoints on neural myelin sheaths (EA, electroacupuncture), a custom-designed model of CSCI was used, the behavioral, morphological and molecular biological methods were used in this study. After CSCI, the rats were spastic, paralyzed and incontinent. The Basso, Beattie, and Bresnahan (BBB) locomotor rating scale scores were decreased as time passed. The compressed spinal cord slices were ischemic. Myelin sheaths became swollen and degenerative; these sheaths were broken down after CSCI with the duration of time. MBP (myelin basic protein) expression was down-regulated after CSCI and consistent with the degree of demyelination. Oligodendrocyte apoptosis occurred at 1 day after CSCI and increased as caspase-12 [a representative of endoplasmic reticulum (ER) stress] expression was enhanced and cytochrome-c (Cyt c, an apoptotic factor and hallmark of mitochondria) was released. Id2 (an oligodendrocyte lineage gene) expression was increased with time after CSCI and distributed widely in the white matter. EGFR signaling (a regulator for survival of oligodendrocytes) were elevated slightly. These results demonstrated that demyelination occurred after CSCI and might be partly caused by oligodendrocyte apoptosis, which was positively correlated with ER–mitochondria interactions and enhanced Id2 expression after CSCI in rats. After EA- stimulation in CSCI rats for 14 days, both locomotor skills and ultra-structural features of myelin sheath were significantly improved. Double immunolabeling showed that EA also enhanced the proliferation of OPCs and OLs (oligodendrocyte), as well as the differentiation of OPCs by promoting Olig2 (the basic helix-loop-helix protein) and attenuating Id2. EA could improve MBP and protect existing OLs from apoptosis by inhibiting caspase-12 and Cyt-c. EGFR signaling also significantly increased. Therefore, the results indicated that the protective effect of EA on neural myelin sheaths is mediated via promotion of OLs proliferation and inhibition of OLs death after CSCI. Thus, we concluded that demyelination is initiated by OLs apoptosis through endoplasmic reticulum – mitochondria interactions after CSCI and attenuated by EA via promotion of OPCs proliferation/differentiation and protection OLs from death.

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