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Protective effects of glucosamine-kynurenic acid after compression-induced spinal cord injury in the rat

Research Article

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Abstract: Kynurenic acid (KYNA), a metabolite of the essential amino acid L-tryptophan, is a broad spectrum antagonist of excitatory amino acid receptors, which have also anticonvulsant and neuroprotective properties. After spinal cord injury (SCI), excitotoxicity is considered to play a significant role in the processes of secondary tissue destruction in both grey and white matter of the spinal cord. In this study, we have tested the potential therapeutic effect of glucosamine-kynurenic acid, administered after experimental compression-induced SCI in the rat. Spinal application of glucosamine-kynurenic acid continually for 24 hr after experimental SCI resulted in improved motor function recovery, beginning from the first week of evaluation and continuing until the end of the study (4 weeks). After 4 weeks' survival, quantitative morphometric analysis of the spinal cord showed that glucosamine-kynurenic acid treatment was associated with improved tissue preservation at the lesion site. These findings indicate that spinal application of glucosamine-kynurenic acid is neuroprotective and improves the outcome even when administered after spinal trauma. Our results suggest that the treatments initiated in early posttraumatic period can alleviate secondary injury and improve the final outcome after SCI.

Keywords: *Spinal cord injury • Excitotoxicity • NMDA receptor • Kynurenic acid*

1. Introduction

Traumatic spinal cord injury (SCI) is usually caused by excessive forces taking effect on vertebral column. Spinal cord tissues are damaged by displaced bone fragments, intervertebral discs or ligaments. After contusive injury to the spinal cord, pathological changes develop as a two-stage process [1,2]. The primary injury is caused immediately by mechanical damage to neural tissue (including blood vessels), and is followed by secondary processes, leading to posttraumatic autodestruction of spinal cord tissue [3-5]. The mechanisms of secondary injury are not completely understood, but include ischemia, biochemical changes and inflammatory processes contributing to gradual extension of the primary lesion. Histological observations indicate that secondary destruction takes place within the course of days and even weeks after injury [1,5], and that the enlargement of the lesion progresses from the center to the periphery of the spinal cord [6]. Progressive secondary injury involves gradual destruction of the white matter that can play an important role in the final functional deficit after SCI [7]. The detailed understanding of the molecular processes involved in secondary injury is therefore of great importance in SCI research. It would enable more specific therapeutic interventions aimed at minimizing the tissue loss at the injury site.

Studies on the dynamics of the development of secondary changes indicate that the major enlargement of the primary lesion occurs within the first 24 hours after injury. We have therefore proposed that treatments

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