Background context:

Elucidating the mechanism of neuropathic pain (NeP) is crucial as it can result in motor dysfunction and negatively impact quality of life in patients with spinal cord injury (SCI). Although it has been reported that cyclooxygenase 2 (COX2) is involved in NeP in rat models of peripheral nerve injury and that COX2 inhibitors can alleviate NeP, these mechanisms after SCI have not been fully investigated. To investigate the expression of COX2 mRNA and the effect of a COX2 inhibitor on results of the von Frey test in the lumbar spinal cord after thoracic SCI using a rat SCI model.

Methods:

Male SD rats underwent T10 laminectomy under mixed anesthesia, and IH impactors were applied to the same site to create a rat SCI model. Rats that underwent only laminectomy were designated as sham. Lumbar spinal cord at the L4–5 level was harvested at 3, 5, 7, 14, and 28 days after SCI, and COX2 and COX1 were quantified by reverse-transcription PCR (RT-PCR). COX2 expression, expression site, and expression time were determined by immunohistochemistry (IHC) and in situ hybridization histochemistry (ISH) at the same time points. The expression site and time of COX2 expression were also examined at the same time point by ISH. On 5th and 6th day after SCI, saline and COX2 inhibitor (50 μg/day) were administered into the subarachnoid space as a single dose, and the two groups were compared in terms of behavior using the von Frey test.

Results:

COX2 was significantly increased at 5 and 7 days after SCI, but no significant difference in COX1 was observed after SCI. ISH targeting COX2 showed clear expression of COX2 in spinal cord vascular endothelial cells at 5 and 7 days after SCI. COX2 expression was almost abolished at days 14 and 28. Behavioral experiments showed that pain was significantly improved from day 2 after COX2 inhibitor administration compared to the saline group, with improvement up to day 14 after SCI, but no significant difference was observed after day 21.

Conclusions:

The findings suggest that COX2 is increased in vascular endothelial cells in the spinal cord after SCI and that COX2 inhibitors can temporarily improve pain in this SCI model. Therefore, COX2 inhibitors may play an essential role in neuropathic pain.