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The Effect of Early Myelotomy on Glutamate Concentrations in Spinal Cord Injury Rat Model

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ABSTRACT

AIM: To evaluate the effect of early myelotomy on glutamate concentrations in injured spinal cord tissue with a weight drop spinal cord injury rat model.

MATERIAL and METHODS: The rats were assigned randomly to one of four groups, as follows: in group I, rats underwent laminectomy; group II, myelotomy was performed after laminectomy; group III, rats received contusion after laminectomy; and group IV, myelotomy was performed 1 hour after laminectomy and contusion. In order to create a spinal cord injury, a 10-g rod was dropped from a height of 50 mm onto the exposed dura at T10 level. For the myelotomy procedure, a longitudinal 1–1.5 mm depth midline incision was made to the spinal cord. Twelve hours later, rats were decapitated, and the spinal cord tissues were removed. The obtained tissues' glutamate concentrations were measured using the HPLC technique.

RESULTS: The glutamate levels were significantly lower in group III than those of groups I and II. In group IV, glutamate levels were significantly high compared to group III and significantly low compared to group I. Between groups I and II, there was no statistically significant difference.

CONCLUSION: The study results suggest that early myelotomy significantly prevented glutamate depletion from the injured spinal cord. Compared to the normal spinal cord, there was still significant depletion in injured spinal cord with myelotomy because of the initial glutamate release until the myelotomy was performed. It was also concluded that myelotomy was not harmful to the spinal cord as it did not cause significant glutamate depletion.

KEYWORDS: Glutamate excitotoxicity, Spinal cord injury, Secondary injury, Myelotomy, Contusion, Rat

ABBREVIATIONS: SCI: Spinal cord injury, **HPLC:** High performance liquid chromatography, **CSF:** Cerebrospinal fluid

INTRODUCTION

Spinal cord injury (SCI) is a devastating condition that causes impairment of ambulation and sexual and sphincter functions, besides economic consequences for the patient and the community. Primary injury occurs at the time of initial trauma and is most commonly due to compressive-contusive forces caused by the vertebral column displacement. Secondary injury is cellular damage which is set into motion by primary injury. It is a complex cascade of pathophysiologic processes that include apoptosis, ischemia, ion-mediated cell damage and excitotoxicity, neuroinflammation, mitochondrial dysfunction, and oxidative cell damage (6).

Excitotoxicity is one of the important secondary injury mechanisms. After primary injury, excitatory amino acids glutamate and aspartate are released from the cells to the extracellular space. Previously, elevated levels of excitatory amino acids

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have been reported using microdialysis method (15,16). The shift from intracellular to extracellular space causes decreased total tissue levels of excitatory amino acids which was shown with the HPLC method (3,4,21). In SCI, glutamate mainly plays an essential role. In physiological conditions, glutamate is converted to glutamine in astrocytes by ATP-dependent enzyme glutamine synthetase. Glutamine is transported to the nerve endings and is converted back by a mitochondrial enzyme glutaminase to glutamate. Glutamate acts through NMDA and AMPA/KA receptors, which regulates ion channels for Na and/or Ca. Glutamate-induced excitotoxicity causes ion influx and increases intracellular Na and Ca concentrations that lead to cell death (4,6).

Although plenty of researchers focused on these secondary injury mechanisms to find a treatment, there is no sufficient treatment for SCI. Surgical decompression is indicated in case of spinal cord external compression due to bony fracture, dislocation, or hematoma. On the other hand, edema causes increase in spinal cord diameter which decreases volumes of epidural and subarachnoid spaces. This spinal cord internal compression blocks epidural veins, spinal cord arterial supply, and CSF flow, thus further increases edema. These symptoms were named by Cao et al. as "spinal compartment syndrome" inspiring osteofacial compartment syndrome (2). Some surgeons have performed dural release (durotomy) or myelotomy for the spinal cord internal decompression (20). The benefits of longitudinal midline incision of the spinal cord (myelotomy) in contusional SCI were first reported by Allen (1). Since then, few studies have reported clinical improvements after myelotomy in rats with SCI by reducing edema and decreasing the possibility of secondary injuries (18).

Considering the critical role of the glutamate excitotoxicity in secondary injury mechanisms and the promising clinical results of myelotomy in animal studies, the effect of myelotomy on the spinal cord tissue concentrations of glutamate was analyzed in rats with SCI.

MATERIAL and METHODS

Animal Study

Experiments were performed after the study protocol's approval by the Local Animal Research Ethics Committee of Ondokuz Mayıs University (Date:29.05.2008, 2008/21). Twenty-eight male Sprague-Dawley rats weighing 280–300 grams were used. All animals were kept at the same environmental conditions, including temperature, humidity, and light/dark cycles, and had free access to food and water throughout the experiments. Surgical procedures were performed after having the rats anesthetized by intraperitoneal injection of 100 mg/kg ketamine (Ketalar, Eczacibasi, Turkey) and 10 mg/kg xylazine (Rompun, Bayer, Turkey). The rats breathed spontaneously during the procedures. The same surgeon performed all surgical procedures. After the rats' four extremities were pinned to a wooden plate for stabilization, the surgical site was prepared by shaving and disinfecting. At T9-11 levels, a longitudinal midline incision was made, muscles were dissected bilaterally, and laminectomy was performed under an operating microscope. The rats were assigned randomly to one of four groups, as follows: in group I, rats underwent laminectomy (n=7, sham group); group II, myelotomy was performed after laminectomy (n=7, only myelotomy group); group III, rats received contusion after laminectomy (n=7, SCI group); and group IV, myelotomy was performed 1 hour after laminectomy and contusion (n=7, SCI + myelotomy group). In order to create an SCI, after upper and lower spinous processes were clamped for stabilization, a rod (1.5-mm diameter, 10-g weight) was dropped through a perpendicular tube from 50-mm height onto the exposed dura at T10 level. For the myelotomy procedure, the durotomy was performed, and a longitudinal 1–1.5 mm depth midline incision was made to the spinal cord by using an iris scalpel. The myelotomy length was limited to the contusion area in group IV and 5 mm in group II. Durotomy was performed not only in myelotomy groups (group II and IV) but also in groups I and III. After all the procedures were completed, all soft tissue layers were closed, including dura in all groups.

Twelve hours later, rats were euthanatized by decapitation. The spinal cords' contused parts (including 1 mm rostral and 1 mm caudal) of group III and IV rats, 5 mm of the normal spinal cord of group I rats, and 5 mm of myelotomy performed part of the spinal cord of group II rats were removed.

Spinal Cord Tissue Glutamate Assay

Glutamate analysis was performed using the methods described by Pérez-Neri et al. and Diaz-Ruiz et al. with few modifications (4,17).

In a sonicator, the rats' spinal cords were weighed and homogenized with % 85 cold methanol. The samples were centrifuged at –4°C (3,000 g, 15 min), and the supernatants were kept at –80°C until the chromatographic analysis. Prior to analysis, the supernatants were filtered through a 45-µm filter.

For pre-column derivatization, 100 µl of OPA reagent [containing 25 mg phthaldialdehyde (Sigma-Aldrich, Germany)] + 625 µl methanol (Sigma-Aldrich, Germany) + 5.6 ml of 0.4 M borate buffer, pH 9.5 + 25 µl 2-mercaptoethanol (Merck, Germany) were added to 100 µl of each sample. After derivatization, 20-µL aliquots were injected in a HPLC with a fluorescence (HPLC-FLD) detector (Shimadzu Corporation, Kyoto, Japan).

Glutamate analytical standards (purity 99%, Sigma-Aldrich, Germany) were prepared in the mobile phase by serial dilution of standard solution (5 M (0.10 nmol), 4 (0.08 nmol), 3 (0.06 nmol), 2 (0.04 nmol), and 1 M (0.02 nmol)) on a daily basis.

The chromatographic analytical protocol was as follows: a 30-minute linear gradient step from 10% to 35% methanol (A), a 10 min washing step with 99 % A, and a 10 min re-equilibration step with 10% A. A column (3 µm particle size, 100 mm × 4.6 mm, GI Sciences, Japan) was used to separate derivatives. A 1 ml/min flow rate was used. The duration between injections was kept to a minimum of 50 minutes. The results were given in nmol of glutamate/gram of wet tissue.

Statistical Analyses

The data were analyzed using the IBM SPSS version 22.0

for Windows. The Shapiro–Wilk test was used to analyze the glutamate levels' normal distribution assumption, and data were not normally distributed. So, ANOVA was used to compare four experimental groups, and the Mann–Whitney U test was used to compare each two experimental groups, regarding glutamate levels. Descriptive data were presented as mean ± standard deviation (SD) or minimum–maximum (median). P values less than 0.05 were considered statistically significant.

RESULTS

All four groups' glutamate levels are summarized in Table I, as mean ± SD and median values and in Figure 1, as box plots. Glutamate levels were significantly lower in group III than those in groups I and II ($p<0.001$). In group IV, glutamate levels were significantly high compared to group III ($p<0.001$) and significantly low compared to group I ($p<0.001$). Between groups I and II, there was no statistically significant difference ($p>0.05$).

DISCUSSION

Traumatic SCI usually ends up with permanent impairment of motor, sensory, or autonomic functions. Primary injury occurs during the initial impact, but the tissue damage continues with

Table I: Glutamate Levels of All Groups

	Group 1	Group 2	Group 3	Group 4	p*
	mean ± SD median (min-max)	**mean ± SD median (min-max)**	**mean ± SD median (min-max)**	**mean ± SD median (min-max)**	
Glutamate[a,b,c,d,e]	31.92 ± 8.4 31.01 (19.82–43.31)	26.72 ± 4.85 25.46 (20.87–35.65)	16.95 ± 2.56 17.6 (13.62–19.87)	20.66 ± 2.35 21.07 (17.33–23.95)	**<0.001**

SD: *Standard deviation,* ***Min:*** *Minimum;* ***max:*** *Maximum.*
**ANOVA test; [a]Significant difference between group 1 and 3 with Mann–Whitney U test; [b]Significant difference between group 1 and 4 with Mann–Whitney U test; [c]Significant difference between group 2 and 3 with Mann–Whitney U test; [d]Significant difference between group 2 and 4 with Mann–Whitney U test; [e]Significant difference between group 3 and 4 with Mann–Whitney U test.*

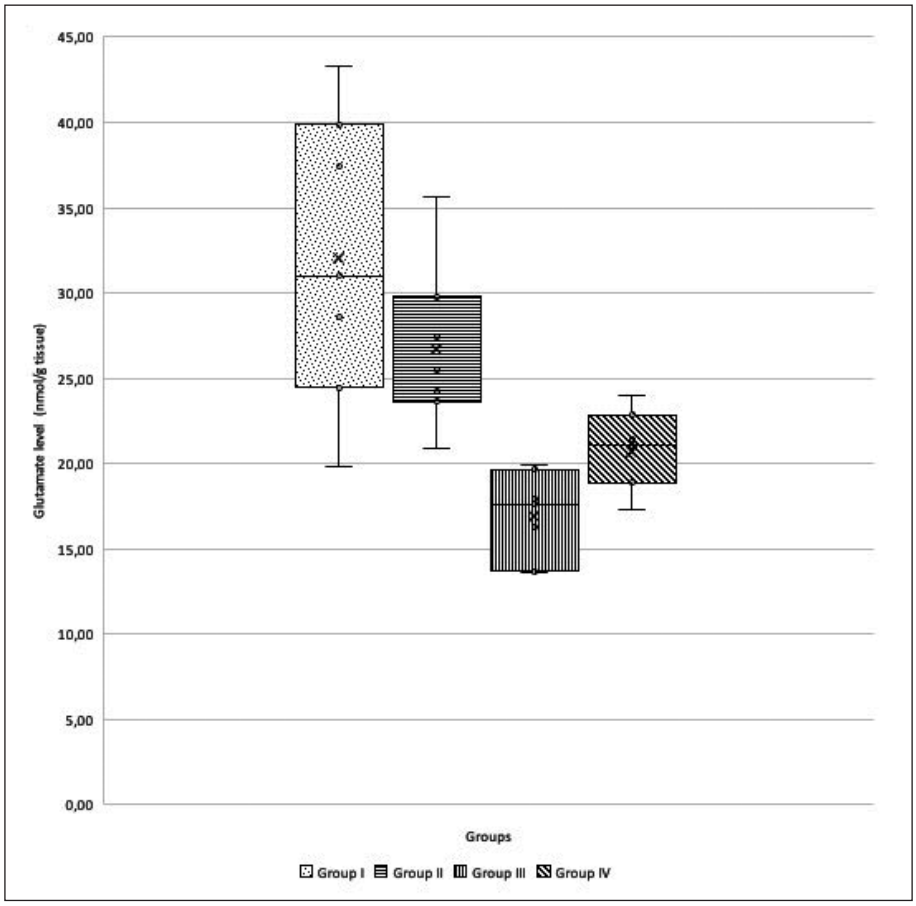

Figure 1: Box plots of all groups (dotted, group I; horizontal stripes, group II; vertical stripes, group III; diagonal stripes, group IV).

secondary injury mechanisms such as vascular impairment, ischemia, glutamate excitotoxicity, apoptosis, and oxidative cell damage. There is no sufficient treatment for SCI. Nothing can be done with primary injury once it has happened. So, all efforts about the treatment should be focused on the prevention of secondary injury. Excitotoxicity is an important mechanism of secondary injury in SCI. Glutamate is the most important excitatory amino acid, and it acts through NMDA, AMPA/Kainate, and metabotropic glutamate receptors. In pathological conditions, excessive glutamate causes cell death by increasing Ca++ concentration in the cell. In previous studies, both necrosis-like cell death and apoptosis-like cell death have been shown. The role of nitric oxide, free radicals, and caspases has been reported (13). Excessive amount of glutamate is released to the extracellular compartment at the acute phase of SCI. The release or leakage of glutamate causes its decreased cellular concentrations. Increased levels of extracellular glutamate have been reported using the micro dialysis technique (14-16). Reciprocal decrease of cellular or tissue glutamate levels after SCI has been published by Demediuk et al. (3). Watanabe and Diaz-Ruiz confirmed decreased tissue levels after SCI with the HPLC technique (4, 21). In this study, decreased tissue glutamate levels after SCI were concordant with the mentioned studies. The glutamate levels were significantly lower in rats that had SCI without myelotomy (group III) than those in rats without SCI (groups I and II).

When the effect of myelotomy on glutamate excitotoxicity in SCI was evaluated, the glutamate levels were significantly higher in rats that had SCI with myelotomy (group IV) compared to rats that had SCI without myelotomy (group III). It can be suggested that myelotomy prevented such a decrease in tissue glutamate levels; in other words, it prevented glutamate shift from cells to the extracellular fluid.

Although myelotomy kept glutamate inside cells in certain degrees, there was a significant decrease in tissue glutamate levels in rats with SCI with myelotomy (group IV) compared to rats without SCI (groups I and II). This difference represents the glutamate release starting from the initial impact. It was thought that myelotomy performed one hour after SCI prevented further release. Even if myelotomy significantly prevented further glutamate depletion, it seems to be a partial solution for glutamate excitotoxicity because of the initial release. It has been shown that in rats with SCI, decreasing the effect of glutamate excitotoxicity with glutamate receptor antagonists improved locomotor function (5,22). We can assume that a significant decrease of glutamate release after myelotomy may have some benefits on locomotor function.

Secondary injury is a continuing process, and it causes edema and hemorrhagic necrosis, which increase pressure inside the injured cord. The surrounding dura and bones do not allow spinal cord enlargement beyond certain limits. The increasing pressure blocks epidural veins, spinal cord arterial supply, and CSF flow and causes spinal compartment syndrome which worsens the clinical picture (2). Removing fractured bones may relieve the pressure on the epidural veins and spinal cord, and it is indicated in case of neurological deficit. Some researchers advocate further decompression by adding the durotomy technique to the bony decompression. However, there are publications claiming bony decompression and/or durotomy is/are incomplete or partial decompression techniques that do not decrease the pressure inside the injured cord as effective as the myelotomy technique (11). Effectively relieving the pressure inside the injured cord has long been the researchers' interest since Allen first reported the benefits of myelotomy a century ago (1). Several animal studies have reported the beneficial effects of myelotomy for motor functions in SCI. Kalderon et al. reported that myelotomy improved structural outcome and locomotor function (10), and Yang et al. and Hu et al. also published the beneficial effects of myelotomy on locomotor functions (7,23).

In animal studies, there is no standard for myelotomy technique. Rivlin and Tator compared half-depth and full-depth myelotomy and reported that while full-depth myelotomy significantly improved motor functions, half-depth myelotomy had no such effect (19). On the other hand, Kalderon et al. performed partial myelotomy by stabbing a needle at 5 points along the midline of the lesion site, which improved locomotor functions (10). Yang et al. penetrated into the spinal cord approximately 1–1.5 mm for myelotomy, and they also reported improvements in locomotor functions (23). Another debate about myelotomy is optimal timing. Yang et al. suggested that myelotomy was effective up to 48 hours, but optimal timing might be between 8 and 24 hours after SCI (23). There are publications suggesting earlier timing for myelotomy such as 1–4 hours after SCI (8-10).

Myelotomy not only relieves the intramedullary pressure, but also reduces the effects of blood or accumulated fluid within the injured spinal cord. Almost all of the animal studies about myelotomy evaluated either locomotor function or the injured tissue's histopathology. Hu et al. showed that myelotomy reduced the edema, and it was associated with decreased expression of aquaporin-4 and aquaporin-9, which had essential roles in the regulation of water homeostasis (7). The current study is the first study which evaluates the effect of myelotomy on secondary injury mechanisms other than edema, particularly the glutamate excitotoxicity. It was found that myelotomy prevented glutamate shift from the spinal cord cells to the extracellular space. The glutamate release after SCI is proportional with the degree of trauma and maximum at 4–8 hours after trauma (4). Secondary injury mechanisms that increase intramedullary pressure such as edema exacerbate the effect of primary injury. Even if we could not entirely explain the mechanism, early myelotomy might have prevented further glutamate release by relieving the intramedullary pressure.

Although myelotomy has been regularly used to remove spinal cord tumors, fear of further damage limited the use of this technique in SCI treatment. There are only a few clinical reports about myelotomy after SCI. Koyanagi et al. reported the results of four patients in which myelotomy was performed at 5 to 21 hours after SCI (12). None of the patients showed motor improvement on their lower extremities, but the two patients' upper extremities were better. Zhu et al. reported the results of 30 patients with complete SCI (ASIA-A) (24). After

myelotomy and early rehabilitation, all the patients were able to walk at least with a wheeled weight support and a weight-bearing knee joint stabilizer. The optimal time for surgery was 4–14 days after SCI. According to the animal studies, myelotomy does not cause further damage in the injured spinal cord (18). Since there are not enough clinical studies supporting animal studies, most of the surgeons do not prefer to perform myelotomy for SCI treatment.

In the current study, the tissue glutamate levels were lower in rats with laminectomy and myelotomy (group II) compared to those with laminectomy (group I), but this difference was not statistically significant. These results suggest that the injury caused by myelotomy in the spinal cord is not remarkable to cause significant glutamate release. It can be said that myelotomy may not aggravate secondary injury mechanisms, particularly the glutamate excitotoxicity.

There are some limitations of this study. As mentioned before, several studies have reported that neutralizing glutamate effect by using glutamate receptor antagonists improves locomotor functions in rats after SCI (5,22). The benefits of myelotomy on locomotor functions after SCI in rats were also reported in several articles (7,10,19,23). It would be better to support our results with the clinical findings of rats. However, even in that case, we would not explain the clinical results of myelotomy if they were only the results of decreased glutamate release or the other complex cascades of secondary injury mechanisms were involved. Another limitation was that computer-assisted impactor was not used to create SCI, but standard SCI was created as much as possible using the weight drop method as mentioned in the material and methods section.

On the other hand, the current study results are valuable because the effect of myelotomy in SCI and in normal spinal cord tissue was presented regarding glutamate tissue concentrations.

CONCLUSION

In this study, the effect of myelotomy on glutamate excitotoxicity was evaluated using the weight drop SCI model in rats. The results suggest that early myelotomy significantly prevented glutamate depletion from the injured spinal cord. There was still significant depletion in myelotomy performed injured spinal cord compared to the normal spinal cord, because of the initial release of glutamate until the myelotomy was performed. We can also conclude that myelotomy was not harmful for the spinal cord as it did not cause significant glutamate depletion.

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AUTHORSHIP CONTRIBUTION

Study conception and design: AU, AHK

Data collection: AU, DG

Analysis and interpretation of results: AU, AHK

Draft manuscript preparation: AU

Critical revision of the article: AU, AA, AS

All authors (AU, AHK, DG, AA, AS) reviewed the results and approved the final version of the manuscript.

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