EFFECT OF LIDOCAINE ON SPINAL CORD LIPID PEROXIDE LEVELS AFTER ACUTE SPINAL CORD TRAUMA IN RATS

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A.S. Yalçın, Ph.D. ** / F. Özer, M.D. *** / N. Pamir, M.D. *** / K. Emerk, Ph.D. *

* *Professor, Department of Biochemistry, Faculty of Medicine, Marmara University, İstanbul, Turkey.*
** *Associate Professor, Department of Biochemistry, Faculty of Medicine, Marmara University, İstanbul, Turkey*
*** *Associate Professor, Department of Neurosurgery, Faculty of Medicine, Marmara University, İstanbul, Turkey*

SUMMARY

A standard spinal cord trauma was performed on control and lidocaine-treated (5 mg/kg. i.p.) rats. Spinal cord lipid peroxide levels in the lidocaine-treated group were significantly lower than those of controls. No significant difference was observed in plasma lipid peroxide levels. Our results suggest a protective role of lidocaine against lipid peroxidation after experimental spinal cord trauma in rats.

Key Words: Lidocaine - treatment, Lipid peroxidation, Spinal cord trauma, Rats.

INTRODUCTION

It has been suggested that oxygen derived free radicals and lipid peroxidation are among the causes of neurodegenerative changes in various central nervous system disorders including ischemia and trauma (1). This is in part due to the presence of high amounts of phospholipids and polyunsaturated fatty acids which are susceptible to free radical attact and peroxidative changes. Demopoulos et al. (2) have hypothesized a scheme of biochemical events that may lead to spinal cord injury. In this scheme, production of various free radical species such as superoxide radical, hydrogen peroxide and hydroxyl radical are postulated to have a central role.

A system of biochemical defenses has evolved to protect organisms and cells from the free radical damage which is possible in an oxidizing environment (3). These defenses serve to lower the concentrations of free radical species, which might otherwise cause excessive damage to cell components. Free radicals may be quenched by reactions with small molecules. The most important low molecular weight free radical scavengers are tocopherols, ascorbate, carotenoids and glutathione. In addition, a set of enzymes including superoxide dismutase, catalase and glutathione peroxidase scavenge superoxide, hydrogen peroxide and lipid peroxides.

On the other hand, spinal cord injury models have been treated with lipid soluble barbiturates which successfully prevented free radical changes (2). Astrup et al. (4) have reported that lidocaine has a barbiturate-like effect in addition to its membrane stabilizing properties. Therefore, in the present study we have investigated the effect of lidocaine-treatment on lipid peroxide levels in traumatized rat spinal cord tissue.

MATERIALS AND METHODS

Wistar strain rats weighing 300-350 g were divided into control and lidocaine-treated groups. A standard spinal cord trauma described by Dolan and Tator (5) was performed on both groups. Lidocaine-treated group received lidocaine (5 mg/kg, i.p.) 15 minutes before operation.

Surgical procedure

Rats were anesthetized using ketamine and pinned in supine position. Following T3-T11 midline incision, T4-T10 spinous process and laminar arcs were removed under the surgical microscope after paravertebral muscle dissection. The exposed spinal cord was squeezed by Yaşargil aneurysm clip for 45 seconds and then removed by cutting at the two ends. By this procedure it was possible to standardize spinal cord trauma in all rats. The presence of trauma was confirmed by histopathological findings.

Lipid peroxide determination

The degree of lipid peroxide formation was assayed by monitoring thiobarbituric acid reactive substance

formation (6). Briefly, tissue samples were homogenized in ice-cold trichloracetic acid (1 g tissue plus 10 ml 10% TCA) in an Ultra Turrax tissue homogenizer. After centrifugation, a volume of the supernatant was added to an equal volume of 0.67 % thiobarbituric acid and the mixture was kept in a boiling water bath for 15 minutes. Samples were cooled to room temperature and absorbances were measured at 532 nm. Plasma lipid peroxide levels were determined using the procedure of Yagi (7). Tissue and plasma lipid peroxide levels were expressed in terms of malondialdehyde (MDA) equivalents using an extinction coefficient of $1.56 \times 10^5\ M^{-1}\ cm^{-1}$.

RESULTS AND DISCUSSION

Table I shows plasma and spinal cord lipid peroxide levels after experimental spinal cord trauma in control and lidocaine-treated rats. Spinal cord lipid peroxide levels in the lidocaine-treated group were significantly lower than those of controls ($P < 0.001$). No significant difference was observed in plasma lipid peroxide levels.

TABLE I **Plasma and spinal cord lipid peroxide levels after acute spinal cord trauma in control and lidocaine - treated rats**

	CONTROL	LIDOCAINE-TREATED
PLASMA (nmol MDA/ml plasma)	4.8 ± 0.9	4.5 ± 1.1
SPINAL CORD (nmol MDA/g tissue)	17.0 ± 3.7	11.8 ± 2.3*

Values represent mean ± SD of ten experiments
* Significantly different from the controls ($P<0.001$)

The amount of low molecular weight free radical scavengers and antioxidant enzymes is rather low in cerebrospinal fluid (1). For this reason, it is very difficult to prevent oxidative damage in the central nervous system and agents including mannitol, barbiturates, corticosteroids have been used as exogenous free radical scavengers to support endogenous defenses against oxidative changes (2). Since it was observed that lidocaine had a barbiturate-like effect in addition to its membrane stabilizing properties (4), we have attempted to investigate whether it has any effect on lipid peroxidation in spinal cord tissue. Our results suggest a protective role of lidocaine against lipid peroxidation in experimental spinal cord trauma. However, further studies are required to elucidate the mechanism of this protection.

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