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Investigation of the Effectiveness of Sildenafil in Rats with Experimental Spinal Cord Trauma

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Abstract: ***Background*:** Traumatic spinal cord injury (TSCI) initiates a complex secondary injury cascade characterized by oxidative stress, inflammatory activation, and progressive tissue destruction. Sildenafil, a selective phosphodiesterase-5 inhibitor, has been reported to exert neuroprotective effects through modulation of cyclic guanosine monophosphate-mediated pathways. This study aimed to investigate the biochemical effects of sildenafil in an experimental rat model of spinal cord trauma. ***Methods*:** Twenty-eight adult Sprague-Dawley rats were randomly allocated to four groups (n=7 each): control, trauma, trauma + saline, and trauma + sildenafil. After T8–T10 laminectomy, spinal cord injury was induced by epidural clip compression using a 1.43 N aneurysm clip applied for 60 seconds. Sildenafil citrate was administered intraperitoneally at a single dose of 10 mg/kg immediately after trauma. At 48 hours, plasma and spinal cord tissue samples were obtained. Malondialdehyde (MDA) and total antioxidant capacity (TAOC) levels were measured as indicators of oxidative stress and antioxidant defense. ***Results*:** Plasma MDA levels differed significantly among groups and increased markedly after trauma, whereas sildenafil treatment attenuated this increase. Tissue MDA levels were also significantly elevated in the trauma groups but were lower in the sildenafil-treated group. Tissue TAOC levels decreased after trauma and increased toward control values following sildenafil administration. In contrast, plasma TAOC levels did not differ significantly among groups. Post hoc analysis indicated that the principal differences were between the trauma and treatment groups. ***Conclusion*:** Sildenafil reduced oxidative stress-associated biochemical damage in experimental spinal cord trauma and improved tissue antioxidant capacity. These findings suggest that sildenafil may be a promising neuroprotective agent for limiting secondary injury after spinal cord trauma.

Keywords: Spinal cord injury, Sildenafil, Oxidative stress, Malondialdehyde, Total antioxidant capacity, Rat model.

Research Paper

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1. INTRODUCTION

Traumatic spinal cord injury (TSI) is a severe neurological disorder that often results in permanent loss of motor, sensory, and autonomic functions. The condition imposes a substantial burden upon individuals and society due to its high morbidity and the extensive need for long-term care [1, 2]. In 2019, approximately 0.9 million new cases were reported globally, affecting a total of 20.6 million people [2]. These figures indicate that traumatic spinal cord injury is not exclusively an acute neurosurgical concern but amounts to a significant public health issue associated with long-term disability and diminished quality of life [1, 2].

In traumatic spinal cord injury, tissue destruction goes beyond the primary injury caused by the initial mechanical impact [1]. Secondary damage worsens the lesion by impairing vascular permeability, inducing ionic imbalance, promoting glutamate excitotoxicity, impairing cellular metabolism, initiating inflammatory responses, and leading to glial scarring. Oxidative stress and reactive oxygen species are critical contributors to secondary injury, increasing neuronal and glial cell loss. These processes obstruct recovery via mechanisms such as apoptosis, autophagy, ferroptosis, and other forms of regulated cell death [3, 4]. Consequently, current research approaches focus on suppressing secondary damage and preserving neural

tissue rather than addressing irreversible primary injury [1-3].

The importance of early surgical decompression remains central in managing acute traumatic spinal cord injury, especially compared to pharmacological options, where no treatment has yet emerged to substantially change clinical practice in neuroprotection against secondary damage [5, 6]. Methylprednisolone is historically the most debated agent in this field [7]. However, it remains controversial in current guidelines due to conflicting efficacy and safety data, underscoring the need for new, biologically justified agents capable of modulating various stages of secondary injury [5].

Sildenafil, a selective phosphodiesterase-5 (PDE5) inhibitor, elevates cyclic guanosine monophosphate (cGMP) levels. Preclinical studies indicate that sildenafil can cross the central nervous system, influence neurovascular responses, and modulate the biological processes underlying neural damage [8–11]. In experimental models of neuroinflammation and central nervous system injury, sildenafil has been shown to reduce cytokine responses, suppress microglial activation, and exert anti-inflammatory and neuroprotective effects [9-12]. In spinal cord injury models, sildenafil increases intraspinal cGMP levels, enhances microvascular perfusion in contusion-type injuries in mice [13], reduces neurological damage in transient spinal cord ischemia models [14], and improves histopathological and biochemical markers in trauma models. Similarly, tadalafil, another PDE5 inhibitor, has demonstrated improvements in neurological and biochemical parameters in a rat spinal trauma model [15]. Collectively, the results support the therapeutic possibility of targeting the PDE5/cGMP axis in spinal cord trauma.

This study intends to evaluate the neuroprotective efficacy of sildenafil in rats with experimentally induced spinal cord trauma. Specifically, the investigation will assess whether sildenafil reduces secondary damage, preserves tissue stability, and promotes functional recovery.

2. MATERIAL METHODS

Study Design and Ethical Approval

We conducted this animal study at a university research center in Türkiye with approved ethical approval (25.11.2009, 131/2009). We used adult Sprague-Dawley rats (200-250 g), housed under normal conditions with free access to food and water. After trauma, we kept the animals in separate cages and monitored them daily.

Anesthesia and Surgical Preparation

We performed all surgeries under aseptic conditions. We anesthetized rats by intraperitoneal injection of 60 mg/kg ketamine hydrochloride (Ketalar®) and 12 mg/kg xylazine (Rompun®). After anesthesia, we placed the rats on a fixation board, shaved the dorsal thoracic region, and disinfected the area. We performed local field cleaning and draped the T9–T11 region. We incised the skin and fascia, separated the paravertebral muscles, and performed a laminectomy at T8–T10 to expose the spinal cord.

Experimental Spinal Cord Trauma Model

We induced spinal cord trauma by epidurally applying a Yaşargil FE 740K aneurysm clip (Aesculap AG, Germany) with a force of 1.43 N for 60 seconds. All animals except controls showed paraplegia after trauma. This clip-compression method provides consistent thoracic spinal cord injury [16].

Experimental Groups

We randomly assigned the animals to four groups of 7 rats each before trauma.

- Group 1 (Control group): Only a laminectomy was performed. We did not induce spinal cord trauma or give any treatment. We collected blood and spinal cord samples after 48 hours to set baseline biochemical levels.
- Group 2 (Trauma group): We performed a laminectomy, then induced spinal cord trauma using the aneurysm clip for 1 minute. We collected blood and spinal cord samples after 48 hours to assess the trauma's biochemical effects.
- Group 3 (Trauma + solvent group): After trauma, we gave 1 cc of saline solution intraperitoneally. We collected samples at 48 hours to evaluate the solvent's effect on biochemical parameters.
- Group 4 (Treatment group): After trauma, we administered 10 mg/kg intraperitoneal sildenafil citrate (Fako Pharmaceuticals Inc.; Actavis Group/Istanbul–Matrix Laboratories/India). We collected samples 48 hours after treatment to evaluate its effects.

Monitoring, Sacrifice, and Sampling

We monitored nutritional status and motor activity daily. We sacrificed all rats under deep anesthesia 48 hours after the procedure. We collected 3–5 mL of blood intracardially using an 18-G syringe, centrifuged it at 3000 rpm for 10 minutes, and separated plasma for MDA and TAOC analysis. We removed a 1-cm spinal cord segment encompassing the injury. We stored all samples at -80°C for biochemical analysis.

Biochemical Analyses

MDA levels, an indicator of lipid peroxidation, were determined using the method of Ohkawa *et al*., This method measures the color formed by MDA's reaction with thiobarbituric acid at 532 nm. Results were given as nmol/mg protein in tissue and nmol/mL in plasma [17].

We measured total antioxidant capacity using the Rel Assay TAS kit and Erel's colorimetric method on an automated analyzer. We recorded results as mmol Trolox equivalent/L [18].

Endpoint Measures

The primary endpoints were plasma and spinal cord MDA and TAOC levels at 48 hours post-trauma. We evaluated sildenafil's effects on oxidative stress and antioxidant defense by comparing them with the trauma and trauma + solvent groups.

Statistical Analysis

We conducted analyses using SPSS for Windows 25.0 (IBM Corp., Armonk, NY). We presented continuous variables as mean ± standard deviation. We used one-way ANOVA to compare groups and, if significant, performed LSD post hoc tests. We set significance at $p < 0.05$ (95% confidence).

3. RESULTS

Twenty-eight rats were assigned to four groups, with seven animals per group. Malondialdehyde (MDA) and total antioxidant capacity (TAOC) levels were measured in both plasma and spinal cord tissue. Group means and results of analysis of variance are summarized in Table 1.

Table 1: Plasma and tissue MDA and TAOC levels by group

	Control (Group 1)	Trauma (Group 2)	Trauma + Saline (Group 3)	Trauma + Sildenafil (Group 4)	F	p
MDA Plasma	2,94±1,69	8,95±1,97	9,48±1,84	6,14±1,55	20,04	<0,01
TAOK Plasma	1,80±0,09	1,58±0,11	1,69±0,45	1,71±0,07	0,94	0,43
MDA Tissue	0,55±0,27	3,08±3,86	5,80±3,57	2,39±0,86	4,64	0,01
TAOK Tissue	0,30±0,04	0,24±0,03	0,23±0,05	0,29±0,04	4,74	0,01

Data are presented as mean ± standard deviation. One-way analysis of variance (ANOVA) was used for intergroup comparisons. Post hoc evaluation was performed using the LSD test.

Plasma MDA levels differed significantly among groups ($F=20.04$; $p<0.001$), with control animals exhibiting 2.94±1.69 nmol/mL and the sildenafil group showing 6.14±1.55 nmol/mL (Figure 1). No significant differences were observed in plasma TAOC levels ($F=0.94$; $p=0.43$), with mean values of 1.80±0.09, 1.58±0.11, 1.69±0.45, and 1.71±0.07 mmol Trolox Eq/L (Figure 1). Tissue MDA levels also differed significantly ($F=4.64$; $p=0.01$), with controls at 0.55±0.27 nmol/mg protein and the sildenafil group at 2.39±0.86 nmol/mg protein (Figure 2). Tissue TAOC levels were significantly different as well ($F=4.74$; $p=0.01$), with controls at 0.30±0.04 mmol Trolox Eq/L and the sildenafil group at 0.29±0.04 mmol Trolox Eq/L (Figure 2). Both plasma and tissue MDA levels increased following trauma, while sildenafil treatment attenuated this increase and reversed the trauma-induced decrease in tissue TAOC. No significant effect of sildenafil was observed on plasma TAOC. Post hoc least significant difference (LSD) analysis indicated that significant differences were primarily between the trauma and treatment groups ($p<0.05$).

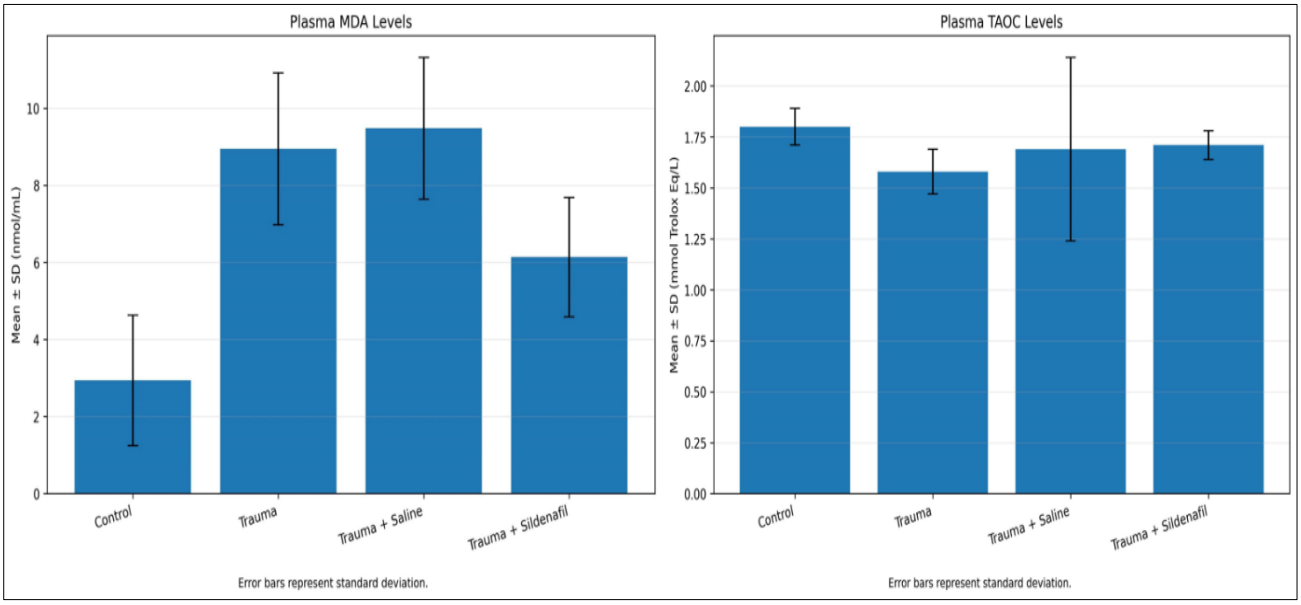

Figure 1: Plasma MDA and TAOC levels in the study groups

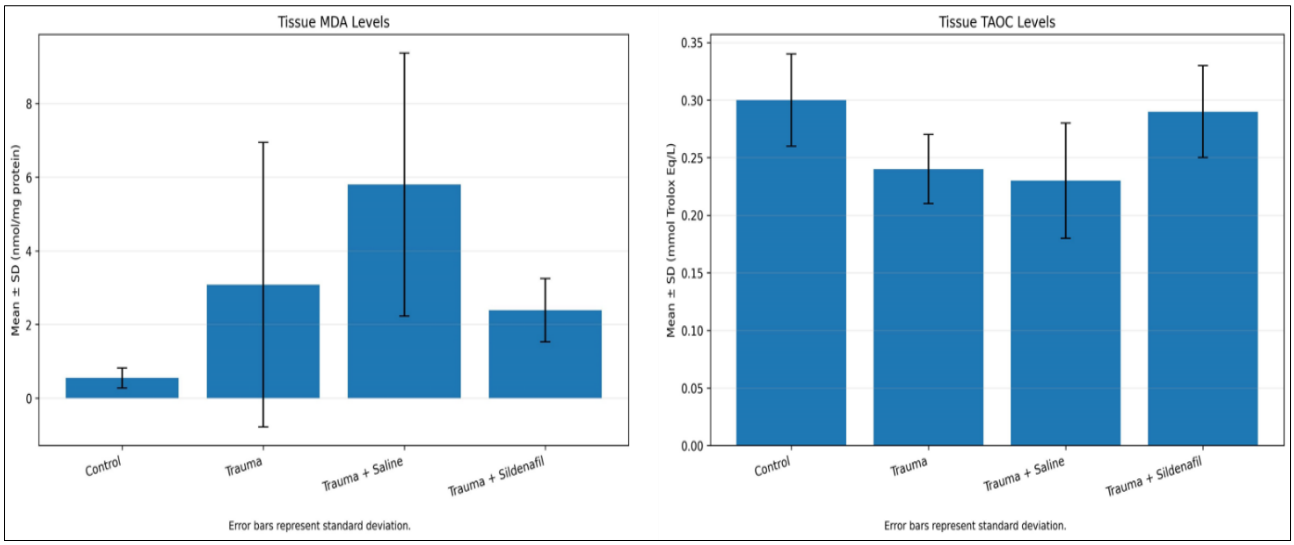

Figure 2: Spinal cord tissue MDA and TAOC levels in the study groups

4. DISCUSSION

The results show that plasma and tissue MDA levels increase, while tissue TAOC levels decrease, following experimental spinal cord trauma compared with controls. Administration of a single intraperitoneal dose of sildenafil after trauma reduces plasma and tissue MDA levels and shifts tissue TAOC levels toward control values, relative to the trauma and saline groups. The comparable findings between the trauma and saline groups confirm that the observed effect is attributable to the active agent rather than the carrier. Although plasma TAOC does not improve with sildenafil treatment, tissue TAOC does, suggesting that sildenafil primarily enhances tissue rather instead of systemic antioxidant capacity during the early acute phase.

In traumatic spinal cord injury, secondary injury following the initial insult exacerbates tissue damage via mechanisms such as inflammation, cellular stress, accumulation of toxic metabolites, and cell death [4-19]. Oxidative stress is recognized as a central contributor to these processes. The accumulation of reactive oxygen species and increased cell membrane damage are associated with mitochondrial dysfunction and neuronal loss. The present studies identified increased cell membrane damage and diminished antioxidant defenses during the early stages of spinal cord trauma [3-21]. Post-trauma, MDA levels rise and tissue TAOC decreases [20-22]. Sildenafil treatment reduces MDA and improves tissue TAOC, showing its power to lessen the detrimental effects of oxidative stress during secondary injury.

Sildenafil increases cGMP levels by inhibiting phosphodiesterase-5 (PDE5), which is present in various central nervous system cells, including neurons and glial cells [9]. The drug has demonstrated neuroprotective and restorative effects, such as reducing cell death, limiting microglial and astrocytic activation, and normalizing inflammatory responses. Additionally, sildenafil supports restorative pathways in nerve injury models [23, 24]. These mechanisms may account for the significant tissue-level redox recovery observed in this study, suggesting that sildenafil functions not only as a vasodilator but also as an encouraging neuroprotective agent.

These outcomes are consistent with previous studies demonstrating the possible benefits of sildenafil in spinal cord injury. Kıymaz *et al*., reported that administration of 10 mg/kg sildenafil at reperfusion significantly improved neurological scores, somatosensory evoked potentials, and histopathological outcomes in a transient spinal cord ischemia model [13]. Myers *et al*., found that sildenafil increased epicentral microvascular perfusion in mice with contusion-type spinal cord injury, although this did not result in improved hindlimb functional recovery [10]. In a rabbit trauma model, Kara *et al*., observed significant histological improvements in the sildenafil group compared to trauma controls, including augmented morphological integrity, leptomeningeal preservation, and reduced hematoma and necrosis in both white and gray matter [14]. Although the present results do not directly address functional or histological outcomes, they contribute to the literature by providing biochemical evidence that sildenafil suppresses oxidative secondary damage during the acute phase.

In this study, plasma TAOC levels did not change significantly, whereas tissue TAOC levels recovered in the sildenafil group. This finding indicates that the acute redox imbalance at 48 hours may be localized to the lesion microenvironment rather instead of systemic circulation. Current empirical studies stress that improvements in serum oxidative markers alone are insufficient to demonstrate biological recovery. Biochemical data should be corroborated by histopathological or functional outcomes. Therefore, although the present data provide a strong biochemical

signal supporting sildenafil, the effects on tissue preservation and neurological function call for further study.

The clinical significance of these data lies in the absence of effective pharmacological treatments capable of reversing spinal cord damage following traumatic injury [25–27]. Current translational and clinical research indicates promise for therapies targeting secondary damage, though single-target approaches are often limited in efficacy. Multimodal strategies that simultaneously address antioxidant, anti-inflammatory, vascular, and regenerative pathways are considered more feasible [28, 29]. Sildenafil supports cGMP-mediated vascular responses and reduces neuroinflammation and cell death in various central nervous system models, making it a potential candidate for repositioning in the treatment of spinal cord trauma [9-23].

The main limitations of this study include the use of only seven animals per group and administration of a single 10 mg/kg intraperitoneal dose of sildenafil following trauma. The follow-up period was limited to 48 hours, and assessments were restricted to biochemical markers such as MDA and TAOC. The absence of histopathological, behavioral, or inflammatory marker evaluations precludes direct confirmation of structural and functional recovery. Additionally, the clip-compression trauma model at T8–T10 focuses on acute secondary damage and does not encompass the full spectrum of human spinal cord injury. Future studies ought to address dose-response relationships, repeated dosing, and extended follow-up, and incorporate histopathological, behavioral, and mechanistic biomarkers such as Nrf2/HO-1, NF-κB, and caspase [25-29].

In conclusion, this study shows that sildenafil exerts early effects in a model of experimental spinal cord trauma by decreasing oxidative stress and supporting tissue antioxidant capacity. These data indicate that sildenafil may be a candidate neuroprotective agent for limiting secondary damage. However, further histological, functional, and multi-period validation studies are required to establish clinical relevance clinical conclusions.

Ethical Statement:

Ethical approvals were obtained from the ethics committee of the healthcare institution where the study was conducted. Compliance with the ethical principles of the Declaration of Helsinki was essential at every stage. No participants were forced to participate in the study.

Data Availability Statement:

The datasets generated and analyzed during the current study are not publicly available due to patient privacy restrictions but are available from the corresponding author on reasonable request.

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