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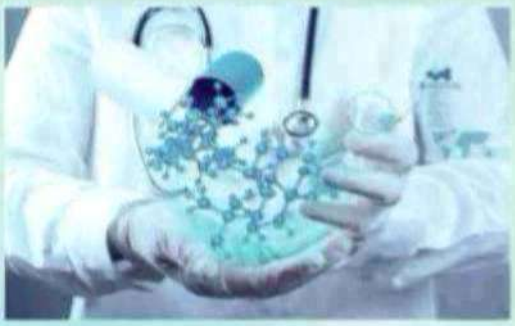

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COMPARATIVE CHARACTERISTICS OF MORPHOMETRIC PARAMETERS OF THE SMALL INTESTINE IN SPINAL CORD INJURIES

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✓ ***Resume***

Spinal cord injury (SCI) triggers complex systemic neurogenic and hemodynamic dysfunctions that extend beyond motor and sensory deficits, profoundly affecting gastrointestinal visceral regulation and mucosal integrity. This study aimed to perform a comprehensive comparative analysis of the morphometric parameters across distinct segments of the small intestine (duodenum, jejunum, and ileum) under conditions of experimental spinal cord injury. Utilizing standardized histological processing and quantitative morphometry, key structural indices including mucosal thickness, villus height, crypt depth, villus-to-crypt ratio, and muscularis layer thickness were evaluated in an SCI model and compared against intact controls. Ultimately, spinal cord injury induces profound segment-specific morphometric disruption and mucosal atrophy throughout the small intestine, providing quantitative criteria that underscore the critical need for early therapeutic interventions aimed at preserving intestinal barrier function following spinal neurotrauma.

Keywords: spinal cord injury, small intestine, morphometry, villus height, crypt depth, mucosal atrophy, neurogenic bowel dysfunction.

СРАВНИТЕЛЬНАЯ ХАРАКТЕРИСТИКА МОРФОМЕТРИЧЕСКИХ ПАРАМЕТРОВ ТОНКОЙ КИШКИ ПРИ ПОВРЕЖДЕНИЯХ СПИННОГО МОЗГА

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✓ ***Резюме***

Травма спинного мозга (ТСМ) вызывает сложные системные нейрогенные и гемодинамические нарушения, которые выходят за рамки двигательного и сенсорного дефицита, оказывая глубокое влияние на висцеральную регуляцию желудочно-кишечного тракта и целостность слизистых оболочек. Целью данного исследования было проведение всестороннего сравнительного анализа морфометрических параметров различных сегментов тонкой кишки (двенадцатиперстной, тощей и подвздошной) в условиях экспериментального повреждения спинного мозга. Используя стандартизированную гистологическую обработку и количественную морфометрию, ключевые структурные показатели, включая толщину слизистой оболочки, высоту ворсинок, глубину крипт, соотношение ворсинок к криптам и толщину мышечного слоя, были оценены в модели SCI и сравнены с интактным контролем. В конечном счете, повреждение спинного мозга вызывает глубокие сегментарные морфометрические нарушения и атрофию слизистой оболочки по всему тонкому кишечнику, обеспечивая количественные критерии, которые подчеркивают острую необходимость ранних терапевтических вмешательств, направленных на сохранение барьерной функции кишечника после нейротравмы позвоночника.

Ключевые слова: повреждение спинного мозга, тонкая кишка, морфометрия, высота ворсинок, глубина крипт, атрофия слизистой оболочки, нейрогенная дисфункция кишечника.

ОРҚА МИЯ ШИКАСТЛАНИШЛАРИДА ИНГИЧКА ИЧАКНИНГ МОРФОМЕТРИК ПАРАМЕТРЛАРИНИНГ ҚИЁСИЙ ХУСУСИЯТЛАРИ

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✓ ***Резюме***

Орқа мия шикастланиши (ОМШ) ошқозон-ичак висцерал регуляцияси ва шиллиқ қаватининг яхлитлигига чуқур таъсир кўрсатадиган восита ва ҳиссий нуқсонлардан ташқарига чиқадиган мураккаб тизимли нейроген ва гемодинамик дисфункцияларни келтириб чиқаради. Ушбу тадқиқот экспериментал орқа мия шикастланиши шароитида ингичка ичакнинг (ўн икки бармоқли ичак, ингичка ичак ва ёнбош ичак) алоҳида сегментлари бўйича морфометрик параметрларни ҳар томонлама қиёсий таҳлил қилишни мақсад қилинган. Стандартлаштирилган гистологик ишлов бериш ва миқдорий морфометриядан фойдаланиб, шиллиқ қаватнинг қалинлиги, ворсинка баландлиги, крипт чуқурлиги, ворсинка-крипт нисбати ва мускул қатламининг қалинлиги SCI моделида баҳоланди ва бузилмаган бошқарув элементлари билан таққосланди. Охир оқибат, орқа мия шикастланиши ингичка ичак бўйлаб чуқур сегментга хос морфометрик бузилиш ва шиллиқ атрофияни келтириб чиқаради, бу эса умуртқа нейротравмадан кейин ичак тўсиғи функциясини сақлашга қаратилган эрта терапевтик аралашувлар учун жуда муҳим эҳтиёжни таъкидлайдиган миқдорий мезонларни таъминлайди.

Калит сўзлар: орқа мия шикастланиши, ингичка ичак, морфометрия, виллус баландлиги, крипт чуқурлиги, шиллиқ қават атрофияси, нейроген ичак дисфункцияси.

Relevance

Global epidemiological estimates from the World Health Organization indicate an annual incidence of 250,000 to 500,000 new spinal cord injury (SCI) cases, contributing to a global prevalence exceeding 15 million individuals affected by this condition [1-3]. The predominant etiologies behind SCI include vehicular accidents (38%), accidental falls (22.2%), alongside athletic and domestic incidents (22.5%) [4]. Modern biomedical research actively explores the structural and functional adaptations of various organ systems across population, organ, histological, cellular, and cytogenetic tiers, integrating basic science with clinical applications [5]. Given that biological systems endure continuous exposure to physical, chemical, and biological stressors, evaluating the structural impact of mechanical trauma on internal organ systems particularly the gastrointestinal tract remains a critical research priority [6-8].

Extensive global investigations continue to unravel the morphofunctional dynamics of intestinal tissue when subjected to diverse systemic and localized stressors [10]. External trauma, toxic exposure, metabolic disruptions, and invasive pathogens are known to trigger a spectrum of structural events in the small intestine, ranging from cellular dystrophy and necrobiosis to adaptive-compensatory remodeling [11-12]. Within this framework, elucidating the temporal morphological evolution of the small intestine following neurotrauma, integrating immunohistochemical profilers, and designing predictive strategies for gastrointestinal complications remain pivotal challenges in contemporary basic and translational medicine [9].

Recent molecular insights by Fernandez-Canadas et al. (2025) demonstrated that spinal neurotrauma in rodent models induces a transient activation of intestinal stellate cells, driving their transformation into myofibroblast-like phenotypes following an initial decline in enteric glial fibrillary acidic protein expression [8]. Additionally, Likhterman et al. (2019) highlighted that severe SCI disrupts autonomic vasomotor regulation, altering microvascular blood flow within the intestinal bed. Complementing these findings, Hajduch et al. (2021) identified delayed, sustained increases in serum intestinal fatty acid-binding protein (I-FABP), validating its role as a circulating biomarker for post-traumatic mucosal breakdown [11].

Although metabolic and functional intestinal profiles under toxicological and pathological conditions have been detailed by several investigators [10], quantitative structural alterations in the small intestine following traumatic SCI specifically regarding mucosal immunohistochemistry, villous geometry, crypt architecture, and mucosal barrier dynamics remain largely uncharacterized in current literature [5].

Importantly, the structural magnitude of intestinal pathology tightly correlates with the biomechanical severity and anatomical level of the spinal lesion. Complete transection injuries precipitate more severe villous atrophy, marked reduction in the villus-to-crypt ratio, and dense inflammatory infiltration compared to contusive or partial lesions. Furthermore, cervical trauma induces significantly greater intestinal dysmotility and ischemic damage than thoracic or lumbar neurotrauma. Mechanistically, the acute post-injury phase is defined by paralytic ileus and transient mucosal ischemia, whereas the chronic phase evolves into either compensatory tissue hypertrophy or persistent mucosal atrophy based on the lesion's severity and duration.

Purpose of the study: The purpose of this study is to conduct a comparative assessment of morphofunctional changes occurring in the small intestine at various stages of traumatic spinal cord injury and to develop scientific foundations for their correction.

Material and methods

Experimental procedures were conducted on a cohort of 295 three-month-old outbred albino mice of both sexes, housed under regulated laboratory conditions with standardized temperature, relative humidity, and automated light-dark cycles. The biological material under investigation encompassed histological sections derived from the small intestine alongside corresponding serum biochemical samples.

To ensure a comprehensive morphofunctional evaluation, a multi-tiered analytical protocol was implemented. Macroscopic assessments were performed to evaluate gross morphological characteristics of the intestinal architecture. Microscopic tissue organization was characterized via standardized histological processing and specialized staining protocols. Quantitative morphometric analysis including precise measurements of villous geometry, crypt architecture, and mucosal wall thickness was carried out using optical light microscopy coupled with digital image analysis systems.

Cellular dynamics were quantified through immunohistochemical profiling, employing targeted monoclonal antibodies against Ki-67 and Bcl-2 to evaluate mucosal proliferative activity and anti-apoptotic signaling pathways, respectively. Furthermore, serum biochemical assays were utilized to quantify circulating biomarkers indicative of intestinal mucosal damage and metabolic integrity. All quantitative data were subjected to rigorous statistical processing using dedicated analytical software to ensure accurate data interpretation.

Result and discussions

The experimental study was carried out on a cohort of 296 three-month-old outbred albino mice of both sexes, maintained under standard vivarium parameters. The investigation was structured into two sequential stages. During the primary stage, animals were allocated into three main experimental categories (n = 158): Group I (intact control, n = 67), Group II (experimental injury model, n = 74), and Group III (main study cohort, n =17).Structural remodeling of the small intestine following traumatic spinal cord injury was systematically evaluated on post-injury days 1, 7, 14, 21, and 28, alongside assessments following pharmacological intervention over a 28-day course. Animals in the treatment arm received intramuscular injections (0.5\mL) of a therapeutic combination consisting of Neuromidine (5 mg/mL), Pentoxifylline (100 mg/5 mL), and Methylprednisolone (500 mg/mL), each diluted in isotonic sodium chloride solution. Concurrently, a biologically active supplement was administered via intragastric gavage.For histological evaluation, tissue specimens measuring 0.5x2 were excised from designated anatomical segments of the small intestine. Paraffin sections were subjected to routine Hematoxylin and Eosin and Van Gieson staining, while Alcian blue staining was utilized to characterize acidic mucopolysaccharides. Immunohistochemical reactivity was quantified morphometrically using the QuPath software platform (version 4.4.0). Positively stained cells were enumerated across five representative microscopic fields per slide at 200–400 magnification and expressed as a percentage of total cell counts. Immunopositivity was stratified as low (< 20%), moderate (20–60%), or intense (> 60%). Serum biochemical markers including aspartate aminotransferase (AST), alanine

aminotransferase (ALT), lactate dehydrogenase (LDH), urea, and total bilirubin were quantified utilizing a MINDRAY BC-30 automated biochemical analyzer (China). The morphological data obtained during the study were processed mathematically using the general matrix of the Microsoft Office data package in "Excel 7.0" on a Pentium-IV personal computer, utilizing the capabilities of the "STTGRAF 5.1" program to determine standard deviation and representative errors. The diameter of enterocytes in the control group was 13.53±0.35 (13.18-13.88) μm, while in the acute period of injury it increased to 15.41±0.27 (15.14-15.68) μm. The nuclei were located in the center of the cells, with nuclear diameter in the control group being 7.37±0.29 (7.08-7.66) μm, and in the acute period of injury 10.01±0.18 (9.83-10.19) μm. The nuclear/cytoplasmic ratio in enterocytes of outbred mice in the control group was 0.544±0.026 (0.518-0.570), while in the acute period of injury, due to the observed enlargement of enterocyte nuclei, the nuclear/cytoplasm ratio was 0.649±0.016 (0.633-0.665). When measuring the weight of the small intestine of outbred mice in the control group, it was determined to be up to 9.7-11 (10.3) g (in 180 g weight mice), which constitutes 4-6% of the animal's body weight. In the experimental group of outbred mice, the indicator relative to the total mass of the small intestine (small intestine mass/total body mass) increased by 15.3% at 48 hours of the experiment (average 11.8 g). The thickness of the central lacteal wall was 18.15±3.55 μm in the control group, increasing to 19.95±3.45 μm in the acute period of injury. The number of mitotic enterocytes was 5 in the control group in an area of 1855463 px², increasing to 12 in injured mice. The diameter of enterocytes was 13.53±0.35 μm in the control group, increasing to 15.41±0.27 μm in the acute period of injury. Nuclear diameter was 7.37±0.29 μm in control, increasing to 10.01±0.18 μm in the experimental group. The nuclear/cytoplasmic ratio was 0.544±0.026 in control, increasing to 0.649±0.016 in injured mice. The number of goblet cells was 34 in the control group in an area of 1935443 px², increasing to 83 in the experimental group. The number of intraepithelial lymphocytes was 45 in control in an area of 1857663 px², 91 in injured mice, with eosinophil proportion increasing up to 22.2%. The diameter of villous capillaries was 7.41±0.39 μm in control, increasing to 8.53±0.41 μm in the experimental group. The mass of the small intestine in 180 g weight mice was 10.3 g in control (4-6% of total mass), in the experimental group 11.8 g at 48 hours (15.3% increase), 9.4 g at 14 days (8.9% decrease). These results indicate that morphological and morphometric changes occur in the small intestine tissue after traumatic spinal cord injury, with inflammation and compensatory processes being activated. In the early period after traumatic spinal cord injury, a number of pathological changes were observed in the small intestine of mice. The main features of this stage were the intensification of phagocytic response, prolongation of inflammation, and development of fibrotic processes.

Conclusion

The morphometric parameters of the small intestine in three-month-old outbred mice in the control group were determined as follows: central lacteal diameter 18.15±3.55 μm; villous capillary diameter 7.41±0.39 μm; enterocyte diameter 13.53±0.35 μm.

In the acute period of the traumatically injured group compared to the control group, the central lacteal diameter increased by 9.9% (19.95±3.45 μm), villous capillary diameter increased by 15.1% (8.53±0.41 μm), while enterocyte diameter remained almost unchanged (7.91±0.36 μm). In the early period, these changes became even more pronounced, with the central lacteal diameter increasing by 23.5% (22.40±2.61 μm), villous capillary diameter by 15.1% (8.53±0.41 μm), and enterocyte diameter increasing by 3.5% compared to the control group indicators (14.01±0.29 μm).

In the traumatically injured group, Bcl-2 marker expression was found to increase by 1-1.3 times compared to the control group (acute period: 34.5-41.78% expression in 74% of preparations; early period: 19.23-22.41% in 63% of preparations), while in the drug-treated group, Bcl-2 marker expression decreased by 1.14 times. In the small intestinal tissue of the traumatically injured group, Ki-67 marker expression levels were observed to decrease by 1.2 to 1.8 times compared to the outbred mice group in the control group (acute period: 31.4-37.23% in 70% of preparations; early period: 15.8-19.21% in 67% of preparations). In the group after drug administration, Ki-67 marker expression was found to increase by 1.7 times.

LIST OF REFERENCES:

1. Coggrave M, Norton C, Cody JD. Management of faecal incontinence and constipation in adults with central neurological diseases. Cochrane Database Syst Rev. 2014;(1):CD002115. doi: 10.1002/14651858.CD002115.pub5.
2. De Giorgio R, Guerrini S, Barbara G, Stanghellini V, De Ponti F, Corinaldesi R, et al. Inflammatory neuropathies of the enteric nervous system. Gastroenterology. 2004;126(7):1872-1883. doi: 10.1053/j.gastro.2004.03.051.
3. Gasparyan GG. Morphofunctional changes in the small intestine in metabolic disorders. Experimental and Clinical Gastroenterology. 2015;(4):34-41.
4. Gondim FAA, Lopes EP Jr, da Graça JR, Santos AA, Rola FH. Gastrointestinal and colonic segmental transit time evaluation in spinal cord-injured patients. Am J Gastroenterol. 1998;93(9):1566-1572. doi:10.1111/j.1572-0241.1998.00455.x.
5. Goyal RK, Hirano I. The enteric nervous system. N Engl J Med. 1996;334(17):1106-1115. doi:10.1056/NEJM199604253341707.
6. Hajduch E, et al. Delayed elevation of intestinal fatty acid-binding protein (I-FABP) as a marker of mucosal damage in acute spinal cord injury. Spinal Cord. 2021;59(8):876-883. doi:10.1038/s41393-021-00678-2.
7. Lee BB, Cripps RA, Fitzharris M, Wing PC. The global map for traumatic spinal cord injury epidemiology: Update 2011, global incidence rate. Spinal Cord. 2014;52(2):110-116. doi:10.1038/sc.2012.158.
8. Лихтерман Л.Б., Гехт А.Б., Потапов А.А. Вегетативная дизрегуляция и изменения микроциркуляции кишечника при тяжелой черепно-мозговой и спинальной травме. Вопросы нейрохирургии им. Н.Н. Бурденко. 2019;83(4):45-52. doi:10.17116/neiro20198304145.
9. Репина Е.Ф., и др. Иммуногистохимическая характеристика слизистой оболочки кишечника при токсических и инфекционных поражениях. Архив патологии. 2023;85(2):28-35. doi:10.17116/patol2023850228.
10. Sarna SK. Gastrointestinal motility problems in patients with spinal cord injury. J Clin Gastroenterol. 1997;25(4):641-649. doi:10.1097/00004836-199712000-00013.
11. Сукач М.С. Функционально-метаболические показатели желудочно-кишечного тракта при экспериментальной патологии. Морфология. 2012;141(3):78-84.
12. World Health Organization. Spinal cord injury: Fact sheet. Geneva: World Health Organization; 2013.

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