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MORPHOLOGICAL CHANGES IN THE SMALL INTESTINE OF WHITE RATS WITH TRAUMATIC BRAIN INJURY

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Abstract
In this article, the morphological changes in the lymphoid structures of the small intestinal wall of a white outbred rat after traumatic brain injury were determined as material and materials taken from the intestine were morphologically examined. Changes in the mucous membrane and intestinal barrier TBI leads to dysfunction of the "brain-gut" axis, which contributes to: atrophy of intestinal villi decrease in their height and thickening of the crypts. This reduces the absorption capacity of the intestine. Due to destruction by enterocytes, it leads to increased intestinal permeability, which contributes to dystrophic changes - vacuolization of the cytoplasm, pycnosis of nuclei, damage to microvilli. Edema of the mucous membrane, stagnation of blood in the capillaries, impaired microcirculation and the formation of microthrombi in small vessels due to endothelial dysfunction.

Keywords: Traumatic brain injury, brain, small intestine, lymphoid tissue.

Introduction

Domestic and industrial injuries, including traumatic brain injury (TBI) are diseases that are the main public health problem in all industrialized countries and lead to persistent loss of working capacity, as well as high mortality, disability and lead to high treatment costs [1, 2]. The most common causes of TBI include road accidents and falls. In the structure of mortality, traumatism consistently ranks 3rd. Traumatic brain injury is the most common type of injury and accounts for up to 40% of all types of injuries. According to statistics from the World Health Organization, the level of this pathology increases by 2% annually. According to forecasts of this organization, TBI as one of the main causes of death and disability by 2020 may compete with such socially significant diseases as coronary heart disease and cerebrovascular diseases [3]. There is an increase in more severe forms of injury due to the development of science and technology. In the Russian Federation, TBI rates are 4.5 per 1000 population over 12 months. The mortality rate for this pathology in mild form reaches 5-10%, in severe forms 41-85% [4, 5]. Every year, more than 5 million people die from TBI worldwide [6]. Diagnosis and treatment of this pathology is one of the most important problems in modern medicine, therefore, the development and implementation of new experimental methods and substances that can reduce the degree of brain damage and the mortality rate remains one of the main tasks of modern healthcare. Also, changes in the lymphoid tissue of the intestine in TBI have not been studied.

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The aim of the study was to examine the degree of formation of intestinal lymphoid structures in mongrel rats with TBI on the third and sixth days after traumatic brain injury.

MATERIALS AND METHODS

The experiment was conducted on 48 sexually mature rats, 5-6 months old (m=220-240g). The rodents were kept in compliance with all the rules of laboratory practice for preclinical studies in Uzbekistan. The animals were kept in standard conditions that meet sanitary rules. Acclimatization and selection of animals were carried out for the experiment. Quarantine for at least 10 days. Special marks on the body were used to identify the rodents. During the experiment, the animals were healthy, with no behavioral changes. Closed craniocerebral trauma was inflicted using a freely falling load of varying mass from a hollow pipe 100 cm high, fixed vertically in a tripod. At the end of the pipe there was a striker with a stopper. The stroke of the striker with different variations of force application was 5mm. A load of a fixed mass was placed in the cavity of the tube at a certain height, then released and struck the striker, which then struck the rat's skull. The location of the impact load was determined based on the anatomy of the rat's cerebral cortex. The impact was carried out in the area of localization of the motor and sensory cortex. To avoid fracture of the cranial vault bones, the rat's head was not rigidly fixed. Fixation was carried out on a laboratory table by the hind limbs and additional traction by the upper incisors. The damage zone was located in the frontal-parietal region of the left hemisphere. The posterior edges of the orbit and the crest of the parietal bone served as anatomical landmarks on the skull. A line was constructed between the posterior edges of the orbits and the crest of the parietal bone was determined by palpation. The impact was applied directly inward from these lines, in the area of the parietal bone adjacent to the frontal-parietal suture. In the experiment, the rats were anesthetized by intraperitoneal administration of chloral hydrate (350 mg/kg). The study protocol included the following stages: modeling of TBI; morphological study. Four groups (n=12) of animals were distinguished: 1) intact; 2) TBI with a load lifting height of 0.6 m, weight of 145 g; 3) TBI with a load lifting height of 0.6 m, weight of 155 g; 4) TBI with a load lifting height of 0.6 m, weight of 170 g. The animals of the control group were fixed in the installation, but no injury was inflicted. In the control groups, on the 3rd and 6th days after modeling TBI, in compliance with the principles of humane treatment of animals, some animals were removed from the experiment by euthanasia, under chloroform anesthesia, by puncture of the left ventricle until complete exsanguination. The obtained biomaterial (intestine) was fixed in 10% formalin solution. After fixation, the intestinal tissue was excised and embedded in paraffin using the standard technique. Then, 5-7 μm thick sections were made and stained with hematoxylin and eosin. Micro copying and photography were performed using an optical system consisting of a Leica microscope and a DCM-510 eyepiece camera at magnifications of x100, x200 and x400 magnification, included in the eyepiece camera delivery set. The study of the intestinal tissue structure in intact rats that underwent anesthesia did not reveal any morphological abnormalities.

RESULTS AND DISCUSSION

The following changes were obtained when determining the optimal parameters for reproducing craniocerebral trauma: 1) intact – 0%, 2) weight 145g. – 10%, no deaths were recorded on the first

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day, and one animal died in the following 3 days (10%), 3) weight 155g. – 30%, one animal died intraexperimental, 2 animals died in the following 3 days (30%), 4) weight 170g. – 60%, 3 animals died intraexperimental, and three more animals died in the following 3 days (60%). Microscopic examination of brain sections in dynamics was performed on the 3rd and 7th days after modeling craniocerebral trauma in rats of this group. Study of the structure of the intestinal lymphoid tissue in intact rats that underwent anesthesia did not reveal any morphological abnormalities. Under experimental conditions, on the 3rd day, the following structural changes in the intestinal lymphoid substance are visualized in microphotographs, consisting of the presence of areas of desquamation of the mucous membrane in combination with interstitial edema, hyperplasia of lymphoid follicles (Fig. 1).

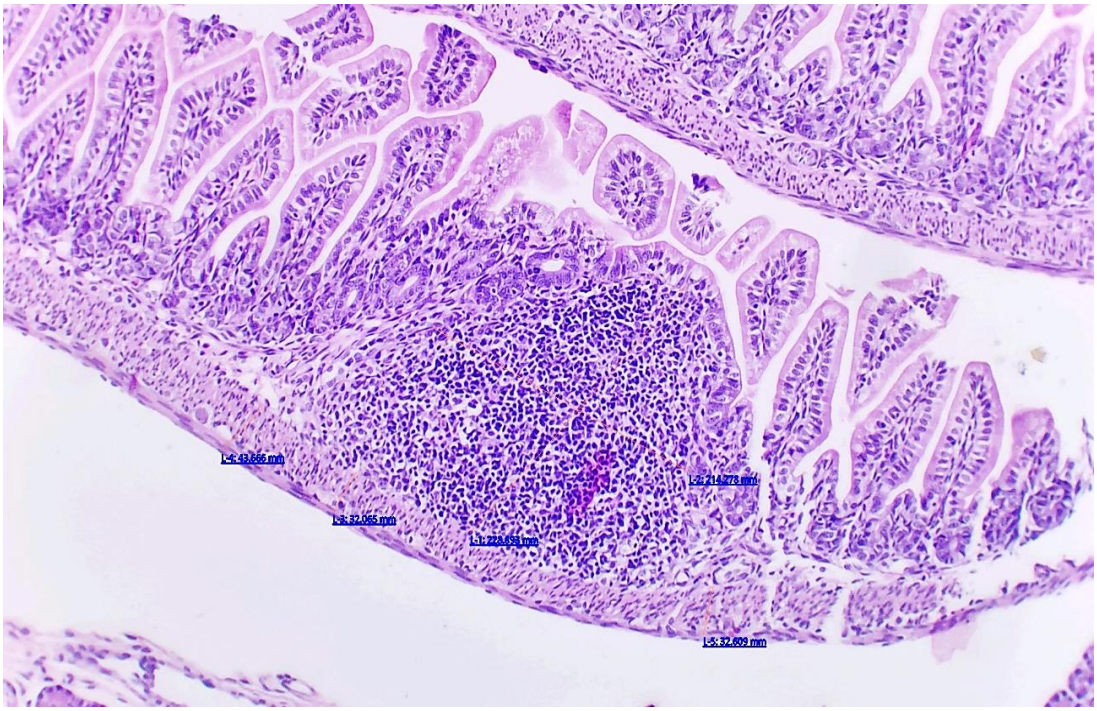

Fig. 1. In the intestinal lymphoid tissue, there are lymphocytes, small, uniform, the interstitial tissue is thickened due to the proliferation of fibrous connective tissue. Stained with hematoxylin and eosin. 10x10.

Under high magnification, the saturation of intestinal tissue with erythrocytes is visualized and a large number of macrophages are determined. Around the blood vessels of the intestinal tissue, perivascular edema is expressed, and in the lymphoid tissue, blood filling and expansion of their lumen were observed **(Fig. 2).** No changes were found. In animals that have undergone experimental TBI, on the contrary, significant morphological disorders are noted in the injury zone. These changes are well visualized and can be subjected to quantitative and qualitative assessment, and their dynamics can also be assessed.

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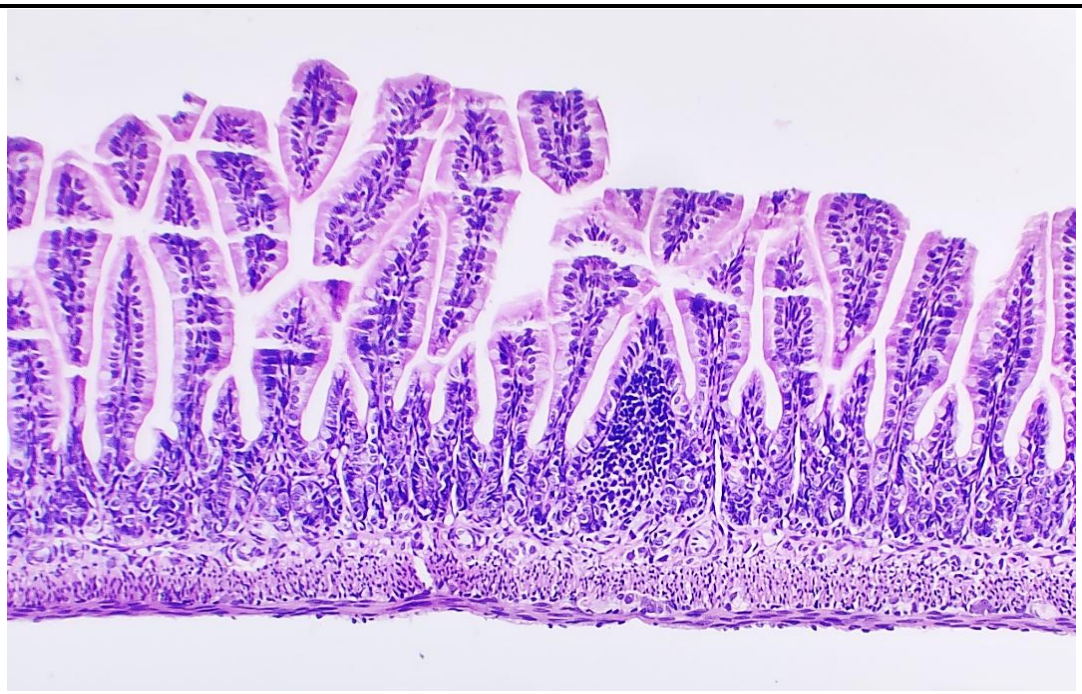

Fig. 2. Minor thickening of the intestinal mucosa and submucosal layer, single lymphoid follicles are also visible. Staining with hemotoxylin and eosin. 10x10.

On the 6th day of the experiment, the mucosa and submucosal base are thickened along the entire intestinal surface, its vessels are full-blooded and dilated (Fig. 3). Pericellular edema is observed around all parenchyma cells. Glandular structures are visualized, mainly in the intestinal lumen. The cell density per unit area is significantly higher than on the 3rd day of the experiment.

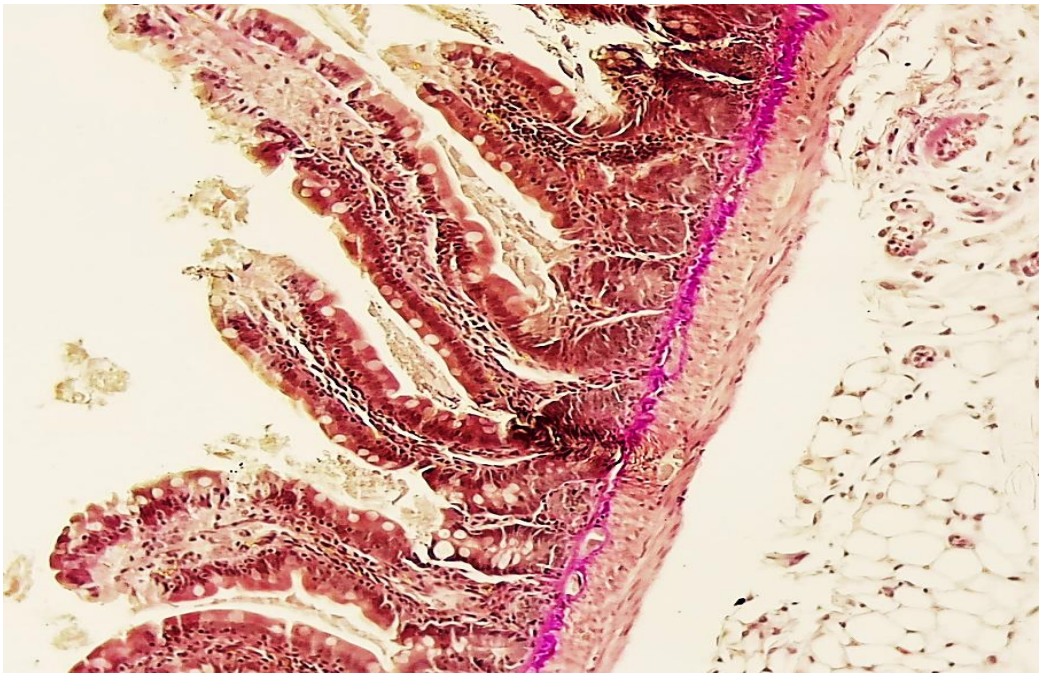

Fig. 3. Desquamation of the epithelium is visible in the intestinal villi, connective tissue is stained as pink stripes, lymphohistiocytic infiltration is also visible. Staining: Van Gieson, 10x10.

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Histological changes in the intestine in rats with traumatic brain injury (TBI) can be considered in the context of barrier function disorders, inflammatory processes, microcirculatory disorders and changes in the nervous regulation of the intestine. Changes in the mucous membrane and intestinal barrier TBI leads to dysfunction of the brain-gut axis, which contributes to: atrophy of the intestinal villi decrease in their height and thickening of the crypts. This reduces the absorption capacity of the intestine. Due to destruction by enterocytes, it leads to increased intestinal permeability, which contributes to dystrophic changes - vacuolization of the cytoplasm, pycnosis of nuclei, damage to microvilli. Edema of the mucous membrane, stagnation of blood in the capillaries, impaired microcirculation and the formation of microthrombi in small vessels due to endothelial dysfunction. Traumatic brain injury causes significant pathological changes in the intestine, which can aggravate the severity of the post-traumatic condition.

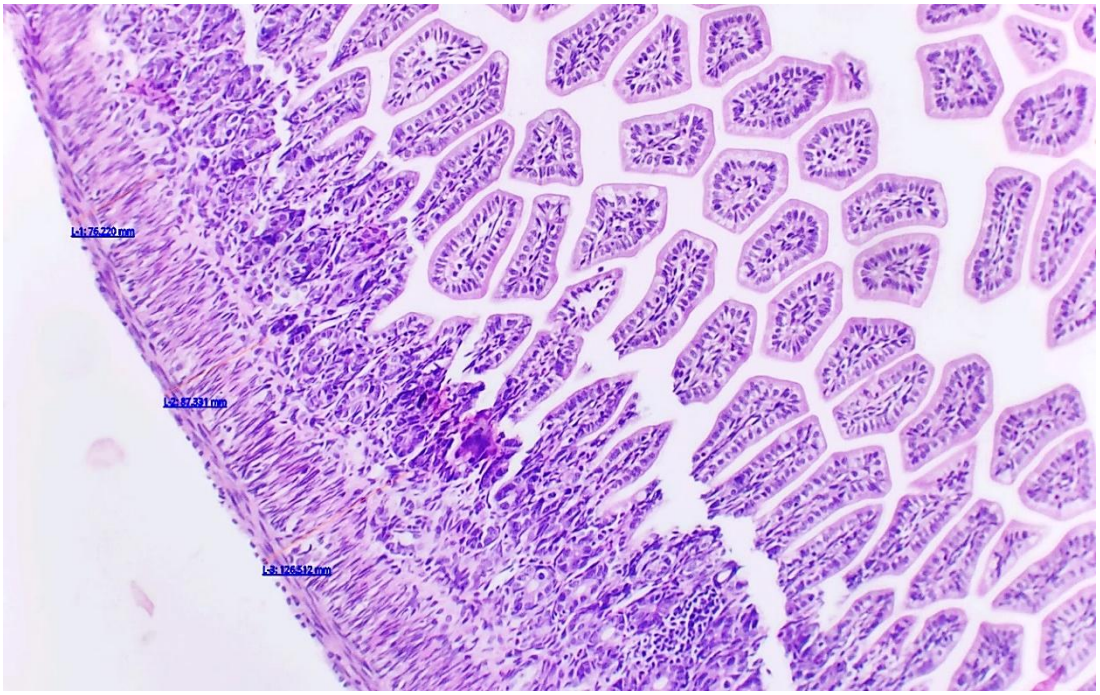

Fig. 4. The intestinal lymphoid tissue is represented by two clearly distinguishable areas - the mucous and submucous base, and connective tissue strands and vessels are also visible. Staining: hematoxylin and eosin. 10x10.

Microscopic examination showed that the colon mucosa is thickened to varying degrees due to the presence of foci of inflammation and damage in mild traumatic brain injury. It was found that the integumentary epithelium migrated in almost all areas, its crypts were deformed due to edema and inflammatory infiltrate, and in some areas the crypts disappeared and were replaced by chronic proliferative inflammatory infiltrate (Fig. 4). When studying the cellular composition of the inflammatory infiltrate, it was found that predominantly proliferating and activated lymphoid and histiocytic cells were located diffusely and densely. Small and medium lymphocytes and plasma cells are scattered among the lymphoid cells. When studying the cellular composition of the inflammatory infiltrate, it was found that predominantly proliferating and activated lymphoid and histiocytic cells are located diffusely and densely. Small and medium lymphocytes and plasma cells are scattered among the lymphoid cells (Fig. 4). It was found that the connective tissue of the

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submucosal layer is relatively thin, and the superficial part is occupied by a proliferative infiltrate. It was found that the blood vessels of the submucosal layer have expanded and hemorrhages have appeared.

CONCLUSION

The parameters of this model (weight of the load is 155 grams, height of the fall is 0.6 meters, the area of impact is frontal-parietal) make it possible to obtain a variety of morphological changes in the lymphoid tissue and in dynamics. The obtained results of structural changes in the intestinal tissues correlate with the data of the behavioral status of laboratory animals (by day 6, structural changes in the intestinal tissues persist against the background of the appearance of signs of reparative processes).

REFERENCES

1. Beloshitsky V.V. Principles of modeling traumatic brain injury in an experiment // Ukrainian neurosurgical journal. 2008. No.4. p. 29.
2. Sokolova T.F., Redkin Yu.V. Method of inflicting dosed closed traumatic brain injury in white rats // Problems of neurosurgery. 1986. No.2. pp. 68-69.
3. Juraeva G.B. Histopathology of intestinal lymphoid tissue in children with AII // Science and education of southern Kazakhstan. Chimkent, 2007 – No.5. pp.105-107.
4. Juraeva G.B., Rasulova O.T. Nadzhapova B. Pathomorphological changes and immunological parameters in acute intestinal infection in newborn children under one year old // IX Russian Conference of Morphologists: Abstracts. M., 2008. pp.43.
5. Juraeva G.B., Allaberdiev B.T. Morphometric indicators of intestinal lymphoid tissue in acute intestinal infections in children // Information letter. Bukhara, 2008 - pp.1-4.
6. Juraeva G.B., Isroilov R.I. Pathomorphological characteristics of intestinal lymphoid tissue in infants with ACI // Information letter - Bukhara, 2008- pp. 1-4.
7. D.P. Terry [et al.] Effect of depression on cognition after mild traumatic brain injury in adults// Clin Neuropsychol. 2019 Jan. Vol. 33(1). pp. 124-136.
8. Sabella S.A., Andrzejewski J.H., Wallgren A. Financial hardship after traumatic brain injury: a brief scale for family caregivers // Brain Inj. 2018. Vol. 32(7). pp. 926-932.
9. Epidemiology of traumatic brain injury in Europe / W. Peeters [et al.] // Acta Neurochir (Wien). 2015. Vol. 157(10). pp. 1683-96.
10. Nozdracheva A.D. Anatomy of a rat (laboratory animals). St. Petersburg: Lan, 2001. P. 15-30.
11. Murray C.J., Lopez A.D. Evidence-based health policy – lessons from the Global Burden of Disease Study // Science. 1996. Vol. 274(5288).

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