тестостерону призводить до ендотеліальної дисфункції, розвитку фіброзу, знижує вироблення оксиду азоту та зрушує про- / антиоксидантний баланс на користь прооксидантів без збільшення інтенсивності перекисного окислення ліпідів. Центральна депривація синтезу тестостерону призводить до фіброзу з подальшим порушенням структурної організації звивистих сім'яних канальців, порушень гемодинаміки, ендотеліальної дисфункції, збільшення щільності судинної стінки кровоносних судин і системному застою. Зниження продукції NO з конститутивних ізоформ NO-синтази відіграє основну роль у розвитку структурних змін інтерстиціальної тканини сім'яників на 270-й день експерименту.

Ключові слова: сім'яники, інтерстиціальні ендокріноцити, сустентоцити, NO-синтаза, iNOS, cNOS, L-аргінін, супероксиддисмутаза, щури.

Стаття надійшла 22.09.2019 р.

депривация синтеза тестостерона приводит к эндотелиальной дисфункции, развитию фиброза, снижает выработку оксида азота и сдвигает про- / антиоксидантный баланс в пользу прооксидантов без увеличения интенсивности перекисного окисления липидов. Центральная депривация синтеза тестостерона приводит к фиброзу с последующим нарушением структурной организации извитых семенных канальцев, нарушениям гемодинамики, эндотелиальной дисфункции, увеличению плотности сосудистой стенки кровеносных сосудов и системному застою. Снижение продукции NO из конститутивных изоформ NO-синтазы играет основную роль в развитии структурных изменений интерстициальной ткани семенников на 270-й день эксперимента.

Ключевые слова: семенники, интерстициальные эндокриноциты, сустентоциты, NO-синтаза, iNOS, cNOS, L-аргинин, супероксиддисмутаза, крысы.

Рецензент Костенко В.О.

DOI 10.26724/2079-8334-2020-3-73-215-219
UDC 616.342.018.25-091:616.5-001.17.:616.379-08.64:57.084.

I.O. Tymoshenko, E.V. Cherkasov, K.V. Shepitko[1]
Bogomolets National Medical University, Kyiv
[1]Ukrainian Medical Stomatological Academy, Poltava

MORPHOMETRIC ASSESSMENT OF STRUCTURAL CHANGES IN THE DUODENAL WALL OF RATS CAUSED BY SKIN BURN INJURY UNDER CONDITIONS OF EXPERIMENTAL DIABETES

e-mail: iryna.tymoshenko@i.ua

This work is devoted to the morphometric assesment of structural changes in the duodenal wall of rats with skin burn injury under conditions of experimental diabetes mellitus. The control group included 21 intact animals without any signs of somatic pathology, experimental group I consisted of 21 rats with experimental skin burn injury, experimental group II consisted of 21 rats without skin burns but with experimentally simulated diabetes mellitus, and experimental group III consisted of 21 rats with both skin burn injury and experimentally simulated diabetes mellitus. The following morphometric parameters have been studied: mucosal thickness, villi height and thickness, crypt depth and width, thickness of lamina muscularis of mucosa, submucosa thickness, muscular layer thickness, serosa thickness, height of the epitheliocytes in the middle part of the villi, mitotic index of columnar epitheliocytes. The obtained data confirm the progredient course of changes characteristic of diabetic enteropathy, which gradually worsen after 7, 14 and 21 days of the experiment.

Key words: skin burn injury, streptozotocin-induced diabetes mellitus, duodenal wall, morphometric assessment.

The work is a fragment of the research project "Morphological features and changes of the digestive system organs in experimental skin burn injury", state registration No. 0119U101618.

Burn injuries and related complications are becoming more common in the current conditions of widespread use of thermal energy in production and everyday life [10, 11]. Pathogenesis of diabetes mellitus and related pathology of the digestive system are also a topical issue for present day medicine [7]. It should be noted that in the global breakdown of general injuries [12, 15] skin burn injuries accompanied by changes in the internal organs prevail [5, 1], and are the subject of current experimental studies [2, 3, 4, 6, 9]. In general, severe burns cause burn disease, with diabetic enteropathy being its component manifesting itself as intestinal dysfunction [6, 7]. However, the morphometric study of the structural features of the duodenal wall in skin burns in terms of its association with diabetes has not been the subject of special studies so far.

The purpose of the study was to perform morphometric assessment of structural changes in the duodenal wall of rats with skin burns in the conditions of experimental streptozotocin-induced diabetes mellitus.

Materials and methods. The study was performed on 84 laboratory white sexually mature male rats weighing 180-210 g. The control group was formed of 21 intact animals without signs of somatic pathology, experimental group I consisted of 21 rats with experimentally simulated skin burn injury, experimental group II - of 21 rats without skin burns with experimentally simulated diabetes mellitus, and experimental group III - of 21 rats with skin burns and experimentally simulated diabetes mellitus. The keeping of rats and all manipulations with them were carried out in full compliance with the recommendations of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986); the provisions of the European Council

Directive 86/609 / EEC (1986); requirements of the Law of Ukraine № 3447 - IV "On protection of animals from cruel treatment"; standards of "General ethical principles of animal experiments", approved by the First National Congress of Ukraine on Bioethics (Kyiv, 2001). The model of experimental diabetes mellitus [13] was simulated by a single intraperitoneal administration of streptozotocin to rats at a dose of 50 mg / kg. Steptozotocin was pre-dissolved in 0.1 M citrate buffer solution (pH-4.5). The reference value for the development of hyperglycemia in rats of experimental group II was blood glucose level of 24.24 ± 0.79 mmol / L (in the control group it was 8.03 ± 0.4 mmol / L). Burns to the skin were inflicted following the generally accepted model of F.C. Regas, H.P. Ehrlich [14], modified by I. Gunas et al. [8]. According to this model two copper plates had been pre-soaked in water at a constant temperature of 100°C for 10 minutes and then, under conditions of ether anesthesia, were tightly pressed to 2 symmetrical pre-shaved dorsal areas of the rat body for 10 seconds at a time. Burn damage to the rats' skin corresponded to II-A or II-B degree of superficial burn and covered 21-23% of the total body surface area, which causes the initiation of signs of burn shock. In a number of studies [2, 3, 4, 6, 9] it was proved that adherence to this model of experimental skin burn resulted in emergence of such characteristic signs of burn disease as endogenous intoxication, multiorgan dysfunction, generalized catabolic reaction, systemic inflammatory and systemic apoptotic response. For histological and morphometric studies a fragment of the duodenum was harvested and its bioptates processed by conventional methods of light microscopy. Paraffin blocks were prepared and their sections stained with H&E. The main criteria for evaluating duodenal wall damage were the results of histological and morphometric data assessment over time, i.e. 7, 14, and 21 days after skin burns. In the above terms, rats of the respective groups were sacrificed by way of a single intraperitoneal administration of a large dose of sodium thiopental and subsequent decapitation.

For integral objective assessment of the course of compensatory, adaptive and destructive processes in the rats' duodenal wall in the setting of the conducted experiment, a morphometric study was performed using a system of visual analysis of histological slides. Images from histological slides were displayed on a computer monitor from a MICROmed SEO SCAN microscope and a Vision CCD Camera. Morphometric studies were performed using VideoTest-5.0, KAAPA Image Base and Microsoft Excel on a personal computer. The study of H&E stained slides was performed within the identified timeline of the experiment. Mucosal thickness, villi height and thickness, crypt depth and width, thickness of lamina muscularis of mucosa, submucosal thickness, muscular layer thickness, serosa thickness, height of the epitheliocytes in the middle part of the villi, mitotic index of columnar epitheliocytes were evaluated.

The values of the arithmetic mean (M), the arithmetic mean error (m) and the standard deviation (Σ) were calculated for all morphometric parameters. Significance of differences between the independent quantitative values was determined by the normal distribution (evaluation of the type of distribution was checked by Pearson's test) by Student's t-test (Bonferonni correction was used when comparing more than two groups), and in other cases by Mann-Whitney U-test. Differences at $p < 0.05$ were considered significant.

Results of the study and their discussion. Histological examination revealed the most significant changes in the rats' duodenal mucosa (RDM) in all groups. RDM villi were in the state of general polymorphism caused by their deformation and discomplexation. Processes of destructive and adaptive restructuring of the villi took place against the background of concomitant edema of loose connective tissue of the lamina propria, and leukocyte and lymphocyte infiltration (fig.1, 2).

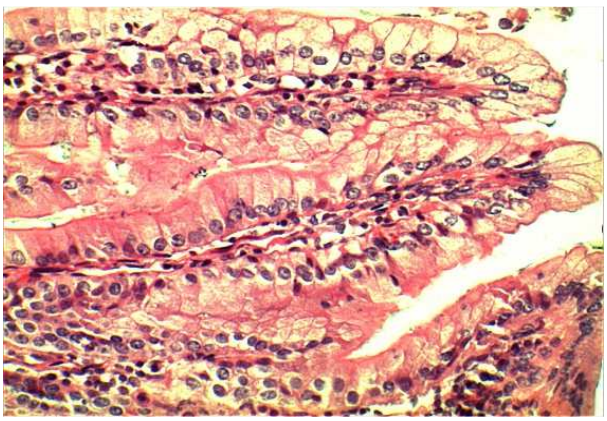

Fig. 1. Edema, leukocyte and lymphocyte infiltration of the duodenal mucosa villi of a rat from experimental group II 14 days after the start of the experiment. Microphotograph. H&E staining. Magnification 20000

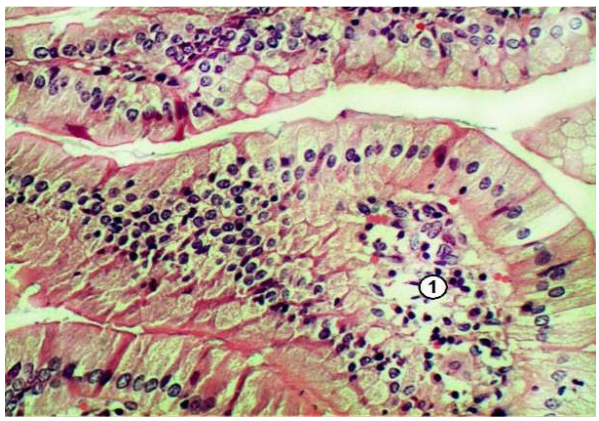

Fig. 2. Edema, leukocyte and lymphocyte infiltration of the duodenal mucosa villi of a rat from experimental group II 21 days after the start of the experiment. 1 - area of necrosis. Microphotograph. H&E staining. Magnification 200

Desquamation of columnar epitheliocytes at the apices of the RDM villi is widespread; number of goblet cells is increased; lumens of intestinal crypts are unevenly expanded; the number of typical acidophilic granules in the swollen cytoplasm of Paneth cells is reduced. Most blood vessels are dilated; there is erythrocyte stasis in the venous vessels and paravasal interstitial edema with loci of diapedetic hemorrhages and leukocyte infiltrates. Central lymphatic vessels of the villi are in the state of lymphocytosis with dilated lumen.

Table 1

Morphometric parameters of the duodenal structural components of animals of the intact group and animals with burns (experimental group I) at different terms of observation after thermal injury

Parameter (M±m)	Intact animals, n≥30	Animals with burn skin injury, n≥30		
		Day 7	Day 14	Day 21
Thickness of the mucous membrane, µm	762.51±12.35	775.63±15.49	783.76±13.46	769.77±18.89
Height of the villi, µm	581.58 ±14.23	593.06±16.86	598.09±14.87	587.51±18.03
Thickness of the villi, µm	69.30±1.97	75.27±1.98*	78.08±1.40***	77.09±2.26*
Crypt depth, µm	171.26±7.40	169.26±3.14	168.36±4.38	169.03±3.52
Crypt width, µm	38.87±1.78	40.11±0.87	40.87±1.03	39.92±1.15
Thickness of the lamina muscularis of mucosa, µm	15.05±0.20	15.12±0.17	15.17±0.26	15.11±0.28
Submucosa thickness, µm	185.45±6.08	201.80±7.80	215.64±8.36**	210.38±10.36*
Muscular layer thickness, µm	82.61±2.57	81.62±1.14	83.26±1.38	83.11±1.34
Serosa thickness, µm	7.36±0.12	7.49±0.15	7.42±0.17	7.39±0.16
Height of epitheliocytes in the middle part of the villi, µm	17.21±0.21	18.11±0.46	18.01±0.22*	17.97±0.23*
Mitotic index of columnar epitheliocytes %	3.367±0.147	2.667±0.111***	2.800±0.111**	2.867±0.171*

Notes: An asterisk indicates values that are significantly different statistically from those of the intact animals group (* – p<0.05; ** – p<0,01; *** – p<0.001).

In 14 days manifestations of cellular discomplexation, deformation and polymorphism of the RDM villi were revealed in group III; as well as areas of necrosis and apoptosis, columnar epitheliocytes which are typical for branched villi, and loss of typicality - for the shortened villi. The epithelial coating of the villi is flattened, the nuclei of epitheliocytes are shifted from the basal pole to the center of the cytoplasm. There are single lymphocytes between the epitheliocytes; shapes and sizes of goblet cells in the RDM villi are numerous and variable. The depth of intestinal crypts is reduced, their lumens are unevenly expanded and have eosinophilic content. Closer to the bottom of the crypts in the epithelial monolayer, poorly differentiated cells at different stages of mitosis were found. The cytoplasm of columnar epitheliocytes is swollen. The connective tissue of RDM lamina propria is swollen with areas of focal diapedetic hemorrhages near hemocapillaries and venules with the phenomena of stasis and sludging of erythrocytes in the vascular lumens.

Table 2

Morphometric parameters of the duodenal structural components of the intact animals group and animals with experimental streptozocin-induced diabetes mellitus (experimental group II) at different observation terms

Parameter (M±m)	Intact animals n≥30	Experimental group II, n≥30		
		Day 7	Day 14	Day 21
Thickness of the mucous membrane, µm	762.51±12.35	776.28±14.58	782.32± 5.02	784.70±16.17
Height of the villi, µm	581.58±14.23	594.66 ±13.58	605.05±19.51	607.57±11.86
Thickness of the villi, µm	69.30±1.97	78.36±1.85**	81.48±2.13***	82.80±1.86***
Crypt depth, µm	171.26±7.40	163.44±4.44	161.18±4.55	159.66±5.80
Crypt width, µm	38.87±1.78	41.94±1.46	43.83±1.52*	44.87±1.38**
Thickness of the lamina muscularis of mucosa, µm	15.05±0.20	15.10±0.42	15.30±0.24	15.36±0.27
Submucosal thickness, µm	185.45±6.08	216.47±9.63**	227.12±10.05***	237.03±10.82***
Muscular layer thickness, µm	82.61±2.57	81.59±2.97	83.65±1.48	83.87±2.98
Serosa thickness, µm	7.36±0.12	7.52±0.31	7.48±0.29	7.41±0.21
Height of epitheliocytes in the middle of the villi µm	17.21±0.21	18.29 ±0.26**	18.34±0.29**	18.39±0.19***
Mitotic index of columnar epitheliocytes %	3.367±0.147	1.967±0.102***	1.833±0.118***	1.700±0.085***

Notes: An asterisk indicates values that are significantly different statistically from those of the intact animals group (* – p<0,05; ** – p<0,01; *** – p<0,001).

In 21 days in group III RDM the phenomenon of deformation of villi was found, as a consequence of discomplexation (violation of the cellular elements ratio). Structural rearrangement with a pronounced polymorphism of villi and the appearance of adjacent villi with sharply different features were found.

Morphometric study confirmed the described qualitative changes in RDM. Quantitative signs of RDM reorganization were noted: statistically significant changes in the villi thickness, crypt width, submucosal thickness, epitheliocyte height in the middle part of villi and mitotic index of columnar epitheliocytes (Tables 1,2,3). These values are statistically significantly different from those of the intact group animals, and in animals of group II the thickness of the duodenal submucosa is 235.28 ± 8.60 μm and is not only statistically significantly different ($p < 0.001$) from animals of the intact group (185.45 ± 6 , 08 μm) and group I ($p < 0,005$). This value is the highest in animals of group III after 21 days (237.03 ± 10.82 μm with the significance of difference from group II $p < 0.005$). The most pronounced is the dynamic pattern of changes in the thickness of the villi and the mitotic index of columnar epitheliocytes. The mitotic index of columnar epitheliocytes of group III rats after 21 days is 1,700 ± 0,085%, which is statistically significantly less ($p < 0,001$) than the identical value of rats from the intact group (3,367 ± 0,147%) and group I (2,867 ± 0,171%).

Table 3

Morphometric parameters of the duodenal structural components of animals of the intact group and animals with burn skin injury and concomitant streptozotocin-induced diabetes mellitus (experimental group III) at different observation terms

Parameter (M±m)	Intact animals n≥30	Animals with thermal skin injury + diabetes, n≥30		
		Day 7	Day 14	Day 21
Thickness of the mucous membrane, μm	762.51±12.35	772.68±17.48	780.17±12.85	782.64±14.12
Height of the villi, μm	581.58 ±14.23	592.01±15.61	600.86 ±14.08	602.54±19.05
Thickness of the villi, μm	69.30 ±1.97	78.13±1.93**	79.63±2.19***	81.08±1.79***
Crypt depth, μm	171.26 ±7.40	165.53±3.37	163.75±5.15	163.02 ±3.02
Crypt width, μm	38.87±1.78	41.76 ±1.29	42.50 ±1.49	44.04 ±1.56*
Thickness of lamina muscularis of mucosa, μm	15.05±0.20	15.11±0.25	15.25±0.40	15.27±0.26
Submucosal thickness, μm	185.45±6.08	211.88±8.41*	224.18±11.79**	235.28±8.60***
Muscular layer thickness, μm	82.61±2.57	80.77±2.20	83.67±2.14	83.72±1.35
Serosa thickness, μm	7.36±0.12	7.47±0.17	7.44±0.24	7.36±0.19
Height of epitheliocytes in the middle part of the villi μm	17.21 ± 0.21	18.22 ± 0.33*	18.35 ± 0.27**	18.37±0.23***
Mitotic index of columnar epitheliocytes %	3.367±0.147	2.400±0.123***	2.133±0.104***	2.067±0.135***

Notes: An asterisk indicates values that are significantly different statistically from those of the intact animals group (* – $p<0,05$; ** – $p<0,01$; *** – $p<0,001$).

According to the scientific literature, diabetes and its complications are quite common. In the review article [11], the authors emphasize new perspectives and the need to study the pathogenetic factors of diabetic enteropathy. The data obtained by us indicate that structural transformations of RDM greatly contribute to the development of diabetic enteropathy with worsening in association with diabetes mellitus. A possible common trigger for changes is a pronounced catabolic response and ER stress in RDM cells [3].

The practical value of our data is the recommendation to include people with diabetes into a separate combustiological risk group (the possibility of enteropathy development, disruption of the intestinal epithelial barrier of the mucous membrane - translocation of the intestinal microbiota and other intraluminal content of toxic and immunogenic action).

Conclusion

The results of the conducted studies have shown the structural changes in the duodenal wall of rats with skin burn injuries to be based on deep structural transformations of its mucous membrane and to worsen in case of association of the burn injury with diabetes. Statistically significant changes of such parameters as villi thickness, crypt width, mitotic index of columnar epitheliocytes and submucosal thickness of the duodenal wall confirm the progredient course of changes characteristic for diabetic enteropathy, which gradually worsens on days 7, 14 and 21 of the experiment.

Prospects for further research in this area are related to the simulation of burn diabetic enteropathy and the study of the effects of drugs that eliminate the catabolic reaction caused by intoxication on the condition of experimental laboratory animals.

References

1. Chaikovsky YuB, Makarova OI, Chereshnyuk IL, Lysenko DA. Acomparative characteristics of cell cycle and dna fragmentation of lung cells in rats after skin burn injury. World of Medicine and Biology. 2014; 2:181-185.
2. Cherkasov VG, Kovalchuk AI, Dzevulskaya IV, Malikov AV, Lakhtadyr TV, Matkivskaya RM. Structural transformations in the internal organs with infusion therapy for burn disease. Medical Science of Ukraine. 2015; 11(3-4):4-11.

3. Cherkasov VG, Tymoshenko IO. Structural changes of duodenal mucosa enterocytes of rats in burn skin injury under experiment streptozotocin-induced diabetes mellitus. Reports of Morphology. 2019; 25(1):55-61. doi: 10.31393/morphology-journal-2019-25(1)-08
4. Dzevulska IV, Kovalchuk OI, Cherkasov EV, Majewskyi OYe, Shevchuk YuG, Pastukhova et al. Influence of lactoproteinum solution with sorbitol on dna content of cells of endocrine glands on the background of skin burn in rats. World of Medicine and Biology. 2018; 64(2):033-039. doi: 10.26724/2079-8334-2018-2-64-33-39
5. Evers LH. The biology of burn injury. Exp. Dermatol. 2010; 19(2),9:777-783. doi: 10. 1111/j.1600-0625.2010.01105.x
6. Galunko GM. Histological changes in the small intestine in the advanced stages of burn diseases. World of Medicine and Biology. 2017; 3(61):90-96. doi: 10.26724/ 2079-8334-2017-3-61-90-96
7. Gotfried J, Priest S, Schey R. Diabetes and the small intestine. Curr Treat Options Gastroenterol. 2017; 15(4):490-507. doi: 10.1007/s11938-017-0155-x
8. Gunas I, Dovgan I, Masur O. Method of intermal burn trauma correction by means of cryoinfluence. Abstracts are presented in zusammen mit der Polish Anatomical Society with the participation of the Association des Anatomistes Verhandlungen der Anatomishen Geselleschaft, Olsztyn Jena-Munchen: Der Urban & Fisher Verlag; 1997. p. 105.
9. Gunas IV, Guminskiya YuI, Ocheretna NP, Lysenko DA, Kovalchuk OI, Dzevulska IV, et al. Indicators cell cycle and DNA fragmentation of spleen cells in early terms after thermal burns of skin at the brackground of introduction 0.9% NaCl solution. World of Medicine and Biology. 2018; 1(63):116-120. doi: 10.26.724/2079-8334-2018-1-63-116-120
10. Haagsma JA, Greatz N, Bolliger I. The global burden of injury: incidence, mortality, disability – adjusted life years and time trends from the Global Burden of Diseases study 2013. Injury Prevention. 2016; 22(1):3-18.
11. Jeschke MG, Pinto R, Kraft R. Inflammation and the Host Response to Injury Collaborative Research Program. Morbidity and survival probability in burn patients in modern burn care. Crit. Care. Med. 2015; 43(2), 4:808-815.
12. Kearney L, Francis E C, Clover AJ. New technologies in global burn care - a review of recent advances. Int. J. Burns Trauma. 2018; 8(4):77-87. PMID: 30245912
13. Natrus LV, Ryzhko IN, Kozak AI, Kryvosheieva OI, Stechenko LA. Ultrastructural base of the connective tissue skin cells interactions at burn injury in the hyperglycemic white rats. World of Medicine and Biology. 2017; 4(62):157-162. doi: 10.26724/2079-83342017-4-62-157-162
14. Regas FC, Ehrlich HP. Elucidating the vascular response to burns with a new rat model. J. Trauma. 1992; 32(5):557-563.
15. Smolle Ch, Cambiaso-Daniel J, Forbes AA. Recent trends in burn epidemiology woredwide: A systemic review. Burns. 2017; 43(2):249-257.

Реферати

МОРФОМЕТРИЧНИЙ АНАЛІЗ СТРУКТУРНИХ ЗМІН СТІНКИ ДВАНАДЦЯТИПАЛОЇ КИШКИ ЩУРІВ ПРИ ОПІКОВІЙ ТРАВМІ ШКІРИ ЗА УМОВ ЕКСПЕРИМЕНТАЛЬНОГО ЦУКРОВОГО ДІАБЕТУ

Тимошенко І.О., Черкасов Е.В., Шепітько К.В.

Робота присвячена морфометричному аналізу структурних змін стінки дванадцятипалої кишки щурів при опіковій травмі шкіри за умов експериментального цукрового діабету. Групою контролю була 21 інтактна тварина без ознак соматичної патології, I експериментальну групу склали 21 щур з експериментальною опікою травмою шкіри, II експериментальну групу склали – 21 щур без опіку шкіри з експериментально відтвореним цукровим діабетом, III експериментальну групу склали – 21 щур з опіковою травмою шкіри та експериментально відтвореним цукровим діабетом. Експериментальний цукровий діабет моделювали шляхом внутрішньочеревного введення щурам стрептозотоцина одноразово в дозі 50 мг / кг. Опікове ушкодження шкіри у щурів відповідало II - А чи II - Б ступеня - поверхневого опіку площею 21-23% загальної поверхні тіла з розвитком опікового шоку.Були досліджено наступні морфометричні показники: товщину слизової оболонки, висоту ворсинок, товщину ворсинок, глибину крипт, ширину крипт, товщину м'язової пластинки слизової оболонки, товщину підслизової основи, товщину м'язової оболонки, товщину серозної оболонки, висоту епітеліоцитів в середній частині ворсинок, мітотичний індекс стовпчастих епітеліоцитів. Одержані дані підтверджують прогредієнтний перебіг характерних для діабетичної ентеропатії змін, поглиблення яких поступово збільшується через 7, 14 та 21 добу експерименту.

Ключові слова: опікова травма шкіри, стрептозотоциніндукований цукровий діабет, стінка дванадцятипалої кишки, морфометричний аналіз.

Стаття надійшла 20.09.2019 р.

МОРФОМЕТРИЧЕСКИЙ АНАЛИЗ СТРУКТУРНЫХ ИЗМЕНЕНИЯХ СТЕНКИ ДВЕНАДЦАТИПЕРСТНОЙ КИШКИ КРЫС ПРИ ОЖОГОВОЙ ТРАВМЕ КОЖИ В УСЛОВИЯХ ЭКСПЕРИМЕНТАЛЬНОГО САХАРНОГО ДИАБЕТА

Тимошенко И.А., Черкасов Е.В., Шепитько К.В.

Работа посвящена морфометрическому анализу структурных изменений стенки двенадцатиперстной кишки крыс при ожоговой травме кожи в условиях экспериментального сахарного диабета. Группой контроля была 21 интактна животная без признаков соматической патологии, I – экспериментальную группу составили 21 крыса с экспериментальной ожоговой травмой кожи, II – экспериментальную группу составили – 21 крыса без ожога кожи с экспериментально воспроизведенным сахарным диабетом, III экспериментальную группу составили – 21 крыса с ожоговой травмой кожи и экспериментально воспроизведенным сахарным диабетом. Экспериментальный сахарный диабет моделировали путем внутрибрюшинно введением крысам стрептозотоцина однократно в дозе 50 мг / кг. Ожоговое повреждение кожи у крыс соответствовало II – А или II – Б степени - поверхностного ожога площадью 21-23% общей поверхности тела с развитием ожогового шока. Были исследованы следующие морфометрические показатели: толщину слизистой оболочки, высоту ворсинок, толщину ворсинок, глубину крипт, ширину крипт, толщину мышечной пластинки слизистой оболочки, толщину подслизистой основы, толщину мышечной оболочки, толщину серозной оболочки, высоту эпителиоцитов в средней части ворсинок, митотический индекс столбчатых эпителиоцитов. Полученные данные подтверждают прогредиентное течение характерных для диабетической энтеропатии изменений, углубление которых постепенно увеличивается через 7, 14 и 21 сутки эксперимента.

Ключевые слова: ожоговая травма кожи, стрептозотоцининдуцированый сахарный диабет, стенка двенадцатиперстной кишки, морфометрический анализ.

Рецензент Єрошенко Г.А.