Реферати

ФАГОЦИТАРНА АКТИВНІСТЬ НЕЙТРОФІЛІВ КРОВІ В МЕХАНІЗМАХ РОЗВИТКУ ЕКСПЕРИМЕНТАЛЬНОГО БАКТЕРІАЛЬНО-ІМУННОГО ПАРОДОНТИТУ

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Однією із важливих ланок в механізмах розвитку, що призводять до ушкодження структурних компонентів пародонтального комплексу та розвитку запального процесу з різним ступенем тяжкості, є фагоцитарна активність лейкоцитів. Мета дослідження полягала в оцінці активності нейтрофілів крові при експериментальному змодельованому бактеріально-імунному пародонтиті. У статті наведені результати досліджень показників спонтанного та активованого тесту з нітросинім тетразолієм на 7-му, 14-ту і 30-ту добу експериментального бактеріально-імунного запалення в пародонті. При цьому наводяться дані щодо характеру змін фагоцитарної активності нейтрофілів у процесі формування і хронізації запального вогнища в пародонтальних тканинах. Також встановлено, що характер перебігу даного змодельованого запального процесу запалення в пародонтальних тканинах залежав від особливостей змін фагоцитарної активності нейтрофілів крові. При цьому запальний процес супроводжувався підвищенням спонтанного тесту з нітросинім тетразолієм нейтрофілів крові та зниженням показників індукованого НСТ-тесту, резерву оксиген-залежного метаболізму, коефіцієнта метаболічної активації нейтрофілів крові щурів із запальним процесом у пародонті та свідчив про виснаження метаболічних резервів даних клітин і порушення процесів фагоцитозу.

Ключові слова: Фагоцитоз, нейтрофіли, імунна система, бактеріально-імунний пародонтит.

Стаття надійшла 12.06.2019 р.

ФАГОЦИТАРНАЯ АКТИВНОСТЬ НЕЙТРОФИЛОВ КРОВИ В МЕХАНИЗМАХ РАЗВИТИЯ ЭКСПЕРИМЕНТАЛЬНОГО БАКТЕРИАЛЬНО-ИММУННОГО ПАРОДОНТИТА

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Одной из важных звеньев в механизмах повреждения структур пародонтального комплекса и формирования воспалительного процесса с различной тяжестью, является фагоцитарная активность лейкоцитов. Цель исследования заключалась в оценке активности нейтрофилов крови при экспериментальном моделированном бактериально-иммунном воспалении в пародонте. В статье показаны результаты исследований полученных показателей спонтанного и активированного теста с нитросиним тетразолием на 7-ые, 14-ые и 30-ые сутки экспериментального пародонтита. При этом приводятся данные о характере изменений активности нейтрофилов в процессе формирования и хронизации воспалительного очага в тканях пародонта. Установлено, что характер течения экспериментального воспаления в тканях пародонтального комплекса зависит от особенностей изменений фагоцитарной активности нейтрофилов. При этом воспалительный процесс сопровождался повышением спонтанного теста с нитросиним тетразолием нейтрофилов крови и снижением показателей индуцированного НСТ-теста, резерва кислород-зависимого метаболизма, коэффициента метаболической активности нейтрофилов крови крыс с воспалительным процессом в пародонте и свидетельствовал об истощении метаболических резервов данных клеток и нарушении процессов фагоцитоза.

Ключевые слова: Фагоцитоз, нейтрофилы, иммунная система, бактериально-иммунный.

Рецензент Єрошенко Г.А.

DOI 10.26724/2079-8334-2020-2-72-156-159
UDC 616-0014:616-001.17]092.5

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MORPHOLOGICAL CHANGES IN RAT HEART MUSCLE IN EXPERIMENTAL PERITONITIS AGAINST THE BACKGROUND OF DIABETES MELLITUS

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The purpose of the work was to study the morphological changes in the heart muscle of rats with modeled acute peritonitis against the background of diabetes mellitus compared to animals with experimental widespread acute peritonitis. Structural changes of cardiomyocytes (focal intracellular myocytolysis and pycnotically altered nuclei), pronounced perivascular edema, focal dystrophic-necrotic changes, polymorphocellular infiltrates, which were located in the stroma, perivascularly and around the necrotically altered cardiomyocytes, were revealed. Structural changes of cardiomyocytes and disturbances in all links of the microhemocirculatory bed, which were manifested in dystonia and paresis of vessels, changes in rheological properties of blood were more pronounced in the conditions of comorbid pathology.

Key words: acute peritonitis, diabetes mellitus, morphological changes of the heart muscle.

The work is a fragment of the research project "Pathogenetic features of the allergic and inflammatory processes course and their pharmacocorrection", state registration No. 0116U004503.

The urgency of timely diagnosis and treatment of widespread acute peritonitis (WAP) against the background of diabetes mellitus (DM) is due to long-term disability, complicated course and high mortality [7, 10]. One of the main provisions in all modern concepts of various diseases' pathogenesis, including WAP, is the cell membrane structure damage. As a consequence, complex morphofunctional changes of the internal organs lead to the development of complications, which are the main causes of lethality in

peritonitis [2, 3, 8, 9]. The inability of the body's defenses to localize infectious factors and to provide adequate excretion of toxins due to the pathogenetic features of diabetes causes the development of multiple organ failure syndrome in these patients [1, 6]. These factors are universal for various critical conditions and affect the course of the disease. Animals with experimental models of WAP and acute inflammation of the peritoneum against the background of DM were used to elucidate structural changes in the heart muscle.

The purpose of the work was to study the morphological changes in the rat heart muscle with simulated WAP against the background of DM compared to animals with experimental WAP.

Materials and methods. The experiment was performed on 48 white rats, which were divided into two groups: the main group - 24 animals with simulated WAP against the background of DM, the comparison group - 24 animals with modeled WAP. All the compared groups of animals were representative by weight, sex and age. Euthanasia of rats was performed under thiopental anesthesia on the 1st, 3rd and 7th days after the injection of feces. As a result of death during the experiment, the number of animals in the groups at the time of euthanasia was accordingly different.

This experimental study was carried out in compliance with the general rules and regulations of the European Convention for the Protection of Vertebrate Animals, which are used for research and other scientific purposes (Strasbourg, 1986), the General Ethical Principles of Experiments on Animals (Kyiv, 2001), and the Law of Ukraine "On the Protection of Animals from Cruel Treatment" (2006).

Experimental DM was induced by means of intraperitoneal administration of streptozotocin produced by "Sigmal" in the fasting state at the dose of 60 mg/kg, which was dissolved in a sodium citrate buffer solution (pH 4.5). Glucose studies were carried out at 9:00 with free access of experimental animals to food and water during the night period. Insulin (0.2 IIU subcutaneously two to five times a week) was administered to rats throughout the observation period.

After 2 weeks upon the use of streptozotocin in the venous blood of rats, which were obtained from the tail vein, glucose content was determined, and in the subsequent studies, only the rats with glucose content of more than 300 mg/L were observed.

14 days after the use of streptozotocin, animals of the main group were administered a 10% solution of filtered stool suspension into the abdominal cavity at the dose of 0.5 ml per 100 g of body weight. Thus, peritonitis was induced by the model proposed by V.A. Lazarenko et al. (2008) [5].

This model of peritonitis by its etiological factors, clinical manifestations and the course phases is close to a similar process in humans. The faecal suspension was obtained by mixing isotonic solution and stool obtained from the feces of 2-3 intact animals, then it was filtered twice through a double layer of gauze. The resulting suspension was injected into healthy rats in a puncture manner no later than 20 min after its preparation. In order to avoid damage to the internal organs when the faecal suspension was introduced into the abdominal cavity, the animals were kept upright, with the caudal end up. Using the method of puncture of the ventral wall in the center of the midline of the abdomen, directing the end of the needle alternately into the right and left hypochondrium, right and left iliac areas, the equal amount of faecal suspension was introduced.

Histological examination of the heart tissues was performed, they were fixed in 10% neutral formalin solution and Lilly fixator, followed by embedding in paraffin. The sections obtained by means of the sledge microtome were stained with hematoxylin and eosin, by Heidingen and by Shabadash. The nature and depth of morphological changes were determined using Olimpus microscope and the histological slide imaging system.

Results of the study and their discussion. In the histological study of myocardial tissue in animals with simulated WAP, ob the 1st day of the experiment, we found a slightly pronounced blood filling of arterial vessels in the epicardium and a slight expansion and vascular venous type congestion. In addition, there was indolent perivascular edema and single diapedetic hemorrhages. In some fields of view polymorphocellular elements were observed. The stroma was sharply loosened by edema, which also extended to the intercellular spaces (fig. 1). Some stromal vessels were somewhat dilated, irregularly filled with blood. In most fields of view, cardiomyocytes were well visualized. However, in several specimens, single cells with optically empty cytoplasm were encountered in the myocardial thickness.

In animals with WAP, cell infiltrates were observed both perivascularly and in myocardial stroma (fig. 2). In addition, hemorrhages localized within the thickness of the myocardium could be detected in individual specimens. Certain areas with necrotic altered cardiomyocytes were surrounded by polymorphocellular infiltrates.

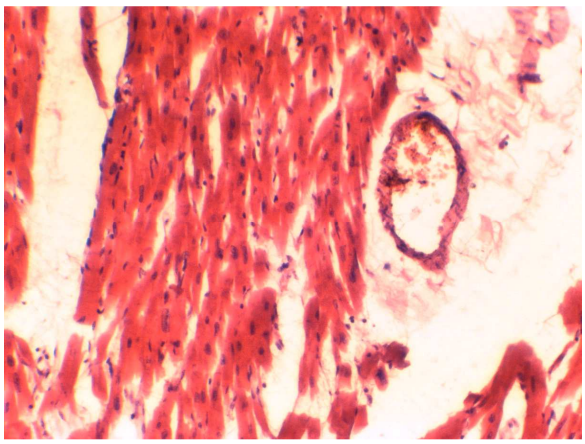

Fig. 1. - Histological structure of the myocardium in an animal with WAP on the 1st day of the experiment. Staining with hematoxylin and eosin. × 200

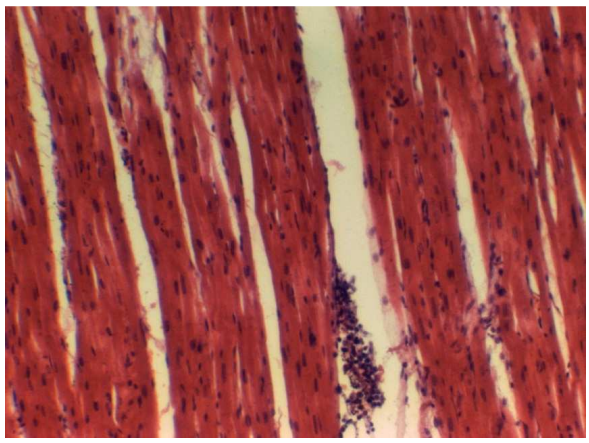

Fig. 2. - Histological structure of the myocardium in an animal with WAP against the background of DM on the 1st day of the experiment. Staining with hematoxylin and eosin. × 200

In the comparison group on the 3rd day of observation pronounced blood filling of blood vessels of the arterial type in the epicardium and expansion and full blood vessels of the venous type were revealed. In addition, moderate perivascular edema was observed, with diapedetic hemorrhages and polymorphocellular elements in some places. In the animals of the main group, there was pronounced edema of the perivasal stroma, which contained single erythrocytes and polymorphic cells (fig. 3). Cell infiltrates were visualized both in the perivascular areas and in the stroma of the myocardium, whose vessels were slightly enlarged and irregularly filled with blood. In some animals, hemorrhage in the thickness of the myocardium and single cells with focal intracellular myocytolysis and pycnotically altered nuclei were found. In the endocardium, there were single aggregations of lympho- and histiocytes.

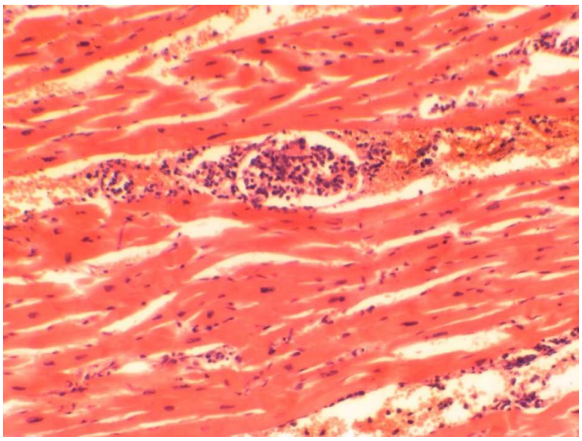

Fig. 3. - Histological structure of the myocardium of an animal with WAP against the background of DM on the 3rd day of the experiment. Staining with hematoxylin and eosin. × 200

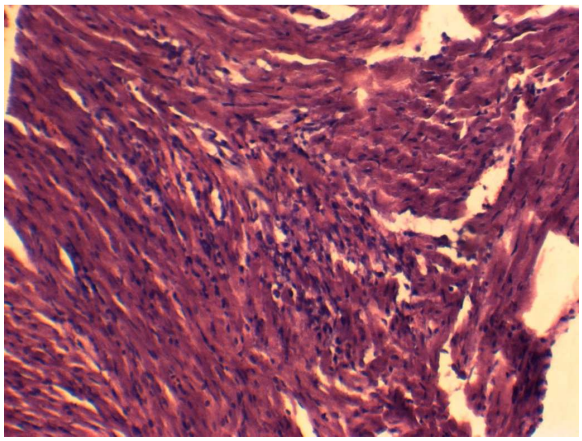

Fig. 4. - Histological structure of the myocardium in an animal with WAP against the background of DM on the 7th day of the experiment. Staining with hematoxylin and eosin. × 180

On the 7th day of experimental WAP modeling, the myocardial stroma of the animals in the comparison group was moderate, loosened, containing single erythrocytes and polymorphocellular elements. Stromal vessels were irregularly blood-filled. Cardiomyocytes were well visualized in the vast majority of visual fields.

Accordingly, the pathological changes that occurred at all levels of the microhemocirculatory bed, in animals with WAP against the background of diabetes mellitus, were manifested by dystonia and angioparesis. Changes in the rheological properties of blood, namely blood stasis, formation of cellular aggregates in the lumen of vessels were observed. Plasma separation was seen in individual vessels. Dystonic manifestations and overflow of vessels with blood led to active transudation of liquid blood into perivasal spaces. The perivasal stroma was loosened by edema extending to the intercellular layers, disintegrating the muscle layers.

In animals of the main group, in the thickness of the myocardium, it was found that individual cardiomyocytes had fuzzy contours, areas of compact placement of cells alternated with the foci of their loosening. Longitudinal sections showed wide fields of disordered placement, tortuous path, and myocyte fragmentation (fig. 4). The cross-striation was fuzzy, unevenly pronounced, cytoplasm was heterogeneous, irregular in color. Somewhere between cardiomyocytes small cell infiltrates were noted. In some specimens, a small number of cells with cleared cytoplasm were identified and changes in the structure of

nuclei were observed, indicating that the presence of focal dystrophic-necrotic changes. Small areas with necrotic altered cardiomyocytes were surrounded by polymorphocellular infiltrates.

Our data are consistent with the results of I.Ya. Hushul [4], who revealed congestion of veins, venules and irregularly pronounced edema of interstitium in predominantly subendocardial compartments for 24 and 48 hours from the time of WAP modeling. The onset of pronounced interstitial edema, which we found early in the WAP modeling period against the background of DM, and the rapid increase of the dyscirculatory phenomena in the hemomicrocirculatory bed of the heart, combined with hypertransudation throughout the experiment, indicate that DM is significantly deepens and accelerates the processes of organ dysfunction.

Conclusion

The onset of pronounced interstitial edema at the early stages of widespread acute peritonitis modeling against the background of diabetes mellitus and a rapid increase in structural changes of cardiomyocytes, dyscirculatory phenomena in the haemomicrocirculatory bed of the heart in combination with hypertransudation throughout the experiment indicate a rapid generalization of the process caused by the comorbid pathology which deepens and accelerates the organ dysfunction processes.

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Реферати

МОРФОЛОГІЧНІ ЗМІНИ В СЕРЦЕВОМУ М’ЯЗІ ЩУРІВ ПРИ ЕКСПЕРИМЕНТАЛЬНОМУ ПЕРИТОНІТІ НА ТЛІ ЦУКРОВОГО ДІАБЕТУ

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Метою дослідження було вивчення морфологічних змін у серцевому м’язі щурів при змодельованому гострому поширеному перитоніті на тлі цукрового діабету порівняно із тваринами з експериментальним гострим поширеним перитонітом. Виявлено структурні зміни кардіоміоцитів (осередковий внутрішньо-клітинний міоцитоліз та пікнотично змінені ядра), виражений периваскулярний набряк, вогнищеві дистрофічно-некротичні зміни, поліморфноклітинні інфільтрати, які розташовувалися у стромі, периваскулярно та навколо некротично змінених кардіоміоцитів. Структурні зміни кардіоміоцитів та порушення у всіх ланках мікрогемоциркуляторного русла, які відображались у дистонії і парезі судин, зміні реологічних властивостей крові були більш вираженими в умовах коморбідної патології.

Ключові слова: гострий поширений перитоніт, цукровий діабет, морфологічні зміни серцевого м’язу.

Стаття надійшла 26.06.2019 р.

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В СЕРДЕЧНОЙ МЫШЦЕ КРЫС ПРИ ЭКСПЕРИМЕНТАЛЬНОМ ПЕРИТОНИТЕ НА ФОНЕ САХАРНОГО ДИАБЕТА

Дзюбановский И.Я., Вервега Б.М., Подручная С.Р., Мельник Н.А.

Целью исследования было изучение морфологических изменений в сердечной мышце крыс при смоделированном остром распространенном перитоните на фоне сахарного диабета по сравнению с животными с экспериментальным острым распространенным перитонитом. Выявлены структурные изменения кардиомиоцитов (внутриклеточный миоцитолиз и пикнотически измененные ядра), выраженный периваскулярный отек, очаговые дистрофически-некротические изменения, полиморфноклеточные инфильтраты, которые располагались в строме, периваскулярно и вокруг некротические измененных кардиомиоцитов. Структурные изменения кардиомиоцитов и нарушения во всех звеньях микрогемоциркуляторного русла, которые отображались в дистонии и парезе сосудов, изменении реологических свойств крови были более выраженными в условиях коморбидной патологи.

Ключевые слова: острый распространенный перитонит, сахарный диабет, морфологические изменения сердечной мышцы.

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