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Morphological and Morphometric Characteristics of the Pancreas in the Dynamics of the Development of the Experimental Model of Pancreatic Necrosis Complicated by Sepsis

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ABSTRACT

Background. The modern principles of treating pancreatic necrosis are ineffective or not effective at all. This dead-end moment, as our analysis of the scientific literature has shown, contributes to the preservation of the so-called "expectant" tactics in the formation of sterile pancreatic necrosis. Only the formation of parapancreatic purulent complications gives rise to the use of various modern surgical technologies, pushing conservative methods of treating pancreatic necrosis into the background. In this regard, we believe that the search for criteria for predicting the course of pancreatic necrosis, and the development of its complication in the form of sepsis, based on the pathogenetic mechanisms of its formation, can increase the effectiveness and efficiency of the complex treatment of patients with this pathology.

Material and methods. To achieve the desired goal, the total array of experimental studies was divided by us into 5 series of experiments: 1-series of experiments - control (intact animals), a 2-series of experiments - animals with an experimental model of acute pancreatitis, a 3-series of experiments - animals with an experimental model of acute sterile pancreatic necrosis, 4-series of experiments - animals with an experimental model of acute infected pancreatic necrosis, 5-series of experiments - animals with an experimental model of acute infected pancreatic necrosis complicated by sepsis. Morpho-

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logical studies included histological and morphometric studies. Pancreatic tissue served as material for research. Morphometric studies were carried out using a standard grid measuring the diameter of the vessels of the microvasculature of the pancreas: arterioles, venules, precapillary arterioles, postcapillary venule, and capillary.

Conclusion. The task aimed at improving the results of treatment of patients with acute pancreatic necrosis complicated by sepsis is possible by studying the factors leading to the progression of the necrobiotic process. Experimental substantiation is required for the subsequent development of effective ways to predict the course of this disease.

Keywords: pancreatic necrosis, pancreatic sepsis, endothelial dysfunction, the morphology of pancreatic necrosis, vascular morphometry

INTRODUCTION

The first case of pancreatic necrosis was described in 1641, when N. Tulpius, having made an autopsy of the corpse of a woman who died of an acute disease of the abdominal organs, discovered an abscess of the pancreas. However, despite such a long period of fame of pancreatic necrosis, this problem today has not lost its relevance. [1,2,3,4]

Until the middle of the last century, pancreatic necrosis remained in the "shadow" of statistical information regarding acute surgical diseases of the abdominal organs. However, since the discovery and introduction into the clinical practice of ultrasound research methods, this disease has entered the leading series among the pathologies of the digestive system. [5,6,7,8]

The problem of treating patients with pancreatic necrosis remains a difficult task in the clinic. At the heart of the problems of treating pancreatic necrosis is the cornerstone for the choice of strategy, tactics, a surgical technologies. At the same time, the necrobiotic process in the pancreas develops very quickly, passing into generalization in a matter of days. The pathological process usually develops not only in the organ itself. Necrosis is usually diffuse, as in the fiber around the organ. [9,10,11,12]

Extensive lesions of the retroperitoneal tissue, omentum bag, large omentum and other structural formations of the abdominal cavity expand the zone of the source of infection and thereby differ in the progressive generalization of the purulent-inflammatory process. That is why, despite the use of extensive, at first glance radical surgical interventions, there is no improvement in the results of treatment. [13,14,15,16]

It is safe to say that today the phase approach is a universally recognized tactic for treating pancreatic necrosis. Based on this, it is still not clear which of the possible three options should be in the priority of treatment tactics. Among them, the predominant conservative treatment, surgical treatment and active-expectant tactics have found their approval. [17,18,19,20]

The development of pancreatic necrosis, complicated by sepsis, even with a sterile necrotic process, often prompts the performance of the surgical intervention. [21,22,23,24]

Unfortunately, the modern principles of conservative therapy are far from being such. Most of them, as the authors themselves indicate, are effective in edematous forms of pancreatitis, and even in small-focal forms of sterile pancreatic necrosis. At the same time, under conditions of emerging total pancreatic necrosis - they are ineffective or not effective at all. This deadlock, as our analysis of the scientific literature has shown, contributes to the preservation of the so-called "expectant" tactics in the formation of sterile pancreatic necrosis. Only the formation of parapancreatic purulent complications gives a start to the use of various modern surgical technologies, pushing conservative methods of treating pancreatic necrosis to the background. [25,26,27,28]

In this regard, we believe that the search for criteria for predicting the course of pancreatic necrosis, and the development of its complication in the form of sepsis, based on the pathogenetic mechanisms of its formation, can increase the effectiveness and efficiency of the complex treatment of patients with this pathology.

The enumeration of numerous surgical methods for the treatment of pancreatic necrosis does not exhaust the wide range of surgical technologies described in the literature used in this pathology. This indicates that the solution to this problem has not been completed and is waiting to be further developed. [29,30]

A qualitatively new stage in surgery was the creation of minimally invasive surgical technologies designed to level the contradiction between the scope of the operation and the trauma from it. Technological progress has allowed these manipulations to take a worthy place in

surgery, in many cases exceeding the capabilities of "open" surgery. [31]

Along with this, it should be noted that it has been established that the interaction process between the infect and the macroorganism is more complex than previously thought and is characterized by the versatility of the latter's response to microbial invasion. From the standpoint of the present stage of cognition, sepsis is more correctly considered not only as a progressive systemic inflammation but as a life-threatening dysregulation of the response (dysregulation) to infection with acute organ dysfunction that reflects damage to one's own tissues. [32]

MATERIAL AND METHODS

All experimental studies fully complied with the terms of the Council of Europe Convention on the Protection of Animals of 1986.

Experimental studies were conducted on white laboratory rats of the Wistar line located in the vivarium of the central research laboratory of the Bukhara State Medical Institute. In total, 170 animals weighing 150-250 grams of both sexes, without external signs of the disease, were used. Animals were mandatory before the start of experimental studies were in a 10-day quarantine. Before the experimental studies began, all animals ate a standard diet. During the experimental studies, the animals ate exclusively grain food a day before taking blood samples for laboratory tests.

The entire protocol of the planned pilot studies was preliminarily reviewed, discussed, and approved by the Bioethical Committee under the Ministry of Health of the Republic of Uzbekistan.

To achieve the desired goal, the total array of experimental studies was divided into 5 series of experiments. This, on the one hand, was due to the need to process a large digital information array, on the other hand, it was necessary to identify the regularity of the pathological process associated with various forms of pancreatic necrosis. In our studies, the model of pancreatic sepsis was studied in stages and consisted of the following comparative and main series:

1-series of experiments – control. These were intact animals in the amount of 10 pieces, without any interventions and without modelling any pathological conditions.

2-series of experiments - comparative-A. These were animals in the amount of 40 pieces, with an experimental model of acute pancreatitis. The technique of reproducing the model of acute pancreatitis began with the performance of an upper median laparotomy up to 3.0±0.1 cm in length. The stomach and duodenum were brought out into the wound along with the pancreas and by transillumination visualized the Wirsung duct of the pancreas was. Under the control of vision, a double ligature filament was performed number "0" with the help of a curved stabbing surgical needle through the mesentery of the duodenum with the capture of the marginal vessel and the proximal third of the duct of the gland near the intestinal wall. The laparotomic wound was sutured tightly.

3-series of experiments - comparative-B. These were animals in the amount of 40 pieces, with an experimental model of acute sterile pancreatic necrosis. To simulate this pathological process, we applied an improved technique, based also on ligation of the Wirsung duct through laparotomic surgical access. On the 3rd day after the operation, a relaparotomy was performed and 0.5 ml of a 10% solution of calcium chloride was injected through the formed defective zone into different points of the pancreatic parenchyma.

4-series of experiments - comparative-C. These were animals in the amount of 40 pieces with an experimental model of acute infected pancreatic necrosis. To simulate this pathological process, we also used an improved technique based on the repetition of all manipulations of the 3-series of experiments with an additional introduction of 0.5 ml of 20% of the animal autocalls suspension into the pancreatic parenchyma one day after the simulation of acute sterile pancreatic necrosis according to the above-described method.

The 5-series of experiments is the main one. These were animals in the amount of 40 pieces with an experimental model of acute infected pancreatic necrosis, complicated by sepsis. To simulate this pathological process, we have developed an original technique. The method of modelling acute infected pancreatic necrosis, complicated by sepsis in small laboratory animals, was carried out as follows. In the first stage, to change the reactive properties of the macroorganism, rats were administered antilympholin-Cr for two days intraperitoneally at a dose of 0.03 mg per 100 grams of the animal. After another 3 days of experimental modelling of the pathological process, the above-described methods of modelling acute infected pancreatic necrosis were used, that is, ligation of the Wirsung duct, injection into the pancreas of 0.5 ml of a 10% solution of calcium chloride, subsequently (after a day) the introduction of 0.5 ml of a 20% solution of animal autocalls into the pancreas. Subsequently, starting 1 day after the last manipulation,

the development of the entire clinical and laboratory manifestation of acute infected pancreatic necrosis, complicated by sepsis, was observed.

Over the next 7 days, the animals developed a full-fledged clinical picture of pancreatic sepsis with such clinical and laboratory signs as tachycardia, tachypnea, hyperthermia and leukocytosis). Additional hemoculture studies only confirmed the high reproducibility of the model.

Morphological studies included histological and morphometric studies. Pancreatic tissue served as a material for research. The tissue pieces were fixed in 10% neutral formalin for 72 hours. After washing in a flowing stench for 2-4 hours, dehydration was carried out in alcohols of increasing concentration and chloroform and poured into paraffin. Histological sections with a thickness of 5–8 μm were made from paraffin blocks, dewaxinization was carried out in a thermostat and the sections were stained with hematoxylin and eosin. The drugs were studied under a light microscope using a lens 10, 20, and 40, the necessary areas were photographed.

Morphometric studies were carried out using a standard grid measuring the diameter of the vessels of the microvasculature of the pancreas: arteriole, venule, precapillary arteriole, postcapillary venule, and capillary.

Slaughter of animals after the completion of the experiments was carried out in accordance with the regulation of the Ethical Committee of the Ministry of Health of the Republic of Uzbekistan "On the humane treatment of laboratory animals during experimental studies".

Studies were conducted in dynamics on 1,3,7,14 days of modelling of the corresponding pathological process. The data obtained during the study were subjected to statistical processing on a Pentium-IV personal computer using the Microsoft Office Excel-2016 software package, including the use of built-in statistical functions and Bio Stat for Windows (version 2007).

Methods of variational parametric and non-parametric statistics were used to calculate the arithmetic mean of the studied indicator, the mean square deviation, the standard error of the mean, relative values (frequency, %), the statistical significance of the obtained measurements when comparing the average quantitative values were determined by the parametric criterion of Student (t) with the calculation of the probability of error in checking the normality of the distribution (according to the criterion of excess) and the equality of general variances (F - Fisher's criterion).

RESULTS

The dynamics of the morphological picture in the modelling of various variants of acute pancreatitis revealed staged changes in the structural transformations of the pancreas. Characteristic of the changes in the structure of acinar cells. There were also pronounced changes of an inflammatory nature. The above cells were in a damaged state, and in the pancreas itself, there was a pronounced interstitial oedema. In some places, small foci of necrosis were detected. They were noted in the early stages of experimental observations in the organ itself. In the future, the same foci of necrosis were noted in the tissues surrounding the pancreas.

A microscopic picture of acute pancreatitis revealed pancreatic oedema, which was in two versions: interlobular and intralobular. The acinar cells clustered. The formed individual cells of the destroyed acinar cells were dispersed, forming separation due to connective tissue, which acted as a matrix.

With the development of destructive processes, necrobiosis, and the progression of the disease, histologically revealed vast areas of necrosis of cells and stromal structures. In histological sections of the pancreas obtained from the caudal part, microabscesses, and thrombosis in the capillary network were also detected. In the zone of the body and the tail part - foci of haemorrhage of a small and extensive nature.

If the 1-3-day modelling of acute pancreatitis, the prevailing changes were in the form of inflammatory oedema and infiltration (see figure-1), then on the 7-14th day of the reproduction of the pathological process, areas of enzymatic lysis shrouded infiltration, as well as areas of necrotic tissues prone to sequestration, were increasingly detected (see figure-2).

In animals with uninfected pancreatic necrosis on the 14th day of modeling, resorption of hemorrhagic exudate from foci of necrotic lesions was noted. In these animals, in the early stages of modelling the disease, necrosis of pancreatic cells was mixed. It was dominated by components of a fatty and hemorrhagic nature.

In infected pancreatic necrosis, the detected areas of steatonecrosis were combined with focused areas of haemorrhage (see figure-3).

And although in the case of uninfected pancreatic necrosis in surviving animals, such areas were subjected to resorption, nevertheless, in the conditions of modeling of infected pancreatic necrosis, complicated by predomi-

nant sepsis, there was a transformation of them into purulent masses (see figure-4).

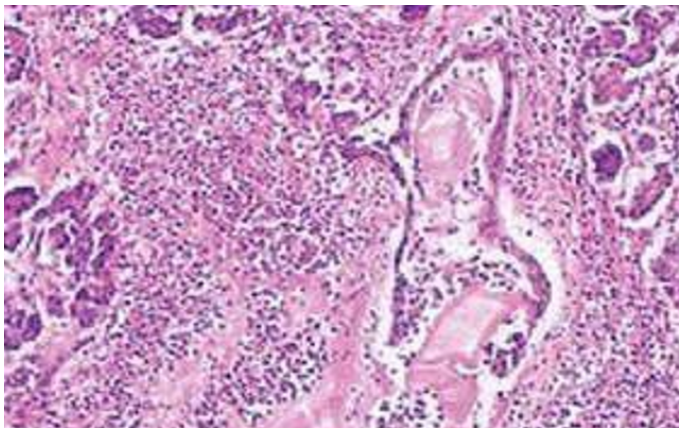

Figure-1. Inflammatory reaction in the pancreas around the dilated duct. Coloration of hematoxylin-eosin. Magnification: 250 times

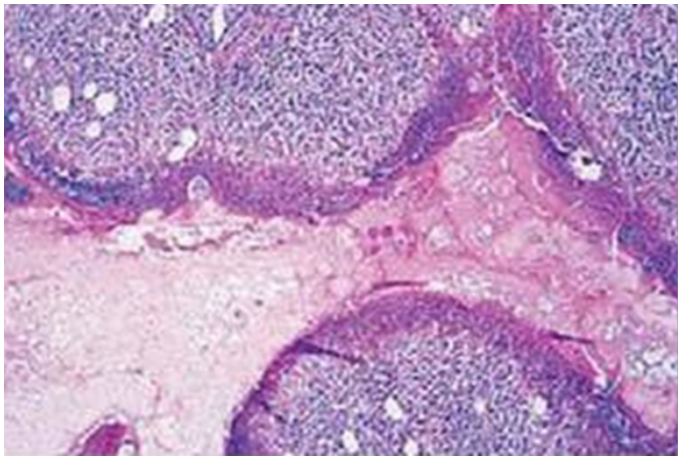

Figure-2. Foci of necrosis along the pancreatic duct. The colouration of hematoxylin-eosin. Magnification: 400 times

Necrosis of the pancreatic parenchyma, caused by the defeat of acinar cells, was colliquative in nature.

Morphometric examination of pancreatic vessels in intact animals and in the dynamics of the development of various forms of acute pancreatitis showed that, in general, the aggravation of the pathological process ambiguously affects the structure of the vessels of the microvasculature of this organ. It is necessary to highlight a close direct correlation between the change in the diameters of venules and postcapillary venules.

And changes in the microcapillary system of the pancreas in the conditions of modelling acute pancreatitis, for the entire period of the study, the revealed changes were not reliable in relation to intact animals. The identified deviations, in the form of vasodilation of the venous series, quickly passed and were more of a compensatory nature.

As the pathological process progressed, as well as in cases of modelling acute uninfected pancreatic necrosis, there were significant shifts in violations of the capillary system and precapillary arterioles. So, if in the case of modelling acute pancreatitis, the diameter of the capillaries in the pancreas increased from 2.48±0.14 μm to 3.20±0.21 μm ($p<0.05$), then in the case of modeling uninfected pancreatic necrosis, the increase in capillary diameter was up to 4.05±0.18 μm ($p<0.05$), that is, 1.46 times.

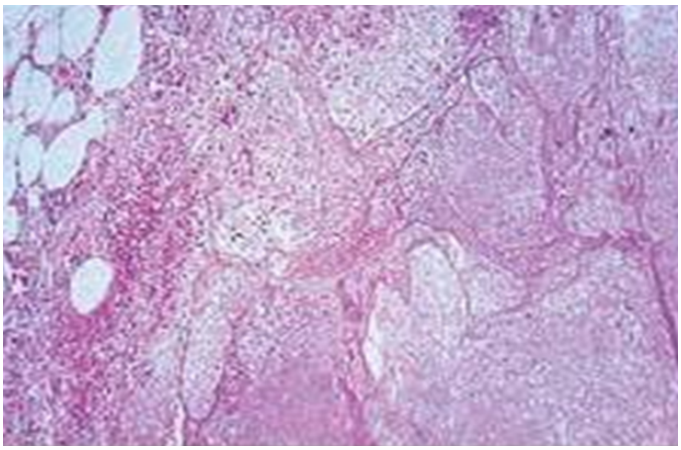

Figure-3. Necrosis in combination with haemorrhage. The coloration of hematoxylin-eosin. Magnification: 250 times

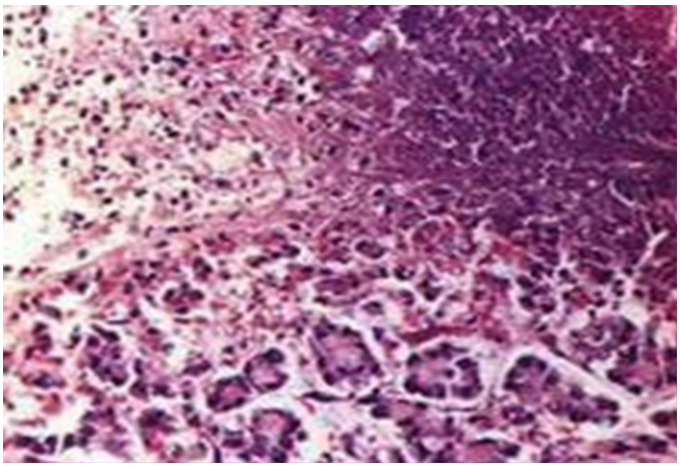

Figure-4. An extensive area of haemorrhage with necrosis impregnated with pus. The coloration of hematoxylin-eosin. Magnification: 250 times

Precapillary arterioles also begin to join the general picture of changes, the diameter of which increased from 14.08±0.12 μm ($p<0.05$) to 16.88±0.16 μm ($p<0.05$). The role of other types of vessels of the microvasculature of the pancreas was not so pronounced, although it tended to increase (see table).

All these prerequisites were confirmed in cases where the infection had already joined. The leaders in the dynamics of change began to act as precapillary arterioles and postcapillary venules.

The increase in the diameter of precapillary arterioles in animals with infected pancreatic necrosis was 1.48 times ($p<0.05$), and postcapillary venules - 1.27 times ($p<0.05$). And in this case, the leading in increase in diameter compared to intact animals were capillaries (1.77 times; $p<0.05$).

When modeling acute infected pancreatic necrosis complicated by sepsis, the structural picture of the microvasculature changes completely, although the prerequisites for such changes have already been traced among animals with acute infected pancreatic necrosis without sepsis.

Leading in the increase in diameter were capillaries (2.82 times; $p<0.05$). The morphological picture of their transformation was characterized by the development of not only stasis and cell aggregation but also with the formation of microthrombi. Further, the process of damage to the vessels of the microvasculature was directed towards the venous system, increasing the volume of both postcapillary venules (2.25 times; $p<0.05$) and the venules themselves (2.54 times; $p<0.05$). It should also be noted the increase in the diameters of the arterial bed vessels, both arterioles (1.45 times; $p<0.05$), and precapillary arterioles (1.72 times; $p<0.05$). This indicates the breakthrough of the angiogenic barrier, the complete destruction of the capillary system, the opening of the corresponding arteriole-venous shunts and the progression of lymphostasis.

Table. The nature of the change in morphometric indicators of the microvasculature of the pancreas in animals with acute pancreatic necrosis complicated by sepsis

DYNAMICS	AVERAGE DIAMETER OF MICROVASCULAR (M±M), μm				
	arteriole	precapillary arteriole	capillary	postcapillary venule	venula
1-day	35.1±5.2*	1.42±0.21*	1.65±0.6*	2.26±0.92*	2.0±0.12*
3-day	35.9±4.1*	1.44±0.11*	1.70±0.52*	2.57±0.55*	2.12±0.25*
7-day	36.2±3.2*	1.45±0.08*	1.74±0.48*	3.07±0.13*	2.40±0.16*
14-day	37.1±1.8*	1.49±0.04*	1.78±0.35*	3.36±0.21*	2.49±0.11*
R**	0.787±0.012	0.819±0.013	0.784±0.011	0.744±0.013	0.884±0.011
R***	0.758±0.015	0.286±0.022	0.958±0.012	0.946±0.016	0.980±0.013
R****	0.966±0.01	0.978±0.012	0.931±0.011	0.900±0.022	0.982±0.016
R*****	0.991±0.007	0.982±0.009	0.968±0.011	0.954±0.024	0.973±0.011

*p<0,05 – reliably in relation to intact animals.
R – the value of the correlation coefficient in relation to:
** - intact animals;
*** - animals with acute pancreatitis;
**** - animals with acute, uninfected pancreatic necrosis;
***** - animals with acute infected pancreatic necrosis.

DISCUSSION

Based on the conducted morphological and morphometric studies, in comparison with the clinical and laboratory data of the course of various forms of acute pancreatitis, the following scheme for the formation of pancreatic sepsis can be assumed.

The etiological factor provokes a trigger effect on the pancreas provoking the first stage of the body's inflammatory reaction - alteration. Local changes occurring in the pancreas are more manifested by the occurrence of foci of necrosis, which are not associated with a violation of the blood circulation of the organ. Foci of necrosis, increasing perifocal inflammation, in turn, provoke the intensity of exudation, which, as our research has shown, begins with the organ itself, and then into the retroperitoneal space and even into the abdominal cavity. [33]

General changes occurring in the body are manifested by the syndrome of a systemic inflammatory reaction, as a result of which changes occur in the microvailculatory system, aimed primarily at increasing thrombosis. This reaction is aimed at suppressing increased exudation from the focus of destruction and thereby starts to limit the process and possible involution of necrotic sites.

There is an aseptic sequestration of necrotic areas of the pancreas because of the enzymatic melting of necrotic tissues.

However, the accession of an endogenous or exogenous infection, especially when using unjustified early surgical interventions on an inflamed pancreas, and under the influence of enzymatic lysis of organ tissues, purulent-putrefactive melting of both the pancreas and the cellular spaces around the organ begins. Under these conditions, the syndrome of a systemic inflammatory reaction, as a clinical manifestation of pancreatic sepsis, takes the form of sepsis syndrome and / or severe sepsis when organ dysfunction is attached. There is an aggravation of the process, where the prevailing step is the sepsis itself.

As our previous studies have shown, the leading role in these transformations is attributed to morphometric changes in the vessels of the microvasculature, which is probably a manifestation of endothelial dysfunction.

All of the above naturally, with the exception of infectious effects, gives rise to the main question - when should surgical intervention be performed? At the same time, the results of the analysis of literary sources are reduced to orientation through the evaluation of X-ray visual data. At the same time, as shown by the results of the analysis of the treatment of patients with a similar traditional approach, often end fatally. [34]

CONCLUSION

The task aimed at improving the results of treatment of patients with acute pancreatic necrosis complicated by sepsis is possible by studying the factors leading to the progression of the necrobiotic process. Experimental substantiation is required for the subsequent development of effective ways to predict the course of this disease.

Information on the ethical aspects of the studies - All experimental studies fully complied with the terms of the Council of Europe Convention on the Protection of Animals of 1986. The entire protocol of the planned experimental studies was preliminarily reviewed, discussed, and approved by the Bioethical Committee under the Ministry of Health of the Republic of Uzbekistan.

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Conflict of interest is not.

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SEPSIS BILAN ASORATLANGAN PANKREONEKROZI EKSPERIMENTAL MODELINING RIVOJLANISH DINAMIKASIDA MORFOLOGIK VA MORFOMETRIK XUSUSIYATLARI

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ABSTRAKT

Dolzarbligi. Pankreonekrozni davolashning zamonaviy printsiplari afsuski samarasiz. Ilmiy adabiyotlarni tahlil qilishimiz ko'rsatganidek, steril pankreonekroz shakllanishida "poylash" deb atalgan taktikaning saqlanib qolishiga hissa qo'shadi. Faqat parapankreatik yiringli asoratlarning paydo bo'lishi turli zamonaviy jarrohlik texnologiyalarini qo'llashga sabab bo'ladi, pankreas nekrozini davolashning konservativ usullarini orqa fonga ketadi.

Material va usullar. Maqsadga erishish uchun eksperimental tadqiqotlar 5 seriya tajribalarga bo'lindi: 1-seriya - nazorat (intakt hayvonlar), 2-seriya - o'tkir pankreatitning eksperimental modeliga ega hayvonlar, 3-seriya - o'tkir steril pankreonekrozining eksperimental modeliga ega hayvonlar, 4-seriya - o'tkir infektsiyali pankreonekrozning eksperimental modeliga ega bo'lgan hayvonlar, 5-seriya - sepsis bilan asoratlangan o'tkir infektsiyali pankreonekrozning eksperimental modeliga ega bo'lgan hayvonlar. Morfologik tadqiqotlar gistologik va morfometrik tadqiqotlarni o'z ichiga olgan. Pankreas to'qimasi tadqiqot uchun material bo'lib xizmat qilgan. Morfometrik tadqiqotlar oshqozon osti bezi tomirlari diametrini o'lchagan standart to'r yordamida amalga oshirildi: arteriollar, venules, prekapilyatsion arteriollar, postkapili venule, kapillyar.

Xulosa. Sepsis bilan asoratlangan o'tkir pankreonekroz bilan og'rigan bemorlarni davolash natijalarini yaxshilashga qaratilgan vazifa nekrobiyotik jarayonning rivojiga olib keluvchi omillarni o'rganish orqali mumkin. Ushbu kasallikning kechishini bashorat qilishning samarali yo'llarini keyinchalik ishlab chiqish uchun eksperimental asos talab etiladi.

Tayanch iboralar: oshqozon osti bezi nekrozi, pankreatogen sepsis, endotelial disfunktsiya, oshqozon osti bezi nekrozining morfologiyasi, qon tomirlar morfometriyasi.

МОРФОЛОГИЧЕСКАЯ И МОРФОМЕТРИЧЕСКАЯ ХАРАКТЕРИСТИКА ПОДЖЕЛУДОЧНОЙ ЖЕЛЕЗЫ В ДИНАМИКЕ РАЗВИТИЯ ЭКСПЕРИМЕНТАЛЬНОЙ МОДЕЛИ ПАНКРЕОНЕКРОЗА, ОСЛОЖНЕННОГО СЕПСИСОМ

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АБСТРАКТ

Актуальность. Современные принципы лечения панкреонекроза мало эффективны или не эффективны вообще. Данный тупиковый момент, как показал наш анализ научной литературы, способствует к сохранению так называемой «выжидательной» тактики при формировании стерильного панкреонекроза. Только формирование парапанкреатических гнойных осложнений дает старт к применению различных современных хирургических технологий отодвигая консервативные методы лечения панкреонекроза на второй план.

Материал и методы. Для достижения искомой цели общий массив экспериментальных исследований был разделен нами на 5 серий опытов: контрольная, животные с экспериментальной моделью острого панкреатита, острого стерильного панкреонекроза, инфицированного панкреонекроза и осложненного сепсисом. Морфологические исследования включали в себя гистологические и морфометрические исследования. В качестве материала для исследований служила ткань поджелудочной железы. Морфометрические исследования проводились при помощи стандартной сетки с измерением диаметра сосудов микроциркуляторного русла поджелудочной железы: артериолы, венулы, прекапиллярной артериолы, посткапиллярной венулы, капилляра.

Заключение. Решение задачи, направленной на улучшение результатов лечения больных с острым панкреонекрозом, осложненным сепсисом, возможно путем исследования факторов, приводящих к прогрессированию некробиотического процесса. Требуется экспериментальное обоснование для последующей разработки эффективных способов прогнозирования течения данного заболевания.

Ключевые слова: панкреонекроз, панкреатогенный сепсис, эндотелиальная дисфункция, морфология панкреонекроза, морфометрия сосудов.