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Comparative Clinical and Laboratory Characteristics of the Course of the Experimental Model of Pancronecrosis Complicated with Sepsis

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Abstract

Relevance. The pathogenesis of acute pancreatitis is not completely understood. Currently, most scientists adhere to the enzymatic theory of the pathogenesis of acute pancreatitis. The study of the pathogenetic aspects of the development of pancreatic necrosis requires a more in-depth analysis of the ongoing changes in the body, which can be carried out first in the experiment, and only then tested in the clinical setting.

Methods. Experimental studies were carried out on white laboratory rats of the Wistar line. A total of 170 animals weighing 150-250 grams of both sexes, without external signs of disease were used. Studies were conducted on animals with experimental models of acute pancreatitis, sterile pancreatic necrosis, infected pancreatic necrosis and pancreatic necrosis complicated by sepsis. The activity of α-amylase and lipase enzymes in blood serum was studied.

Conclusion. Modeling of acute pancreatitis was characterized by a cyclic change in the activity of enzymes specific for this disease in the peripheral blood. With a relatively low activity of lipase in the peripheral blood on days 1–3 of the modeling of the pathological process, the activity of α-amylase had more significant values in changes in relation to intact animals of the control series of experiments. Considering that all animals were in a state of starvation, in the dynamics of the study, the correlation between the activity of α-amylase and lipase in the control series of experiments was direct, while in modeling acute pancreatitis it increased without changing its correlative character. However, one should also consider the fact that the correlation value of the increase in α-amylase activity between the studied series of experiments was in a lower direct relationship than the activity of lipase. Indicators of the level of activity of α-amylase and lipase in peripheral blood in animals with various types of pancreatic necrosis can only indicate the presence of a destructive process in the pancreas but cannot give an accurate verification conclusion about ongoing inflammatory processes, both local and general. It is required to study more reliable indicators that could increase the level of reliability about ongoing pathological processes and allow making the right decision in choosing therapeutic measures.

Key words: pancreatic necrosis, pathogenesis of pancreatic necrosis, diagnosis of pancreatic necrosis, pancreatogenic enzymes, experimental models of pancreatic necrosis, pancreatogenic sepsis.

INTRODUCTION

Acute pancreatitis is a polyetiological disease resulting from damage to the acinar cells of the pancreas, hypersecretion of pancreatic juice and difficulty in its outflow with the development of acute hypertension in the pancreatic duct, with the activation of enzymes of the gland itself. Moreover, for the development of destructive forms of pancreatitis, a combination of several adverse factors is necessary. [3,6,10,29]

According to the literature, there are about 140 different factors: leading to the development of acute pancreatitis. Listing all the etiological factors in the development of acute pancreatitis does not make sense, however, summing up the literature data, the approximate distribution of the causes of its occurrence is as follows: alcohol abuse - an av-

erage of 4 0%; cholelithiasis - 40%; different (taking medications and drugs, intoxication, trauma, hyperlipidemia, hypercalcemia, etc.) - 10%; idiopathic acute pancreatitis - 10%. [4,8,12,24]

The development of the destructive process in acute pancreatitis occurs in time in three phases (enzymatic, reactive-infiltrative, purulent-necrotic), each and from which has its own characteristic features. [1,2,5,9]

Phase I - enzymatic - develops during the first 5 days from the onset of the disease and is characterized by the formation of foci of necrosis in the pancreatic tissue, interstitial edema of the gland and parapancreatic fiber, the formation of effusion in the abdominal cavity containing many proteolytic enzymes, as well as severe endogenous intoxication, microvasculatory disorders and pronounced abdominal syndrome in the upper floor of the abdominal cavity.

Phase II - reactive-infiltrative - develops at the 2nd week after a short-term 1-2-day light interval and is characterized by the formation of a dense, palpatively determined infiltrate in the upper abdomen, as well as signs of resorptive fever.

Phase III - purulent-necrotic - develops at the 3rd week of the pathological process and is characterized by melting and sequestration * of necrotic areas of the pancreas and retroperitoneal tissue with the formation of purulent-necrotic foci in the pancreatic tissue and the formation of phlegmon in the parapancreatic fiber, retroperitoneal space, as well as severe intoxication, hectic fever. The most severe form of acute pancreatitis remains a common infected pancreatic necrosis (extensive phlegmon of retroperitoneal tissue), occupying three or more cellular spaces in the retroperitoneal space (parapancreatic, paranephral and paracolic left and right, paraaortic, mesocolon fiber, mesentery of the small intestine)

The pathogenesis of acute pancreatitis is not completely clear. Currently, most scientists adhere to the enzymatic theory of the pathogenesis of acute pancreatitis. A universally recognized link in the pathogenesis is the activation of the pancreas' own enzymes (trypsin, chemotrypsin, elastase, lipase, phospholipase, etc.) by lysosomal cytokinases. In this whole chain of organ and systemic damage, the role of the triggering and infecting factor is given to the reaction of pathological activation of trypsinogen with the formation of an activator of all pancreatic enzymes - trypsin. The mechanism of conversion of trypsinogen to trypsin is recognized as universal for the initial stages of development of any forms of acute pancreatitis. Enterokinase has a pronounced resistance to the action of proteases and is not inhibited by any of the known natural protease inhibitors. Enterokinase has a high specificity to only one single proenzyme of the human body. The effect of enterokinase on trypsinogen is to spatially rearrange and stabilize the active site and the entire trypsin molecule. The release of active trypsin leads to the activation and inclusion in the pathological process of a group of pancreatic secretory and tissue enzymes - an enzyme cascade that, together with the products of autolysis, has a damaging effect on the gland itself, surrounding tissues and the whole organism as a whole.

The study of the pathogenetic aspects of the development of pancreatic necrosis requires a more in-depth analysis of the changes occurring in the body, which can be carried out first in the experiment, and only then - tested in clinical conditions.

In this regard, it seems to us that conducting an equal clinical and laboratory assessment of the course of various experimental models of pancreatic necrosis will reveal several fundamental patterns that underlie severe cases of this necrobiotic process.

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 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 modeling 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. 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 5th 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 modeling acute infected pancreatic necrosis, complicated by sepsis in small laboratory animals, was carried out as follows. At the first stage, in order 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 animal. After another 3 days of experimental modeling of the pathological process, the above-described methods of modeling 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 from 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.

Blood in animals was obtained from the tail vein. The activity of enzymes α-amylases (IU / ml) and lipases (U / ml) in the blood serum was studied on the biochemical analyzer "ASRAMED" (China), using a set of dry chemistry reagents. Studies were conducted in dynamics on 1,3,7,14 days of modeling 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 was 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 first stage in the study of the features of the course of the experimental model of pancreatic necrosis, complicated by sepsis, in our opinion, should be reduced to an assessment of the state of the enzymatic system of the pancreas. The most significant among them, according to the Atlantic classification of acute pancreatitis, are indicators of activity in the blood of α-amylase and lipase.

Analysis of the dynamics of changes in the level of α-amylase and lipase in peripheral blood serum in animals with different models of acute pancreatitis was not unambiguous in the arithmetic value but had a clear picture of the regular flow of digital significance. This is important, since according to one of the three criteria of the Atlanta classification, in order to make a diagnosis of acute pancreatitis, it is required to have a 3-fold increase above the upper limit of the norm of serum α-amylase or lipase.

The average level of increased serum α-amylase activity in animals with acute pancreatitis was 632.31±116.82 U/ml, while lipases were only 11.55±1.31 U/ml (Table 1). A significant jump in the activity of α-amylase in the dynamics of modeling acute pancreatitis was recorded by us on the 7th day of observation (by 398.9±113.22 U/ml; $p<0.05$). The average increase in the activity of this enzyme at the level from 266.2±100.95 IU/ml ($p<0.05$) to 267.1±111.01 U/ml ($p< 0.05$) was recorded by us on the 1st and 14th day of the development of the pathological process. All this can be confidently interpreted as the presence of a turning point in the modeling of acute pancreatitis occurring on the 7th day and the final modeling on the 14th day of the dynamics of the course of the experiment. However, when studying the level of change in the activity of α-amylase in comparison with the control series of experiments, it is possible to note a progressive increase in its value in all periods of the dynamics of the pathological process. So, if on the 1-3-day of reproduction of the experimental model of acute pancreatitis, the level of activity of α-amylase in the peripheral blood increased by an average of 1.65±0.22 times ($p<0.05$), then on the 7-14th day - by 2.75±0.36 times ($p<0.05$). It should be noted that the 14th day of development of the experimental model of acute pancreatitis in terms of the activity of

α-amylase in peripheral blood fully corresponded to one of the criteria of the Atlanta classification for the diagnosis of this disease (the increase was 3.0±0.21 times (p<0.05).

Table 1

Comparative nature of changes in enzyme activity in peripheral blood in the dynamics of modeling acute pancreatitis

DYNAMICS	ENZYME ACTIVITY		R
	α-amylase (U/ml)	lipase (U/ml)	
Control	532,45±111,52	29,75±13,65	0,800± 0,012
1-day	798,6±129,4	35,8±7,4	0,858± 0,015*
3-day	931,9±111,02*	37,1±6,4*	
7-day	1330,8±116,5*	38,5±9,2*	
14-day	1597,8±215,4*	53,8±9,8*	
<x>	1164,76±211,4*	41,30±10,4*	
R	0,329±0,054	0,684±0,031	

*p<0,05 – reliably in relation to the control series of experiments.

Regarding the nature of the change in lipase activity in peripheral blood in the dynamics of modeling acute pancreatitis, it should be noted relatively low percentages of change compared to the control series of experiments during 1-7 days of the pathological process (an average of 1.25±0.09 times; p<0.05). Only on the 14th day of observation was a jump in activity of 1.8±0.08 times (p<0.05) revealed.

When assessing the level of activity of the studied enzymes in peripheral blood in the dynamics of modeling uninfected and infected pancreatic necrosis, almost identical nature of the changes was revealed (Tables 2 and 3).

Table 2

Comparative nature of changes in enzyme activity in peripheral blood in the dynamics of modeling acute uninfected pancreatic necrosis.

DYNAMICS	ENZYME ACTIVITY		R
	α-amylase (U/ml)	lipase (U/ml)	
Control	32,45±11,52	29,75±13,65	0,800± 0,012
1-day	1331,0±117,4*	56,6±4,9*	0,970± 0,011*
3-day	1863,8±110,8*	66,8±9,1*	
7-day	2395,4±112,4*	74,0±8,4*	
14-day	2929,3±116,9*	95,7±10,5*	
<x>	2129,85±111,4*	73,28±9,4*	
R	0,401±0,024	0,428±0,072	

*p<0,05 – reliably in relation to the control series of experiments.

It should be borne in mind that these models of the pathological process were reproduced already against the background of an experimental model of acute pancreatitis.

The average increase in the activity of α-amylase in peripheral blood in animals of the 3rd series of experiments was 1597.4±112.31 U/ml (p<0.05) greater than in intact animals (the increase was 4 times). At the same time, the level of lipase activity in the peripheral blood in this series of experiments increased compared to intact animals by only 43.53±9.18 U / ml (p<0.05), that is, by 2.46 times.

This dynamic was expressed in relation to α-amylase, the activity of which in the peripheral blood in animals of the 3-series of experiments increased already on the 1st day of the study by 2.5 times, on the 3rd day - by 3.5 times, on the 7th day by 4.5 times, and on the 14th day - by 5.5 times. In relation to lipase, in animals with uninfected pancreatic necrosis of 1-3 days, the dynamics of the pathological process did not differ progress. However, on the 7-14th day, the level of activity of this enzyme increased significantly by 2.5 and 3.2 times compared with the control series of experiments.

Table 3

Comparative nature of changes in enzyme activity in peripheral blood in the dynamics of modeling acute infected pancreatic necrosis.

DYNAMICS	ENZYME ACTIVITY		R
	α-amylase (U/ml)	lipase (U/ml)	
Control	32,45±11,52	29,75±13,65	0,800± 0,012
1-day	2662,0±121,6*	89,4±6,5*	0,976± 0,011*
3-day	2928,8±122,4*	96,5±8,7*	
7-day	3193,8±125,45*	103,6±10,9*	
14-day	3461,9±129,8*	119,6±11,4*	
<x>	3061,61±120,4*	102,28±12,5*	
R	0,402±0,062	0,408±0,024	

*p<0,05 – reliably in relation to the control series of experiments.

A similar identity of changes was noted by us in 4 series of experiments.

The average level of increased activity of α-amylase in peripheral blood in animals with pancreatic sepsis was 7.88±0.52 (p<0.05) times, while lipases - 6.13±0.54 (p<0.05) times (Table 4). In both cases, the average value was ascertained starting from 7 days of the pathological process (8.0±0.94; p<0.05 and 6.5±0.22; p<0.05 times, respectively). In the fractional value, the level of activity of α-amylase was the maximum. on the 14th day of the development of the pathological process, amounting to 535.0±114.12 U / ml (p<0.05), while in relation to the activity of lipase, this nature of changes accounted for the 7th day of reproduction of pancreatic sepsis (46.8±4.34 U / ml; r<0.05).

The correlation value between the studied indicators of peripheral blood of animals was higher than

the control level by 0.198±0.003 (p<0.05), and was the highest possible level compared to other series of experiments.

Table 4

Comparative nature of changes in the activity of enzymes in peripheral blood in the dynamics of modeling pancreatic necrosis complicated by sepsis.

DYNAM-ICS	ENZYME ACTIVITY		R
	α-amylase (U/ml)	lipase (U/ml)	
Control	32,45±11,52	29,75±13,65	0,800± 0,012
1-day	3726,8±115,2*	134,1±14,1*	0,998± 0,001*
3-day	3993,8±122,1*	163,4±12,5*	
7-day	4258,4±119,7*	192,4±19,4*	
14-day	4793,4±114,6*	239,2±19,8*	
<x>	4193,09±129,7*	182,26±15,4 *	
R	0,531±0,009	0,320±0,022	

*p<0,05 – reliably in relation to the control series of experiments.

For individual correlation values, the dispersion value between α-amylase and lipase was only 0.211±0.001 (p<0.05), although the overall nature of the changes remained the same.

Thus, modeling of pancreatic sepsis, on the one hand, gives a more pronounced picture of changes in the quantitative value of the activity of enzymes α-amylase and lipase in the peripheral blood of animals. All this indicates the layering of several pathological processes into a single model, which determines only the severity of the course of the disease. In other words, the more severe the course of pancreatic necrosis, the more pronounced the change in the activity of the enzymes under study (α-amylase and lactase) in the peripheral blood. Meanwhile, as in the case of the previous series of experiments, in order to obtain a reliable difference in the severity of the destructive process in the pancreas, they are not suitable, since a long-term dynamic measurement of parameters is required, which only in aggregate allow us to judge the dynamics of the process, but not about structural changes in the pancreas. Apparently, in conditions when the generalization of the inflammatory process is already prevalent, which occurred in animals with pancreatic sepsis, the prevailing changes were allocated to other blood parameters that can reflect the essence of the changes taking place, while the duration of the pathological process is manifested only by a stable increase in the activity of the blood enzymes under study.

DISCUSSION

It is known that in acute pancreatitis, the indicators of α-amylase usually increase earlier than the level of lipase. [1 3,14,1 6,17, 23] This conclusion was confirmed in our studies. At the same time, an active increase in the level of lipase in the peripheral blood in animals when modeling acute pancreatitis was noted by us on day 1-3 of the course of the pathological process (on average by 3.75±0.52 U / ml; r<0.05). At the same time, interest is also attracted by an increase in lipase activity on the 7-14th day of modeling the pathological process (on average 15.3±1.15 U / ml (p<0.05).

And the changes in the activity of enzymes α-amylase and lipase in the peripheral blood of animals with both uninfected and infected pancreatic necrosis are identical, despite their different arithmetic values. The relatively high activity of α-amylase in the peripheral blood of animals, compared with lipase, is due to the presence of different sources of origin of this enzyme. So, for example, it is known that α-amylase can be formed not only in the pancreas, but also in the muscles of the skeleton, in the intestines and even in the ovaries. [20] Accordingly, in this case, the determining role is played by the timing of the disease rather than the substrate of the pathological process itself.

CONCLUSION

The treatment of acute pancreatitis was characterized by a cyclic change in the activity of enzymes specific to this disease in the peripheral blood. With a relatively low lipase activity in the peripheral blood for 1-3 days of modeling the pathological process, the activity of the α-amylase had more reliable values in the changes in relation to intact animals of the control series of experiments. Given that all animals were in a state of hunger, in the dynamics of the study, the correlation between the activity indices of α-amylase and lipase in the control series of experiments was direct, whereas when modeling acute pancreatitis, it increased without changing its relative nature. However, it should be borne in mind that the correlation value of the increase in the activity of α-amylase between the studied series of experiments was in a low direct relationship than the activity of lipase.

Indicators of the level of activity of α-amylase and lipase in the peripheral blood in animals with various variants of pancreatic necrosis can only indicate the presence of a destructive process in the pancreas but cannot give an accurate verification conclusion about the ongoing inflammatory processes, both local and general. It is necessary to study more reliable indicators that could increase the level of reliability about the pathological processes occurring and allow to allow make the right decision in the choice of therapeutic measures.

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.

BIBLIOGRAPHY:

1. Acute Purulent-Destructive Lung Diseases as Consequences of Endotheliitis after COVID-19 // Sh.A. Bobokulova, A.O. Okhunov // Publication date – 2022, Journal of education and scientific medicine. -Volume 2, Issue 3, Pages 56-61.

2. Angiogenesis Inhibitor Vasohibin-1 Enhances Stress Resistance of Endothelial Cells via Induction of SOD2 and SIRT1. // H.Miyashita, T.Watanabe, H.Hayashi1, Y.Suzuki, T.Nakamura, S.Ito et al. // PLoS ONE. 2012; (7): e46459.

3. Arginine methylation dysfunction increased risk of acute coronary syndrome in coronary artery disease population. // S.Zhang, S.Zhang, H.Wang, W.Wu, Y Ye. // Medicine. 2017; 96: 7:e6074.

4. Asymmetric Dimethylarginine: Clinical Significance and Novel Therapeutic Approaches. // D.Tousoulis, M.K.Georgakis, E.Oikonomou, N.Papageorgiou, M.Zaromitidou, G.Latsios et al. // Curr. Med. Chem; 2015; (22): 2871–901.

5. Endothelin // A.P.Davenport, K.A.Hyndman, N.Dhaun, C.Southan, D.E.Kohan, J.S.Pollock, D.M.Pollock, D.J.Webb, J.J.Maguire. // Pharm. rev. 2016; 68: 357–418.

6. Intestinal Peptides and their Main Role in the Pathogenesis of Type 2 Diabetes Mellitus / P.Kh. Azizova, Sh.R. Razzakov, I.A. Marupov, A.O. Okhunov, A.M. Abdurakhmanov, D.N. Korikhonov, I.Yu. Yakubov, A.Sh. Yarkulov, Sh.A. Khamdamov // Publication date - 2022/12.- Journal of education and scientific medicine. – Volume 2, Issue 3, Pages 95-100.

7. K voprosu o prognosticheskoy znachimosti S-reaktivnogo belka. // T.V. Blinova, R.S.Rahmanov, L.A.Strahova, S.A.Kolesov // Meditsinskiy almanah. 2016; 2(42): 39–43. [in Russian]

8. Kulik Ye.G., Pavlenko V.I., Narishkina S.V. Faktor Villebranda i disfunktsiya sosudistogo endoteliya u bolnih xronicheskoy obstruktivnoy boleznyu legkih. Amurskiy meditsinskiy jurnal. 2017; 1(17):41–3. [in Russian]

9. Marshall J.C., Cook D.J., Christou N.V., et al. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med. 1995 Oct;23(10):1638-52. Review. PubMed ID: 7587228.

10. Melnik A.A. Narushenie sistemi gemostaza pri sepsise // Xirurgiya, Ortopediya, Travmatologiya, Intensivna terapiya - 2020, № 2 (40).-S.12-16. [in Russian]

11. Method for experimental modeling of diabetic angiopathy / Alisher O Okhunov, Inayat S Sattarov, Feruza Kh Inoyatova, Shukhrat R Razzakov // Publication date 2020.- Central Asian Journal of Medicine - Volume 2020, Issue 3, Pages 92-100.

12. Methods of endothelial function assessment: description and applications. // A.S.Storch, J.D.de Mattos, R.Alves, I.S.Galdino, H.N.M.Rocha // Int. J. Cardiovasc. Sci. 2017; 30(3): 262–73.

13. Morphological aspects of wounds in patients with purulent inflammation of soft tissues in diabetes mellitus and under the influence of granulocyte-colony-stimulating factor // U.I. Pulatov, R.I. Israilov, A.O. Okhunov, F.M. Abdurakhmanov, K.H. Boboev // Publication date 2022.- Journal of education and scientific medicine.- Volume 2, Issue 3.- Pages 43-50.

14. Morphological Characteristics of a New Experimental Model of Chronic Renal Failure in the Background of Diabetic Nephropathy / Authors: A.K. Shadmanov, A.O. Okhunov, F.M. Abdurakhmanov // Publication date – 2022, Journal of education and scientific medicine – Volume 2, Issue 3, Pages 68-76.

15. Morphological characteristics of results of treatment of diabetic angiopathy / AO Okhunov // 2019/6/6 –International journal of diabetes and metabolic disorders. – Volume 4, Issue 4, Pages 1-3.

16. Page A.V., Liles W.C. Biomarkers of endothelial activation dysfunction in infectious diseases. Virulence. 2013; 4(6): 507–16.

17. Politov S. Ya., Balnikov S.I. Viyavlenie grupp riska ranney letalnosti bolnih pankreonekrozom po znacheniyam amilazi krovi v nachale zabolevaniya // Vestnik Natsionalnogo mediko-xirurgicheskogo centra im. N. I. Pirogova 2016, T. 11, № 1-S.56-58. [in Russian]

18. Principles of diagnosis and treatment of acute purulent-destructive lung diseases / A.O. Okhunov, K.X. Boboev, A.F. Valijonov, Sh.A. Valijonova // Publication date - 2022/2/13, World Bulletin of Public Health, Volume 7, Pages 1-2.

19. Prognozirovanie i rannyaya diagnostika poliorgannoy nedostatochnosti / A.S. Radivilko, Ye. V. Grigorev, D.L. Shukevich, G. P. Plotnikov // Anesteziologiya i reanimatologiya, 2018, №6, C. 15 –21 - https://doi.org/10.17116/anaesthesiology201806115 (4.2-5) [in Russian]

20. Shleykin A.G., Skvortsova N. N., Blandov N.N. Prikladnaya enzimologiya. – SPb: Universitet ITMO, 2019–160 s. [in Russian]

21. Sokolov E.I., Grishina T.I., Shtin S.R. Effect of von Willebrand Factor and Endothelin-1 on Formation of Thrombotic Status in Patients with Ischemic Heart Disease. Kardiologiya. 2013; (3): 25–30. [in Russian]

22. Sokolov Ye.I., Grishina T.I., Shtin S.R. Vliyanie faktora Villebranda i endotelina-1 na formirovanie tromboticheskogo statusa pri ishemicheskoy bolezni serdtsa. Kardiologiya. 2013; (3):25–30. [in Russian]

23. Strang F., Schunkert H. C-Reactive Protein and Coronary Heart Disease: All Said – Is Not It? // Mediators of Inflammation. 2014; Article ID 757123.

24. The diagnostic value of adhesive molecules sICAM-1 and sVCAM-1 in ischemic heart disease. // I.S. Belokopytova, O.V.Moskaletz, F.N. Paleev, O.V. Zotova // Ateroskleroz i dislipidemii. 2013; 4: 62–5. [in Russian]

25. The reasons for the generalization of infection in patients with purulent-inflammatory diseases of soft tissues against the background of diabetes

mellitus / AO Okhunov, BD Babadzhanov, UI Pulatov // Publication date 2016, Bulletin of the Tashkent Medical Academy, Volume 4, Pages 89-93.

26. The Role and Place of Nitroxidergic Regulation of The Endothelial System in the Pathogenesis of Acute Lung Abscess / A.O Okhunov, Sh.A. Bobokulova // Publication date - 2022/11/23, Research Square – Volume 1, Issue 11, Pages 1-10. DOI: https://doi.org/10.21203/rs.3.rs-23

27. Vascular endothelial growth factor and the tumors of female reproductive system. // E.S.Gershteyn, D.N.Koushlinskiy, I.V.Tereshkina, V.D.Ermilova, L.K. Ovchinnikov, D.E. Galdava, O.V. Kouznetsova // Part I. Breast cancer. Onkoginekologiya. 2015;(1): 34–41. [in Russian]

28. VEGF expression in pancreatic cancer and other malignancies: a review of the literature. // M.I. Costache, I.Mihai, S.Iordache, D.Ene, C.A.Costache, A.Săftoiu // Rom. J. Intern. Med. 2015; 53(3):199–208.

29. Wu M.D., AtkinsonT.M., Lindner J.R. Platelets and von Willebrand factor in atherogenesis. BLOOD. 2017; 129 (11): 1415-19.

SEPSIS BILAN ASORATLANGAN PANKREANEKROZ KLINIK-LABORATOR KECHISHINING QIYOSIY MEZONI

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Abstrakt

Dolzarbligi. O'tkir pankreatitning patogenezi to'liq tushunilmagan. Hozirgi vaqtda ko'pchilik olimlar o'tkir pankreatit patogenezining fermentativ nazariyasiga amal qilishadi. Pankreatik nekroz rivojlanishining patogenetik jihatlarini o'rganish organizmda davom etayotgan o'zgarishlarni chuqurroq tahlil qilishni talab qiladi, bu o'zgarishlarni birinchi bo'lib eksperimentda o'tkazish va shundan keyingina klinik sharoitda tekshirish mumkin.

Tadqiqot. Eksperimental tadqiqotlar Wistar liniyasining oq laboratoriya kalamushlarida o'tkazildi. Har ikki jinsdagi 150-250 gramm og‘irlikdagi, kasallik belgilari bo‘lmagan jami 170 bosh hayvonlar ishlatilgan. Tadqiqotlar o'tkir pankreatit, steril pankreonekroz, infektsiyalangan pankreanekroz va sepsis bilan asoratlangan oshqozon osti bezi nekrozining eksperimental modellari bilan hayvonlarda o'tkazildi. Qon zardobidagi a-amilaza va lipaza fermentlarining faolligi o'rganildi.

Xulosa. O'tkir pankreatitni modellashtirish periferik qonda ushbu kasallik uchun xos bo'lgan fermentlar faolligining tsiklik o'zgarishi bilan tavsiflangan. Patologik jarayonni modellashtirishning 1-3-kunlarida periferik qonda lipazaning nisbatan past faolligi bilan, a-amilaza faolligi nazorat qatoridagi buzilmagan hayvonlarga nisbatan o'zgarishlarda sezilarliroq qiymatlarga ega bo'ldi. Barcha hayvonlar ochlik holatida bo'lganligini hisobga olsak, tadqiqot dinamikasida a-amilaza va lipaza faolligi o'rtasidagi bog'liqlik nazorat qatoridagi tajribalarda to'g'ridan-to'g'ri bo'lsa, o'tkir pankreatitni modellashtirishda uning korrelyativ xarakterini o'zgartirmasdan o'sdi. Shu bilan birga, shuni ham hisobga olish kerakki, o'rganilayotgan tajribalar seriyasi o'rtasidagi a-amilaza faolligi oshishining korrelyatsiya qiymati lipaza faolligiga qaraganda pastroq to'g'ridan-to'g'ri bog'liqlikda edi. Har xil turdagi pankreatik nekrozga uchragan hayvonlarda periferik qondagi a-amilaza va lipaza faollik darajasining ko'rsatkichlari faqat oshqozon osti bezida vayron qiluvchi jarayon mavjudligini ko'rsatishi mumkin, ammo davom etayotgan yallig'lanish jarayonlari to'g'risida to'g'ri tasdiqlovchi xulosani bera olmaydi. Davom etayotgan patologik jarayonlarning ishonchlilik darajasini oshiradigan va terapevtik choralarni tanlashda to'g'ri qaror qabul qilishga imkon beradigan ishonchli ko'rsatkichlarni o'rganish talab etiladi.

Kalit so'zlar: oshqozon osti bezi nekrozi, oshqozon osti bezi nekrozining patogenezi, oshqozon osti bezi nekrozining diagnostikasi, pankreatogen fermentlar, oshqozon osti bezi nekrozining eksperimental modellari, pankreatogen sepsis.

СРАВНИТЕЛЬНАЯ КЛИНИКО-ЛАБОРАТОРНАЯ ХАРАКТЕРИСТИКА ТЕЧЕНИЯ ЭКСПЕРИМЕНТАЛЬНОЙ МОДЕЛИ ПАНКРЕОНЕКРОЗА, ОСЛОЖНЕННОГО СЕПСИСОМ

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Абстракт

Актуальность. Патогенез острого панкреатита до конца не ясен. В настоящее время большинство учёных придерживаются ферментативной теории патогенеза острого панкреатита. Исследование патогенетических аспектов развития панкреонекроза требует более углубленного анализа происходящих изменений в организме, которые возможно провести вначале в эксперименте, и лишь потом – апробировать в клинических условиях.

Методы. Экспериментальные исследования проведены на белых лабораторных крысах линии Wistar. Всего было использовано 170 животных весом 150-250 грамм, обоего пола, без внешних признаков заболевания. Исследования проведены на животных с экспериментальными моделями острого панкреатита, стерильного панкреонекроза, инфицированного панкреонекроза и панкреонекроза, осложненного сепсисом. Исследовали активность ферментов α-амилаза и липаза в сыворотке крови.

Заключение. Моделирования острого панкреатита характеризовалось цикличным изменением активности специфических для данного заболевания ферментов в периферической крови. При относительно низкой активности липазы в периферической крови на 1–3-сутки моделирования патологического процесса, активность α-амилазы имела более достоверные значения в изменениях по отношению к интактным животным контрольной серии опытов. Учитывая, что все животные находились в состоянии голода, в динамике проводимого исследования, корреляционная зависимость между показателями активности α-амилазы и липазы в контрольной серии опытов была прямой, тогда как при моделировании острого панкреатита она, не изменяя свой соотносительный характер повышалась. Показатели уровня активности α-амилазы и липазы в периферической крови у животных с различными вариантами панкреонекроза могут свидетельствовать лишь о наличие деструктивного процесса в поджелудочной железе, но не могут дать точное верификационное заключение о происходящих воспалительных процессах, как локального, так и общего характера.

Ключевые слова: панкреонекроз, патогенез панкреонекроза, диагностика панкреонекроза, панкреотогенные ферменты, экспериментальные модели панкреонекроза, панкреатогенный сепсис.