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A Method for Modelling a New Experimental Model of Pancreatic Necrosis Complicated by Sepsis

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ABSTRACT

In our time, finding a more complex inflammatory disease of the abdominal organs in its pathogenesis is more difficult than acute pancreatitis. Over the past 50 years, acute pancreatitis ranks third among acute surgical diseases of the abdominal cavity and accounts for about 12.5% of all urgent pathology. At the same time, diagnostics and surgical tactics for pancreatic necrosis remain one of the far-fetched problems in urgent abdominal surgery. There is no doubt that this problem is related to the difficulties of prognosis and early diagnosis of destructive forms of acute pancreatitis. In the modern view of the mechanism of development of any variant of sepsis, the process associated with the development of the syndrome of systemic inflammatory reaction is put in the first place. At the same time, the presence of a purulent focus in the body, and even more so in the presence of organ dysfunction of at least one vital organ, corresponds to the full presentation of the verdict of surgical sepsis. It is based on these considerations that the main objectives of this article, reflecting the results of our study, were to develop an experimental model of pancreatic necrosis complicated by sepsis, in which the trigger will be the general reaction of the body that takes place in clinical practice.

Keywords: *pancreatic necrosis, pancreatogenic sepsis, experimental modelling*

INTRODUCTION

The clinical picture of the systemic inflammatory response syndrome is a fairly common severe complication in patients with pancreatic necrosis [1-4].

This prompted clinicians to single out a separate form from the group of abdominal sepsis as pancreatogenic sepsis [5-7].

Such a step characterises the peculiarity of the development of pancreatogenic sepsis, which, unlike abdominal sepsis, begins with an aseptic process [8-11].

Only with the addition of an infectious agent, pancreatogenic sepsis, can a complete picture corresponding to abdominal sepsis [12-15].

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The Chicago Consensus Conference on Sepsis back in 1991 made it possible to obtain analytical information on the incidence of sepsis [16].

At the conference, it was decided to divide all forms of generalisation of the inflammatory response of the body into systemic inflammatory response syndrome, sepsis syndrome, severe sepsis and septic shock. At the same time, according to the decision of this conference, it is the development of multiple organ dysfunction or multiple organ failure syndrome in patients with severe sepsis and septic shock that should be considered the leading initiators of the lethal outcomes of this disease.

Studies have long been carried out to model pancreatitis and pancreatic necrosis [17-20].

Meanwhile, experimental modelling of pancreatic necrosis in the standard version of its reproduction does not always allow the form of the course of the disease in the form of a systemic inflammatory reaction syndrome or sepsis syndrome as the initial phase of the development of pancreatogenic sepsis [21-23].

The most popular variant of modelling pancreatitis and pancreatic necrosis in experimental animals are methods with isolated ligation of the Virsung's pancreatic duct. The reproducibility of pancreatitis begins three days after the intervention. However, any process occurring in animals with such an experimental model does not characterise the development of pancreatogenic sepsis. In other words, there are no characteristic clinical manifestations of generalisation infections that determine the symptom complex of any surgical sepsis (body temperature above 38 ºC or below 36 ºC, tachycardia over 90 beats/min, tachypnea over 20 breaths per 1 minute, leukocyte count over $12x10^9$/l or below $4x10^9$/l, or the number of their immature forms exceeds 10%). In the development of sepsis syndrome, the presence of a purulent focus of infection and possible bacteremia is characteristic. All of the above characterise the main phases of the development of sepsis. It is this approach that should determine the process of the course of pancreatogenic sepsis, which is close to clinical conditions. To create conditions that meet these conditions, we conducted several experimental simulations of pancreatogenic sepsis.

MATERIAL AND METHODS

In the first stage, it was necessary to determine the dose of the injected microbial agent. For this purpose, when simulating pancreatogenic sepsis, 1 ml of a 0.9% sodium chloride solution containing a virulent microbial culture of Escherichia Colli at a dose of 20–30 million microbial bodies per 1 gram of animal weight was injected into the pancreas after repeated laparotomy after repeated laparotomy. The choice of the dose of injected microbial bodies is because when injecting animals with relatively small doses (up to 20 million microbial bodies per 1 gram of animal weight), in 5 out of 7 cases, pancreatogenic sepsis did not develop, and there was a regression of the inflammatory process (series 1). In the abdominal cavity, an inflammatory process of a non-infectious nature was detected, and an adhesion process was formed at the injection site.

The animals showed a picture of non-infected pancreatic necrosis. Only in 2 (28.6%) animals in this series of experiments did the autopsy reveal the presence of a limited purulent inflammatory process in the form of an abscess of the abdominal cavity. We did not obtain a generalisation of the inflammatory response.

At the same time, in the second series of experiments (series 2), the dose of microbial bodies in the injected suspension was over 30 million microbial bodies per 1 gram of animal weight. In this series of experiments, 6 out of 7 animals died within 24 hours of injection. At autopsy, signs of the development of infectious and toxic shock were found (putrefactive reserve in the abdominal cavity, hemorrhagic effusion in the abdominal cavity and pleural cavity, acute plethora of internal organs, multiple foci of haemorrhage in the visceral and parietal sheets of the peritoneum), which was associated with the massive intake of microbial bodies into the body. That is, despite the development of the inflammatory process, the latter was of a formal nature since, in fact, we were dealing with the development of septic shock, the entrance gate of which was the abdominal cavity.

With an increase in the number of microbial bodies injected, a lightning-fast course of the inflammatory process occurs without any stages of pancreatogenic sepsis, which takes place in clinical practice. It should also be noted that the process of infected pancreatic necrosis simply does not have time to form, transferring the entire inflammatory reaction from local to general, where the main culprit is the peritoneum and not the pancreas. The inflammatory reaction in the pancreas was no different from the other area of the abdominal cavity, and the lesion proceeded without the formation of a necrobiotic process, and even more so parapancreatic necrosis.

Thus, at this stage of the research, it can be assumed that such an atypical picture of the course of pancreatogenic sepsis is associated, on the one hand, with the use of monoculture of pathogens (in this situation, there was no interspecific struggle of microorganisms). The created conditions allowed the entry of a massive number of microorganisms into the systemic circulation and the development of corresponding changes without an ade-

quate response from the body. On the other hand, the use of monoculture in modelling pancreatogenic sepsis does not correspond to the clinical conditions of the onset of the disease since, in life practice, in the occurrence of any purulent-inflammatory process, the prevailing condition is the presence of poly injection. The latter is the main argument in favour of using suspension obtained from the autocal of the animals themselves as a microbial agent.

To select the dose of the injected animal autocal, we conducted several microbiological studies. It was revealed that the microbial contamination of autocal animals obtained from the rectal cavity was represented by a large number of different microorganisms, both aerobic and anaerobic. In the studied material, the total number of microbial bodies was equal to the value of 10^9-10^{10} CFU/ml. Gram-negative pathogens prevailed (72.4%) over gram-positive ones.

Among the gram-negative ones, Veillonella, Enterobacteriales, Klebsiella, and Proteus were prevalent. In 72% of cases, rod-shaped pathogens were detected and in 28% - cocci. Of the total microbial landscape of the faecal mass of the animal, 68.6% were constitutive groups of obligate anaerobes, and 31.4% were facultative anaerobes.

When choosing the appropriate dose of the injected microbial suspension, we found that the level of admissibility of the reproduction of the purulent-inflammatory process (infection) is x10^4 CFU/ml, which corresponds to 20% of the concentration of the animal autocal solution. The maximum value of microbial invasion in the form of a critical level should be considered the concentration at the level of x10^5 CFU/ml.

The choice of the concentration of microbial suspension of animal autocal was confirmed by us in the following 3rd and 4th series of experiments. When injecting a solution with an amount exceeding x10^5 CFU/ml, bacterial shock also developed. Whereas when injecting a microbial agent in the amount of x10^3 CFU/ml, the inflammatory process, as in the previous block of experiments, simply did not develop.

It should also be noted that pancreatic necrosis simply does not have time to develop in the version that was necessary for the formation of pancreatogenic sepsis. In this regard, we decided to stimulate the necrobiotic process with 10% calcium chloride solutions. Accordingly, the timing of the administration of both 10% calcium chloride and animal autocal should determine the timing of the formation of the main process.

As a result of the research, we made the following conclusions:

- when large doses of microbial suspensions are injected into the pancreas, even if they are polymorphic, the formation of infected pancreatic necrosis and pancreatogenic sepsis does not occur;

- the introduction of massive doses of microbial suspension provokes the development of infectious-toxic (septic) shock, with a high percentage of lethality and a lightning-fast course of the pathological process, which does not allow the model to be used for experimental studies;

- septic shock, which occurs with the administration of large doses of autocal in animals, along with a high percentage of early mortality, proceeds without the formation of purulent pancreatic necrosis and, accordingly, excludes the phases of formation of all links of pancreatogenic sepsis;

- the pancreas acts as an entrance gate for microorganisms when large doses of animal autocal are administered, and the formation of a persistent source of infection (purulent pancreatic necrosis) does not occur;

For the formation of pancreatogenic sepsis with such forms as severe sepsis and sepsis syndrome, preliminary changes in the macroorganism are required, which characterise the subsequent reaction of the animal body, that is, changes in the reactivity of the macroorganism are required, provided that the virulence of the microorganism decreases.

It is known that the process of pancreatogenic sepsis is already formed in the absence of infection in the pancreas. However, this reaction can only be interpreted as a syndrome of a systemic inflammatory reaction of the body, the cause of which may be pancreatic autolysis. Only with the addition of an infectious agent is it possible to form a classic infected pancreatic necrosis with the subsequent development of sepsis syndrome and severe sepsis. Proof of this judgment can be found in a series of experiments with a low concentration of injected animal autocal. The hyporgy reaction that arises in this case forms a picture of a conflict of the aggressive principle between the macroorganism and the microorganism.

Thus, the reproduction of infected pancreatic necrosis and pancreatogenic sepsis is possible only if there are initial destructive changes in the focus of inflammation (necrobiotic) and a decrease in the response of the macroorganism (immunosuppression). Only if the above conditions are met a low concentration of microbial suspension of animal autocal will increase the reproducibility of the required pathological process and experimental model. At the same time, the destructive process in the

pancreas can, as is known, be simulated using a 10% solution of calcium chloride.

To change the reactivity of the macroorganism, we used antilympholine-Cr, which is an immunosuppressive drug. It is obtained from rabbit blood proteins immunised by human thymic lymphocytes. One dose of the drug corresponds to 40-60 mg of protein. Along with this, as already mentioned above, the autocal of the animal is a source of polymorphic pathogenic flora. The latter makes it possible to bring the conditions for the development of the pathological process closer to the clinical ones.

To confirm our judgments, we conducted a new series of experiments (series 5) in which pancreatogenic sepsis was simulated by a preliminary, two-day intraperitoneal injection of antilympholine-Cr at a dose of 0.03 mg per 100 grams of the animal. On the 3rd day of modelling, laparotomy was performed, the stomach, duodenum together with the pancreas were removed into the wound, and Virsung's duct was ligated. After the formation of acute pancreatitis, which usually occurred on the 3rd day of the operation, the abdominal cavity was reopened, and in aseptic conditions to provoke a necrobiotic process, 0.5 ml of a 10% calcium chloride solution was injected into the pancreas. A day later, 0.5 ml of a 20% solution of animal auto-cal was injected into the pancreas through a laparotomy wound. In dynamics, starting from the first day after the injection of microbial faecal suspension, the development of pancreatogenic sepsis against the background of pancreatic necrosis was observed.

Over the next seven days, the animals developed a progressive clinical picture of all forms of pancreatogenic sepsis with signs of a systemic inflammatory reaction syndrome (tachycardia, tachypnea, hyperthermia, leukocytosis). The results of blood cultures in 100% of cases revealed the presence of blood culture already on the 3–4th day of modelling.

The pancreas was in a purulent-necrotic state during all the periods of the experiments. The purulent-destructive process easily spreads to nearby tissues, the hepatic gate, and the mesenteric root. Such results of modelling pancreatogenic sepsis took place in 11 (91.7%) out of 12 rats of this series, one rat died on the 1st day of observation with pathomorphological signs of bacterial shock revealed by us at autopsy. In our model of pancreatic necrosis complicated by sepsis, the initial signs of a systemic inflammatory response syndrome (in the form of respiratory failure, increased rectal body temperature, tachycardia, leukocytosis, or leukopenia) are observed at 10–12 hours of the experiment.

Based on the conditions of staged modelling of pancreatogenic sepsis, when describing the course of the pathological process, we evaluated each stage of the disease separately, which will allow us to identify the patterns of its development and develop methods for early diagnosis.

RESULTS AND DISCUSSION

The first stage on the way to studying 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 the indicators of blood activity of α-amylase and lipase.

The analysis of the dynamics of changes in the level of α-amylase and lipase in the peripheral blood serum in animals with different models of acute pancreatitis was not unambiguous in arithmetic value but had a clear picture of the regular flow of the numerical value. This is important, as one of the three criteria of the Atlantic Classification requires a 3-fold increase above the upper limit of normal serum α-amylase or lipase levels to make a diagnosis of acute pancreatitis.

The mean level of increase in serum α-amylase activity in animals with acute pancreatitis was 632.31±116.82 U/ml, while lipase was only 11.55±1.31 U/ml ($p<0.05$). A significant jump in α-amylase activity in the dynamics of modelling acute pancreatitis was recorded by us on the 7th day of follow-up (by 398.9±113.22 U/ml; $p<0.05$). The average increase in the activity of this enzyme at the level of 266.2±100.95 U/ml ($p<0.05$) to 267.1±111.01 U/ml ($p<0.05$) was recorded by us on the 1st and 14th days of the development of the pathological process. All this can be interpreted with confidence as the presence of a turning point in the modelling of acute pancreatitis, occurring on day 7, and the final simulation of the dynamics of the course of the experiment on day 14. However, when studying the level of changes 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 at all times of the dynamics of the pathological process. Thus, if on days 1–3 of the experimental model of acute pancreatitis, the level of α-amylase activity in the peripheral blood increased by an average of 1.65±0.22 times ($p<0.05$), then on days 7–14 – 2.75±0.36 times ($p<0.05$). It should be noted that the 14-day development of the experimental model of acute pancreatitis in terms of peripheral blood α-amylase ac-

tivity fully corresponded to one of the criteria of the Atlantic classification for diagnosing this disease (the increase was 3.0±0.21 times ($p<0.05$).

As for the nature of changes in lipase activity in peripheral blood in the dynamics of modelling acute pancreatitis, it should be noted that relatively low percentages of change compared to the control series of experiments during 1–7 days of the course of the pathological process (on average, 1.25±0.09 times; $p<0.05$). Only on the 14th day of follow-up a jump in activity by 1.8±0.08 times ($p<0.05$) was revealed.

It is known that in acute pancreatitis, α-amylase levels usually increase earlier than lipase levels [24].

This conclusion was confirmed in our research. At the same time, an active increase in the level of lipase in peripheral blood in animals during the modelling of acute pancreatitis was noted by us on the 1st–3rd day of the course of the pathological process (on average by 3.75±0.52 U/ml; $p<0.05$). At the same time, an increase in lipase activity on days 7–14 of modelling the pathological process by an average of 15.3±1.15 U/ml ($p<0.05$) is also of interest.

Thus, the modelling of acute pancreatitis was characterised by cyclic changes in the activity of disease-specific enzymes in the peripheral blood. With relatively low lipase activity in the peripheral blood on days 1–3 of the simulation of the pathological process, the activity of α-amylase had more reliable values in changes about intact animals in the control series of experiments. Taking into account that all animals were in a state of hunger, in the dynamics of the study, the correlation between the indicators of α-amylase and lipase activity in the control series of experiments was direct, while when modelling acute pancreatitis, it increased without changing its correlative nature. However, it should also be taken into account that the correlation value of the increase in the activity of α-amylase between the studied series of experiments was in a lower direct relationship than the activity of lipase.

When assessing the level of activity of the studied enzymes in the peripheral blood in the dynamics of modelling uninfected and infected pancreatic necrosis, almost identical changes were revealed. At the same time, it should be borne in mind that these models of the pathological process were already reproduced 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 ($p<0.05$) more than in intact animals (the increase was four times). At the same time, the level of lipase activity in the peripheral blood in this series of experiments increased by only 43.53±9.18 U/ml ($p<0.05$), that is, by 2.46 times, compared to intact animals.

This dynamics was expressed about α-amylase, the activity of which in the peripheral blood of animals of the 3rd series of experiments increased by 2.5 times on the 1st day of the study, 3.5 times on the 3rd day, 4.5 times on the 7th day, and 5.5 times on the 14th day. About lipase, in animals with uninfected pancreatic necrosis for 1–3 days, the dynamics of the pathological process did not show much progress. However, on days 7–14, the level of activity of this enzyme significantly increased by 2.5 and 3.2 times compared to the control series of experiments. A similar identity of changes was noted by us in 4 series of experiments.

Thus, 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 to lipase, is due to the presence of different sources of origin of this enzyme. 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 [25].

Accordingly, in this case, the timing of the disease plays a decisive role rather than the substrate of the pathological process itself. This makes it possible to conclude that the 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 ongoing pathological processes and allow you to make the right decision in the choice of therapeutic measures. As proof of our judgment, we cite the results of assessing changes in the level of activity of α-amylase and lipase in peripheral blood in animals with pancreatogenic sepsis.

The average level of increase in α-amylase activity in peripheral blood in animals with pancreatogenic sepsis was 7.88±0.52 ($p<0.05$) times, while lipase was 6.13±0.54 ($p<0.05$) times. In both cases, a higher-than-average value was found starting from the 7th day of the course 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 α-amylase activity was maximum on the 14th day of the development of the pathological process, amounting to 535.0±114.12 U/ml (p<0.05), while about lipase activity, this type of change occurred on the 7th day of pancreatogenic sepsis reproduction (46.8±4.34 U/ml; p<0.05).

The correlation value between the studied parameters of the peripheral blood of animals was higher than the control level by 0.198±0.003 (p<0.05) and was at the highest level compared to other series of experiments.

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

Thus, the modelling of pancreatogenic sepsis, on the one hand, gives a more pronounced picture of changes in the quantitative value of the activity of α-amylase and lipase enzymes 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 changes 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, they are not suitable for obtaining a reliable difference in the severity of the destructive process in the pancreas since a long-term dynamic measurement of parameters is required, which only in the aggregate makes it possible to judge the dynamics of the process, but not the structural changes in the pancreas. Apparently, in conditions where the generalisation of the inflammatory process is already prevalent, which took place in animals with pancreatogenic sepsis, the prevailing changes were assigned to other blood parameters that may reflect the essence of the changes occurring, while the duration of the pathological process is manifested only by a stable increase in the activity of the studied blood enzymes.

Of course, it is not possible to assess the dynamics of the clinical picture of acute pancreatitis in experimental studies in comparison with clinical ones. In this regard, the leitmotif in assessing the clinical picture of pancreatogenic sepsis, in our opinion, should come from the criteria of the syndrome of the systemic inflammatory response of the body.

The general characteristics of the dynamics of the increase in clinical and laboratory signs of the systemic inflammatory reaction syndrome made it possible to identify the stages of the formation of the model of the pathological process. At the same time, based on the maximum number of registered features, the entire total balance of stages is divided into three periods.

The first period, representing up to 50% of the total number of clinical and laboratory signs of the systemic inflammatory response syndrome, included models of acute pancreatitis and uninfected pancreatic necrosis on days 1–7, as well as infected pancreatic necrosis on day 1 of the development of the pathological process. The second period, representing 51% to 75% of the total number of clinical and laboratory signs of the systemic inflammatory response syndrome, included models of acute pancreatitis and uninfected pancreatic necrosis on day 14, as well as infected pancreatic necrosis on day 3 of the development of the pathological process. The third period, representing 76% to 100% of the total number of clinical and laboratory signs of systemic inflammatory response syndrome, included models of infected pancreatic necrosis on days 7–14, as well as all periods of pancreatogenic sepsis development.

Thus, the distribution of the total number of clinical and laboratory signs of the systemic inflammatory response syndrome in animals with different models of pancreatitis made it possible to reveal that the model of infected pancreatic necrosis proceeds gradually and for a longer time than all other series. The models of acute pancreatitis and uninfected pancreatic necrosis were distinguished by a low number of clinical and laboratory signs of the systemic inflammatory response syndrome, while under the conditions of a deliberate change in the reactivity of the animal body, the maximum number of symptoms of the disease was stated with the addition of pancreatogenic sepsis.

In the qualitative analysis of the clinical manifestations of the systemic inflammatory reaction syndrome in animals with acute pancreatitis, isolated signs of the process prevailed at all times of the experiments or did not appear at all. Only on the 14th day of the development of the pathological process in 50% of cases, 2 and 3 clinical and laboratory signs of the systemic inflammatory response syndrome were registered in the total value.

In animals with an experimental model of uninfected pancreatic necrosis, the presence of all signs of the systemic inflammatory reaction syndrome was noted in 8.3% of cases on the 7th day of the development of the pathological process.

Infected pancreatic necrosis was characterised by the manifestation of all signs of the systemic inflammatory reaction syndrome in 16.7% of cases already on the 3rd day of modelling the pathological process. At the same time, the 7–14th day of the course of the pathological

process was manifested by the predominance of 3 and 4 clinical and laboratory signs of the systemic inflammatory response syndrome of the body.

In exactly half of the animals with the experimental model of pancreatogenic sepsis, the presence of all four clinical and laboratory signs of the syndrome of the systemic inflammatory response of the organism was ascertained already on the 1st day of the development of the pathological process, which remained the leading organism in the subsequent periods of the experiments.

CONCLUSION

Modelling of infected pancreatic necrosis complicated by sepsis, manifested by all clinical and laboratory signs of the systemic inflammatory response syndrome of the body, is reliably ahead of all other models of pancreatitis. This once again confirms the phenomenon of the significance of changes in the general reaction of the macroorganism, leading to a rapid generalisation of the inflammatory process and, as a result, the manifestation of all the signs of the systemic inflammatory response syndrome of the body.

Conflict of Interest – None

Ethical aspect – the article is reviewed, and the information presented has a cited reference to primary sources.

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SEPSIS BILAN ASROQLANGAN PANKREONEKROZNING YANGI EKSPERMENTAL MODELINI TAQLID QILISH USULI.

Abdurahmonov F.M.
Toshkent tibbiyot akademiyasi

Abstrakt

Hozirgi vaqtda o'tkir pankreatitga qaraganda, uning patogenezida qorin bo'shlig'i organlarining yanada murakkab yallig'lanish kasalligini topish qiyin. So'nggi 50 yil ichida o'tkir pankreatit qorin bo'shlig'i organlarining o'tkir jarrohlik kasalliklari orasida uchinchi o'rinda turadi va barcha shoshilinch patologiyalarning taxminan 12,5% ni tashkil qiladi. Shu bilan birga, oshqozon osti bezi nekrozining diagnostikasi va jarrohlik taktikasi bizning davrimizda shoshilinch qorin bo'shlig'i jarrohligida hal qilinmagan muammolardan biri bo'lib qolmoqda. Hech shubha yo'qki, bu muammo o'tkir pankreatitning halokatli shakllarini prognoz qilish va erta tashxislash qiyinchiliklari bilan bog'liq. Sepsisning har qanday variantining rivojlanish mexanizmini zamonaviy tushunishda tizimli yallig'lanishli javob sindromining rivojlanishi bilan bog'liq jarayon birinchi o'rinda turadi. Shu bilan birga, tanadagi yiringli fokusning mavjudligi va undan ham ko'proq kamida bitta muhim organning organ disfunktsiyasi mavjud bo'lsa, jarrohlik sepsis hukmining to'liq taqdimotiga mos keladi. Ushbu mulohazalarga asoslanib, tadqiqotimiz natijalarini aks ettiruvchi ushbu maqolaning asosiy maqsadlari sepsis bilan asoratlangan pankreatik nekrozning eksperimental modelini ishlab chiqishdan iborat bo'lib, unda qo'zg'atuvchi omil tananing umumiy reaktsiyasi bo'ladi. klinik amaliyot.

Kalit so'zlar: pankreatik nekroz, pankreatogen sepsis, eksperimental modellashtirish

СПОСОБ МОДЕЛИРОВАНИЯ НОВОЙ ЭКСПЕРИМЕНТАЛЬНОЙ МОДЕЛИ ПАНКРЕОНЕКРОЗА, ОСЛОЖНЕННОГО СЕПСИСОМ

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Абстракт

В наше время трудно найти в своем патогенезе более сложное воспалительное заболевание органов брюшной полости, чем острый панкреатит. На протяжении последних 50-летия острый панкреатит занимает третье место среди острых хирургических заболеваний органов брюшной полости и составляет около 12,5% от всей ургентной патологии. В то же время, диагностика и хирургическая тактика при панкреонекрозе – остаются в наше время в совокупности одной из далеко не решенной проблемой в ургентной абдоминальной хирургии. Несомненным является факт взаимосвязи этой проблемы со сложностями прогнозирования и ранней диагностики деструктивных форм острого панкреатита. В современном представлении механизма развития любого варианта сепсиса на первое место выдвигается процесс, связанный с развитием синдрома системной воспалительной реакции. Вместе с этим наличие гнойного очага в организме, а тем более при наличии органной дисфункции хотя бы одного жизненно-важного органа, соответствуют полному представлению вердикта хирургического сепсиса. Именно исходя из этих соображений, основными задачами данной статьи, отражающего результаты нашего исследования, были разработки экспериментальной модели панкреонекроза, осложненного сепсисом, пусковым механизмом в котором будет играть общая реакция организма, имеющая место в клинической практике.

Ключевые слова: панкреонекроз, панкреатогенный сепсис, экспериментальное моделирование