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New Experimental Model of Pancreonecrosis Complicated with Sepsis

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Abstract

Background. Acute destructive pancreatitis remains one of the far from solved problems of modern urgent surgery. High mortality after pancreatic necrosis, especially when a generalized form of the inflammatory process develops, given the very high mortality rate, is today considered one of the most tragic situations in terms of the outcome of the disease. That is why any experimental research in the field of studying the pathogenesis of pancreatic necrosis complicated by sepsis today is considered relevant.

Metods. In order to model the optimal course of pancreatic necrosis complicated by sepsis, we studied 5 series of experiments on animals.

Results. As our studies and review of the issues discussed on this issue have shown, modeling of acute pancreatic necrosis, which could be complicated by sepsis, remains a far from solved problem of modern experimental surgery. When modeling acute pancreatic necrosis complicated by sepsis, along with the use of all pathogenetically significant factors, a separate link is required, which can radically affect the course of the general reactivity of the organism. Modeling of acute pancreatic necrosis complicated by sepsis is possible only under the condition of a combined approach of both local and general impact.

Keywords: pancreatic necrosis, pancreatogenic sepsis, experimental modeling, systemic inflammatory response syndrome, multiple organ dysfunction

INTRODUCTION

In the modern view of the mechanism of development of any variant of sepsis, the process associated with the development of the systemic inflammatory response syndrome is put forward 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. Based on these considerations, the main objectives of our study were to develop an experimental model of pancreatic necrosis complicated by sepsis, the trigger mechanism in which will be played by the general reaction of the body that takes place in clinical practice. [1]

The clinical picture of the systemic inflammatory response syndrome is a common severe complication in patients with pancreatic necrosis. This prompted clinicians to distinguish a separate form from the group of abdominal sepsis as pancreatogenic sepsis. This step characterizes the peculiarity of the development of pancreatogenic sepsis, which, unlike abdominal, begins with an aseptic process. Only if an infectious agent is attached, pancreatogenic sepsis acquires a complete picture, corresponding to abdominal sepsis as it is. [2]

The holding of the Chicago Sepsis Consensus Conference back in 1991 provided insight into the incidence of sepsis in the future. At the conference, it was decided to separate all forms of generalized inflammatory reactions 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. [3]

For a long time, studies have been conducted on the modeling of pancreatitis and pancreatic necrosis. Meanwhile, experimental modeling of pancreatic necrosis in the standard version of its reproduction does not always allow forming the course of the disease in the form of a systemic inflammatory response syndrome or sepsis syndrome, as the starting phase of the development of pancreatogenic sepsis. [8,9,11,12,13,16]

The most popular option for modeling pancreatitis and pancreatic necrosis in experimental animals are methods with isolated ligation of the Wirsung duct of the pancreas. The reproducibility of pancreatitis starts from 3 days after the intervention. However, any process occurring in animals with a similar experimental model does not characterize the development of pancreatogenic sepsis. In other words, the very characteristic clinical manifestations of infection generalization that determine the symptom complex of any surgical sepsis are absent (body temperature above 38 ºC or below 36 ºC, tachycardia over 90 beats/min, tachypnea over 20 breaths per 1 min, leukocyte count over $12x10^9$/l or below $4x10^9$/l, or the number of immature forms exceeds 10%). In the development of sepsis syndrome, the presence of a purulent focus of infection and possible bacteremia are characteristic. All the above characterizes the main phases of the development of sepsis. It is this approach that should determine the process of the course of pancreatogenic sepsis, close to clinical conditions. To create conditions that meet these conditions, we carried out a number of experimental simulations of pancreatogenic sepsis.

The aim of our study was to develop an optimal experimental model of acute infected pancreatic necrosis complicated by sepsis.

MATERIALS AND METHODS

At the first stage, it was required to determine the dose of the administered microbial agent. To do this, when modeling pancreatogenic sepsis, animals on the 3rd day after ligation of the Wirsung duct were injected into the pancreas after repeated laparotomy with 1 ml of 0.9% sodium chloride solution containing a virulent microbial culture of Escherichia Coli at a dose of 20–30 million microbial bodies per 1 grams of animal weight. The choice of the dose of administered microbial bodies is since when animals were injected with relatively small doses (up to 20 million microbial bodies per 1 gram of animal weight), pancreatogenic sepsis did not develop in 5 out of 7 cases, and the inflammatory process regressed (series 1). An inflammatory process of a non-infectious nature was detected in the abdominal cavity, and an adhesive process formed at the injection site.

Animals had a picture of non-infected pancreatic necrosis. Only in 2 (28.6%) animals in this series of experiments, the autopsy revealed the presence of a limited purulent-inflammatory process in the form of an abdominal abscess. We did not receive a generalization of the inflammatory reaction.

At the same time, in the second series of experiments (series 2), the dose of microbial bodies in the injectable suspension was over 30 million microbial bodies per 1 gram of animal weight. In this series of experiments, 6 animals out of 7 died within 24 hours after injection. Autopsy revealed signs of the development of infectious-toxic shock (putrefaction in the abdominal cavity, hemorrhagic effusion in the abdominal cavity and pleural cavities, acute plethora of internal organs, multiple foci of hemorrhage in the visceral and parietal layers of the peritoneum), which, apparently, was associated with massive influx of microbial bodies into the body. That is, despite the development of the inflammatory process, the latter was of a formal nature, since, in essence, 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 administered, a lightning-fast course of the inflammatory process occurs without any stages of manifestation of pancreatogenic sepsis that occurs 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 area of the pancreas did not differ in any way from the other area of the abdominal cavity, and the lesion proceeded without the formation of a necrobiotic process, and even more so peripancreatic necrosis.

Thus, at this stage of the studies, 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 a 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 appropriate changes without an adequate response of the body. On the other hand, the use of monoculture in the modeling of pancreatogenic sepsis does not correspond to the clinical conditions for the onset of the disease, since in life practice in the occurrence of any purulent-inflammatory process, the prevailing condition is the presence of a polyinfection. The latter is the main argument in favor of using a suspension obtained from the autocalls of the animals themselves as a microbial agent.

To select the dose of administered autocalls animals, we carried out a number of microbiological studies. It was found that the microbial contamination of the autocalls of animals obtained from the cavity of the rectum was represented by many 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 Gram-negatives, Veillonella, Enterobacteriales, Klebsiella, Proteus were predominant. In

72% of cases, rod-shaped pathogens were detected and in 28% - coccus. Of the entire microbial landscape of the animal's feces, 68.6% were excited groups of obligate anaerobes, and 31.4% - facultative anaerobes.

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

The choice of the concentration of the microbial suspension of the animal autocalum was confirmed by us in the next 3 and 4 series of experiments. With the introduction of a solution with an amount exceeding $x10^5$ CFU/ml, a bacterial shock also developed. Whereas, with the introduction of 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 variant that was necessary for the formation of pancreatogenic sepsis. In this connection, we decided to stimulate the necrobiotic process with 10% calcium chloride solutions. Accordingly, the choice of the timing of the introduction of both 10% calcium chloride and the autocalls of animals should determine the timing of the formation of the main process.

RESULTS

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

- with the introduction of large doses of microbial suspensions 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 death and a fulminant course of the pathological process, which does not allow the model to be used for experimental studies;
- septic shock that occurs with the introduction of large doses of autocalls animals, along with a high percentage of early mortality, proceeds without the formation of purulent pancreatic necrosis, and accordingly excludes the phases of the formation of all parts of pancreatogenic sepsis;
- the pancreas with the introduction of large doses of autocalls animals acts as an entrance gate for microorganisms and the formation of a remaining 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 characterize the subsequent reaction of the animal organism, that is, changes in the reactivity of the macroorganism are required, subject to a decrease in the virulence of the microorganism.

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 can be pancreatic autolysis. Only if an infectious agent is attached is it possible to form a classic infected pancreatic necrosis with the subsequent development of sepsis syndrome and severe sepsis. A series of experiments with a low concentration of administered animal autocalls can serve as proof of this judgment. The resulting hypoergic reaction forms a picture of the 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 autocalls will increase the reproducibility of the required pathological process and the experimental model. In this case, the destructive process in the pancreas can, as is known, be modeled using a 10% solution of calcium chloride.

To change the reactivity of the macroorganism, we used antilympholin-Kr, which is an immunosuppressive drug. It is obtained from the blood proteins of rabbits immunized with human thymus lymphocytes. 1 dose of the drug corresponds to 40-60 mg of protein. Along with this, as already indicated above, the animal's autocalum 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.

In order to confirm our judgments, we conducted a new series of experiments (series 5), in which modeling of pancreatogenic sepsis was carried out by preliminary, two-day intraperitoneal administration of antilympholin-Kr at a dose of 0.03 mg per 100 grams of animal (Fig.).

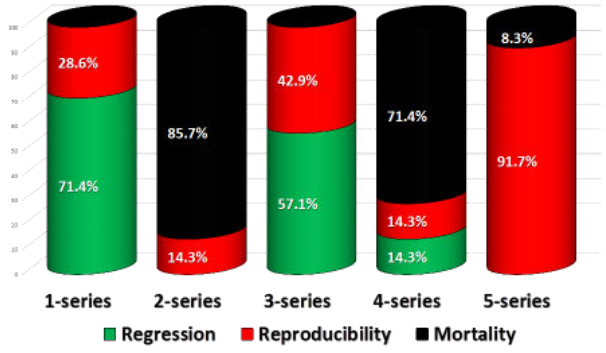

Figure. Comparative assessment of the reproducibility of different models of pancreatic necrosis complicated by sepsis

On the 3rd day of modeling, a laparotomy was performed, the stomach, duodenum together with the pancreas were removed into the wound, and the

Wirsung duct was tied up. After the formation of acute pancreatitis, which usually occurred on the 3rd day of the operation, the abdominal cavity was re-opened and, under aseptic conditions, in order 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 autocalls was injected into the pancreas through the laparotomic wound. In dynamics, starting from the first day after the injection of microbial fecal suspension, the development of pancreatogenic sepsis against the background of pancreatic necrosis was observed.

Over the next 7 days, animals developed a progressive clinical picture of all forms of pancreatic sepsis with signs of systemic inflammatory reaction syndrome (tachycardia, tachypnea, hyperthermia, leukocytosis). The results of blood culture in 100% of cases revealed the presence of hemoculture already on the 3-4th day of modeling.

The pancreas at all times of the experiments was in a purulent-necrotic state. The purulent-destructive process easily spread to nearby tissues, liver gates, mesenteric root. Such results of modeling of pancreatic sepsis took place in 11 (91.7%) of the 12 rats of this series, 1 rat died on the 1st day of observation with pathomorphological signs of bacterial shock, identified by us at autopsy. In our proposed model of pancreatic necrosis complicated by sepsis, the initial signs of a systemic inflammatory reaction syndrome (in the form of respiratory failure, an increase in rectal body temperature, tachycardia, leukocytosis or leukopenia) are observed for 10-12 hours of the experiment.

DISCUSSION

Nowadays, it is difficult to find in its pathogenesis a more complex inflammatory disease of the abdominal organs than acute pancreatitis. Over the past 50 years, acute pancreatitis ranks third among acute surgical diseases of the abdominal organs and accounts for about 12.5% of the total urgent pathology. [4,17] At the same time, the diagnosis and surgical tactics of pancreatic necrosis remain in our time one of the far from solved problems in urgent abdominal surgery. The undoubted fact is the relationship of this problem with the difficulties of forecasting and early diagnosis of destructive forms of acute pancreatitis.

Also, the relevance of the problem is due to the frequency of acute pancreatitis in most patients (65-70%) in the working age. At the same time, in the case of the development of pancreatic necrosis and the use of surgical methods of treatment, disability is noted in more than half of patients - from 62.8 to 75.3% of cases. All this gives the problem the same socio-economic significance. [15]

The mechanism of pathogenesis of acute pancreatitis is multifaceted. And even though 80–90% of acute pancreatitis manifests itself in the form of mild inflammation with a low number of deaths [6], severe forms of this disease, with a progressive syndrome of systemic inflammatory response and pancreatic necrosis, are potentially fatal and form the basis of deaths. [5] At the same time, the basis of deaths in infected forms of pancreatic necrosis is formed by cases of sepsis and organ failure. For example, the overall statistics of deaths in pancreatic necrosis is 3.9-26%, and with infected pancreatic necrosis - up to 85%, with a fulminant course of the disease - 100%. [14]

Difficulties in choosing therapeutic and diagnostic tactics in acute pancreatitis are due to the multivector features of the course of this disease. The issues of choosing diagnostic methods and methods of treatment for uncomplicated and complicated, with severe and mild pancreatitis, with the so-called "edematous" pancreatitis and pancreatic necrosis, with complications of pancreatic toxemia and with destructive complications, with sterile and infected pancreatic necrosis, with early infection and with late destructive complications are discussed.

At the same time, dissimilar and often opposing opinions are expressed on the same issue. [7,10]

CONCLUSION

As our research and review of the issues discussed on this issue have shown, the modeling of acute pancreatic necrosis, which could be complicated by sepsis, remains a far from solved problem of modern experimental surgery.

When modeling acute pancreatic necrosis, complicated by sepsis along with the use of all pathogenetically significant factors, a separate link is required that can radically affect the course of the overall reactivity of the body.

Modeling of acute pancreatic necrosis complicated by sepsis is possible only under the condition of a combined approach of exposure of both local and general nature.

Ethical clearance - All experimental studies were re- viewed, discussed, and approved by the bioethical committee of the Ministry of Health of the Republic of Uzbekistan and fully complied with the terms of the 1986 Council of Europe Convention for the Protection of Animals.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open publication.

Availability of data and material - Available Competing interests - No.

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SEPSIS BILAN ASORATLANGAN PANKREONEKROZNING YANGI EKSPERIMENTAL MODELI

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Abstrakt

Dolzarbligi. O'tkir destruktiv pankreatit zamonaviy shoshilinch jarrohlikning hal qilingan muammolaridan uzoq bo'lib qolmoqda. Pankreas nekrozidan keyin yuqori o'lim, ayniqsa yallig'lanish jarayonining umumiylashgan shakli, o'lim darajasining juda yuqoriligini hisobga olgan holda rivojlanganda, bugungi kunda kasallikning natijasi bo'yicha eng fojiali holatlardan biri sanaladi. Shuning uchun ham bugungi kunga qadar sepsis bilan asoratlangan oshqozon osti bezi nekrozining patogenezini o'rganish sohasidagi har qanday eksperimental tadqiqotlar dolzarb sanaladi.

Tadqiqod. Sepsis bilan asoratlangan pankreas nekrozining optimal variantini simulyatsiya qilish uchun hayvonlarga oid 5 qator tajribalarni o'rganib chiqdik.

Natijalar. Bu masalada muhokama qilingan masalalarni o'rganishimiz va ko'rib chiqishimiz shuni ko'rsatdiki, sepsis bilan asoratlanishi mumkin bo'lgan o'tkir pankreatik nekrozni modellashtirish zamonaviy eksperimental operatsiyaning hal qilingan muammosidan uzoqligicha qolmoqda. Barcha patogenetik ahamiyatga ega omillarni qo'llash bilan birga sepsis bilan asoratlangan o'tkir pankreatik nekrozni modellashtirishda, organizmning umumiy reaktivligi yo'nalishiga tubdan ta'sir ko'rsatishi mumkin bo'lgan alohida bog'lanish talab qilinadi. O'tkir pankreatik nekroz modellashtirish, barcha patogenetik ahamiyatga ega omillarni qo'llash bilan bir qatorda, organizmning umumiy reaktivligi yo'nalishiga tubdan ta'sir ko'rsatishi mumkin bo'lgan alohida bog'lanish talab etiladi. O'tkir o'tkir pankreatik nekrozni modellashtirish, barcha patogenetik jihatdan ahamiyatli omillarni qo'llash bilan bir qatorda, sepsis bilan asoratlangan pankreanekroz faqat mahalliy va umumiy ta'sir birlashgan yondashuv sharoitida bo'lishi mumkin.

Kalit so'zlar: oshqozon osti bezi nekrozi, pankreatogen sepsis, eksperimental modellashtirish, tizimli yallig'lanish reaktsiyasi sindromi, ko'p organli disfunktsiya

НОВАЯ ЭКСПЕРИМЕНТАЛЬНАЯ МОДЕЛЬ ПАНКРЕОНЕКРОЗА, ОСЛОЖНЕННОГО СЕПСИСОМ

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Абстракт

Актуальность. Острый деструктивный панкреатит остается одной из далеко не решенных проблем современной ургентной хирургии. Высокая смертность после панкреонекроза, в особенности, когда развивается генерализованная форма воспалительного процесса, с учетом весьма высокой частоты летальности, считается на сегодняшний день одной из самых трагических ситуаций по исходу заболевания. Вот почему любые экспериментальные изыскания в области изучения патогенеза панкреонекроза, осложненного сепсисом на сегодняшний день, считаются актуальными.

Материал. С целью моделирования оптимального варианта течения панкреонекроза, осложненного сепсисом, нами исследованы 5 серий опытов на животных.

Результаты. Как показали наши исследования и обзор обсуждаемых по данной проблеме вопросов, моделирование острого панкреонекроза, который мог бы быть осложнен сепсисом, остается далеко не решенной проблемой современной экспериментальной хирургии. При моделировании острого панкреонекроза, осложненного сепсисом наравне с применением всех патогенетически значимых факторов, требуется отдельное звено, которое коренным образом может влиять на течение общей реактивности организма. Моделирование острого панкреонекроза, осложненного сепсисом возможно лишь при условии комбинированного подхода воздействия как локального, так и общего характера.

Ключевые слова: панкреонекроз, панкреатогенный сепсис, экспериментальное моделирование, синдром системной воспалительной реакции, полиорганная дисфункция