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# About the Complexity and Mortality Among Patients with Pancreatic Necrosis

B.Z. Khamdamov[1], T.Y. Musoev, F.N. Khaydarov, A.A. Ganiyev, K.Kh. Boboev, F.M. Abdrakhmanov, D.N. Korikhonov, Sh. A. Khamdamov, Sh.A. Bobokulova

**ABSTRACT**

**Background.** Diagnostics and surgical tactics for pancreatic necrosis remain in our time in aggregate one of the far-from-solved problems in urgent abdominal surgery. There is no doubt that this problem is related to the difficulties of predicting and early diagnosis of destructive forms of acute pancreatitis.

**Material.** A retrospective cohort study of the results of a comprehensive examination and treatment of 97 patients with pancreatic necrosis was conducted. All patients were treated and examined in the Bukhara regional branch of the Republican Scientific and Practical Medical Center for Emergency Medical Care from 2013 to 2017.

**Conclusion.** The high mortality rate among patients who have undergone repeated relaparotomy indicates that it is necessary to delay their implementation in infected pancreatic necrosis. They are acceptable when they are performed in conditions of delimitation of processes. Delimited necrosis of the pancreas is lysed and sequestered. In such cases, the sanitizing and draining goal of surgery is easily achievable. This is what allows you to perform the surgical intervention in a more than favourable background, as it will be less traumatic.

**Keywords:** Pancreatic necrosis, pancreatogenic sepsis, systemic inflammatory response syndrome, severe sepsis, septic shock, mortality

## INTRODUCTION

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 all urgent pathology [14].

Also, the urgency of the problem is due to the incidence of acute pancreatitis in many patients (65–70%) at 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 observed in more than half of patients - from 62.8 to 75.3% of cases. All this gives the problem the same socio-economic significance [17].

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 [13], severe forms of this disease, with progressive systemic inflammatory response syndrome and pancreatic necrosis, are potentially fatal and form the basis of deaths [10]. At the same time, the basis of deaths in infected forms of pancreatic necro-

[1] Professor, Doctor of Medical Sciences, Head of the Department of Faculty and Hospital Surgery with Urology Course of Bukhara State Medical Institute, Bukhara, Uzbekistan

sis is formed by cases of sepsis and organ failure. For example, the total statistics of deaths in pancreatic necrosis is 3.9–26%, and in infected pancreatic necrosis – up to 85%, in the fulminant course of the disease – 100% [17].

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

At the same time, dissimilar and often opposing opinions are expressed on the same issue [14].

In the treatment of pancreatic necrosis complicated by sepsis, many attempts are made to study the effect of conservative therapy on the course of the disease. Particular attention should be paid to the work on the effect of anti-cytokine therapy in pancreatic necrosis to prevent the development of sepsis [10].

In particular, it is noted that the use of anti-mediator drugs and antimetabolites is the most promising direction in the correction of inflammatory pathological processes. Of great interest is also the study of possible applications of a new group of drugs (monoclonal antibodies) that can provoke inflammation [9].

The use in the complex therapy of acute necrotizing pancreatitis with drugs that have a normalizing effect on microcirculation and tissue perfusion has reduced the severity of systemic manifestations of acute destructive pancreatitis. By reducing the frequency of development, severity and duration of multiple organ failure and the incidence of complications, there was a decrease in mortality and a decrease in the length of stay of patients in the hospital [6].

Based on this information, it can be concluded that solving the problem of acute pancreatitis, complicated by sepsis, not only surgically, but also having the knowledge to assess the effectiveness of conservative therapy on a targeted basis, will reduce the incidence of complications and deaths in this disease. A critical analysis of the effectiveness of various traditional methods of treating pancreatic necrosis complicated by sepsis is required. This will allow us to find out the main reasons for the unsatisfactory results of treatment of this contingent of patients.

Particular attention should be paid to the study of the pathogenetic mechanisms of the development of pancreatogenic sepsis on an experimental basis [16]. However, in the literature, the information available on this issue is contradictory, sometimes even contradictory to each other. Difficulties are associated with the choice of the optimal method of the experimental model of pancreatic necrosis complicated by sepsis, which should have all stages of the course of this disease [21].

It is known that endothelial dysfunction is the basis of multiple organ failure. Endothelial dysfunction is one of the critical pathophysiological disorders in patients with severe acute pancreatitis [11]. There are research results on the role of predictors of endothelial origin in the development of organ dysfunction in acute pancreatitis [18]. It has been proven that treatment with lexipafant reduces the severity of pancreatitis-associated endothelial barrier compromise, which is also associated with a decrease in systemic concentrations of interleukin, in particular IL-1 [27].

The presented results imply that platelet-activating factors may play an important role in the pathogenesis of pancreatic endothelial dysfunction by signaling and triggering the production and release of certain cytokines. However, the revealed changes in the endothelial system were not confirmed from the point of view of changes in the microcirculatory picture of the pancreas in the dynamics of the progression of pancreatic necrosis complicated by sepsis. This, in turn, would make it possible to objectify the criteria for early diagnosis and prediction of the development of infected forms of pancreatic necrosis and pancreatogenic sepsis, to determine the indications and timing of surgical interventions for pancreatic necrosis complicated by sepsis, to develop a therapeutic and diagnostic algorithm for pancreatic necrosis complicated by sepsis, considering the identified disorders.

## MATERIAL AND METHODS

A retrospective cohort study of the results of a comprehensive examination and treatment of 97 patients with pancreatic necrosis was carried out. All patients were treated and examined in the Bukhara regional branch of the Republican Scientific and Practical Medical Center for Emergency Medical Care from 2013 to 2017.

In 38 (39.18%) cases, pancreatic necrosis was pregnant, and in the remaining 59 (60.82%) cases it was infected. At the same time, among patients with infected pancreatic necrosis, in 20.62% of cases, infected pancreatic necrosis occurred against the background of severe sepsis, and in 4.12% of cases – septic shock. Thus, the

patients were divided into 3 subgroups: subgroup I – patients with sterile pancreatic necrosis (38 patients; 39.18%), subgroup II – patients with infected pancreatic necrosis (35 patients; 36.08%), subgroup III – patients with infected pancreatic necrosis complicated by sepsis (24 patients; 24.74%).

There were 62.9% of male patients and 37.1% of female patients. The base contingent was accounted for by patients of the most able-bodied mature age.

All diagnostic and therapeutic measures in patients with pancreatic necrosis were carried out, in accordance with the approved standards, by the Ministry of Health of the Republic of Uzbekistan.

The basis for the diagnosis of acute pancreatitis was a typical clinical picture of the disease, an increase in amylase in the blood above normal values by 3 or more times and the presence of characteristic signs of changes in the pancreas during the ultrasound. Diagnostic signs of pancreatic necrosis were clinical signs of acute pancreatitis, the presence of skin symptoms (flushing of the face, "marble" shade of the skin, etc.) and the presence of peritoneal symptoms. Hemodynamic disorders, the presence of encephalopathy and laboratory blood tests indicating the presence of organ dysfunction (hyperglobulinemia, leukocytosis, hyperglycemia, increased urea in the blood, etc.) were evaluated.

Patients with pancreatic necrosis were always hospitalized in intensive care and intensive care units and within the first 2 hours from the moment of admission to the clinic, the following mandatory diagnostic measures were carried out:

Clinical: collection of complaints and anamnesis, general examination of the patient, general thermometry, anthropometry, palpation, auscultation, percussion of the abdomen, counting the heart rate, determining the level of blood pressure. It should be noted that for a dynamic assessment of the severity of the course of the disease, the starting position was the time of onset of pain and not the time of hospitalization.

laboratory: general blood test (determination of haemoglobin level, number of erythrocytes and leukocytes, hematocrit level, calculation of leukocyte formula, erythrocyte sedimentation rate, determination of platelet count); determination of blood group and Rh factor; biochemical blood test: determination of bilirubin, urea, total protein, aspartate aminotransferase, alanine aminotransferase, α-amylase, electrolytes (K +, Ca +, Na+, Cl-); determination of blood glucose levels; general urinalysis; determination of the concentration of α-amylase in the urine; coagulogram study: determination of partially activated thromboplastin time, prothrombin index, fibrinogen concentration in plasma.

instrumental: electrocardiogram; survey radiography or fluoroscopy of the abdominal cavity; survey radiography or fluoroscopy of the chest; ultrasound examination of the abdominal organs, esophagogastroduodenofibroscopy.

According to individual indications, the following were carried out: to assess structural changes in the pancreas - computed tomography; laparoscopy; puncture of the pleural cavity; in case of contraindications to computed tomography and suspected biliary pancreatic necrosis - magnetic resonance imaging. Computed tomography was performed no earlier than 72 hours after the onset of the disease with mandatory contrast enhancement.

To assess the severity of patients, generalization of the inflammatory process and organ dysfunction, the protocol for examining patients included the generally recognized diagnostic scale Multiple Organ Dysfunction Score (MODS), determination of C-reactive protein and procalcitonite in blood plasma.

The patients underwent a complex of conservative therapy, which includes measures aimed at relief of pain syndrome (analgin, baralgin, ketorol, no-spa); suppression of secretory activity of the stomach (octreotide, quamatel, dalargin, aspiration of gastric contents through a nasogastric tube); detoxification therapy; correction of microvasculature disorders; antibiotic therapy; normalization of metabolic disorders; replenishment of protein-energy balance.

The volume of infusion therapy consisted of crystalloid and colloidal solutions in a ratio of 4:1. Correction of the volume of infusion therapy was carried out depending on the level of central venous pressure (10 cm of water), the state of diuresis (at least 40-60 ml/h), the state of the acid-base state and the level of electrolytes in the blood. Correction of microcirculatory disorders was carried out by infusion of low molecular weight dextran.

Carbapenems, cephalosporins, and fluoroquinolones were used as empirical antibiotic therapy. The combination of antibacterial drugs was supplemented with metronidazole or its derivatives. In the future, according to the results of the bacteriogram, appropriate corrections were made in the appointments. Starting from the 5th day of antibiotic therapy, antimycotic therapy (fluconazole and its analogues) was added to the course of therapeutic measures.

Enteral nutrition through the probe was carried out only after relief of gastrostasis, intestinal paresis and

with a decrease in the level of α-amylase to a level of less than 3 values of the norm.

All drugs were administered intravenously. However, in patients with severe acute pancreatic necrosis, an intra-arterial route of drug administration was used.

Surgical treatment of patients with acute pancreatic necrosis in phase I of the disease, even with the use of minimally invasive technologies, was performed according to strict medical indications by the decision of the council.

The presence of severe pain, rapidly progressive jaundice, signs of biliary hypertension according to ultrasound and the absence of bile in the duodenum during esophagogastroduodenofibroscopy, indicated the presence of a wedged stone of the large duodenal papilla. Such patients urgently, that is, within 6-12 hours from the moment of hospitalization in the clinic, needed measures aimed at restoring the passage of bile and pancreatic juice into the duodenum.

Laparoscopic surgical interventions in patients with pancreatic necrosis in phase I of the disease were performed by individual decision after a complete clinical, laboratory and instrumental examination, in the presence of positive symptoms of peritonitis, with confirmation by ultrasound of the presence of fluid in the abdominal cavity, and, if necessary, differential diagnosis of acute pancreatitis with other diseases of the abdominal organs.

The main signs of pancreatic necrosis during the laparoscopic intervention were:

1. the presence of effusion in the abdominal cavity with high activity of amylase in it;
2. the presence of foci of steatonecrosis on the surface of the peritoneum;
3. the presence of edema in the root of the mesentery of the transverse colon and/or retroperitoneal space;
4. hemorrhagic impregnation of the large omentum.

In such cases, laparoscopic intervention was completed by removal of peritoneal exudate and drainage of the abdominal cavity.

The presence of small acute fluid or necrotic accumulations in the abdominal cavity or in the omentum bag, in the absence of severe pain syndrome or in an extremely serious condition of the patient, their microdrainage was targeted, followed by replacement of drainage with a larger diameter.

With a relatively satisfactory condition of the patient, in the absence of pain, signs of compression of neighbouring organs, mechanical jaundice, acute intestinal obstruction, infection and others, surgical interventions were not performed. Such formations were usually subjected to involution under the influence of enhanced conservative therapy. However, in the absence of a tendency to decrease against the background of conservative therapy, as well as with complicated and progressive acute fluid / necrotic accumulations, they were drained using the above technique or using video assistance.

In the phase of melting and sequestration of pancreatic necrosis or in the presence of a purulent complication, the choice of treatment method, of course, was surgical. The main purpose of this operation was to perform necrsequestrectomy, opening, drainage and sanitation of foci of destruction both in the abdominal cavity and in the retroperitoneal space. Operations were carried out both in one stage and in several stages.

Laparotomy was performed by the upper-median approach. After the revision of the abdominal cavity, the omentum bag was opened, two-lumen drains were installed in a multidirectional position and taken to opposite zones using contra-aperture approaches. In this position, the drainage channels do not fall off. To prolong the full functioning of the drains, a wide sheet of glove rubber was laid between them. Drainage channels were formed according to the size, shape, and location of pancreatic necrosis.

With the biliary origin of pancreatic necrosis, such operations were combined with operations on the gallbladder and biliary tract (cholecystectomy, drainage of the common bile duct, etc.).

With the development of bleeding from the pancreas, preference was given to X-ray endovascular methods of hemostasis, in the absence of such an opportunity, an open operation was performed.

Assessment of the condition of a patient with acute infected pancreatic necrosis complicated by sepsis was used by the gradation scale developed by us, consisting of the following criteria: dynamics and frequency of development of multiple organ dysfunction syndrome; frequency and severity of purulent complications in the overall structure of therapeutic measures.

The patient's condition was assessed as satisfactory, provided that the heart rate adjusted for pressure ≤10; the number of platelets in the blood >120x$10^3$/l; the level of bilirubin in the blood ≤20 mmol/l; the level of creatinine in the blood ≤100 mmol/l; there are no digestive fistulas and arrosive bleeding; there are no purulent complications. On computed tomography, focal or diffuse increase in size and no accumulation of contrast in ≤30% of the pancreatic parenchyma is determined.

The patient's condition was assessed as moderate, provided that the heart rate adjusted for pressure ≤15; the

number of platelets in the blood >100x10$^3$/l; the level of bilirubin in the blood ≤30 mmol/l; the level of creatinine in the blood ≤150 mmol/l; there are digestive fistulas that are amenable to conservative treatment and suppuration of the wound, which does not require repeated surgery. Computed tomography determines the presence of inflammatory changes in the pancreas and peripancreatic adipose tissue, as well as the non-accumulation of contrast in 30-50% of the pancreatic parenchyma, single weakly delimited peripancreatic accumulation of fluid.

The patient's condition was assessed as severe, provided that the heart rate adjusted for pressure ≤20; the number of platelets in the blood >70x10$^3$/l; the level of bilirubin in the blood ≤60 mmol/l; the level of creatinine in the blood ≤201 mmol/l; there are digestive fistulas requiring surgical intervention and arrosive bleeding amenable to conservative treatment; there is retroperitoneal phlegmon; diffuse serous, hemorrhagic or fibrinous peritonitis. On computed tomography, it is determined and not the accumulation of contrast in >50% of the pancreatic parenchyma.

The patient's condition was assessed as extremely severe, provided that the heart rate adjusted for pressure ≤30; the number of platelets in the blood >30x10$^3$/l; the level of bilirubin in the blood ≤80 mmol/l; the level of creatinine in the blood ≤300 mmol/l; there are digestive fistulas and/or arrosive bleeding requiring surgical intervention; there is retroperitoneal phlegmon; diffuse purulent peritonitis. Computed tomography determines fluid accumulations and the complete absence of accumulation of contrast in the pancreatic parenchyma, even with bolus administration.

The data obtained in 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 aggregation functions and BioStat for Windows. The methods of variational parametric and nonparametric statistics were used. The level of significance $p<0.05$ was taken as statistically significant changes.

## RESULTS

The alcoholic etiology of the disease accounted for 28.9% of patients. In 43 (44.3%) patients, the cause of pancreatic necrosis was cholelithiasis. The gastrogenic origin of the disease was detected in 25 (25.8%) patients. In 1 patient (1.03%), the etiological cause of pancreatic necrosis could not be determined.

The treatment of patients in the clinic since the onset of the disease was not unambiguous but differed in a certain pattern among the subgroups. In the group of patients, active surgical tactics were predominantly used, consisting in conducting early laparotomies, despite the phase (sterile or infected) of the pathological process and the form (small-focal, large-focal, subtotal, total) of necrotic lesions. The main indications for surgical operations were the negative dynamics of the disease, despite the ongoing conservative therapy and/or the presence of signs of peritonitis.

In most cases, pancreatic necrosis proceeded with various complications, which were often combined and thus could manifest themselves in several variants and the same patient. It is they who determine the urgency of surgical intervention and tactics for pancreatic necrosis.

Peritonitis was detected in 49 (55.5%) patients. Local peritonitis was diagnosed in 16.3% of patients, diffuse peritonitis in 22.4% and diffuse peritonitis in 61.2% of patients. The serous nature of the effusion was noted in 18.4% of patients, hemorrhagic in 32.6%, purulent in 14.3% and fibrinous-purulent in 34.7% of patients.

Of course, it should be borne in mind that peritonitis in patients was an indication for surgical intervention. However, given the fact that with the serous or hemorrhagic nature of the exudate, minimally invasive interventions would be very effective, nevertheless, at that time of medical care, the main surgical technique remained a more complex and traumatic laparotomy. And laparoscopy was performed only for diagnostic purposes and verification of the final diagnosis of the disease. At the same time, it was half of the deceased patients who had peritonitis. And here it should be stated that peritonitis was one of the frequent complications of pancreatic necrosis.

Postretroperitoneal phlegmon was in 2nd place (34.0%) after peritonitis. This type of complication of pancreatic necrosis was detected at different times from the onset of the disease, on average, 7.2±3.8 days. The lesion of the right half of the retroperitoneal space was noted by us in 24.2% of patients, the left half in 63.6% of patients and total in 12.1% of patients. At the same time, in 28.9% of patients, lesions of the retroperitoneal space were determined only by infiltration without signs of suppuration at the time of intraoperative verification. However, with repeated surgery, 11.3% of patients were diagnosed with pancreatic abscesses.

The nature of the complications characterizing the generalization of the purulent-inflammatory process in patients with different phases of pancreatic necrosis was not ordinary (Table 1).

**Table-1**
**The nature of the frequency of registration of generalization of the inflammatory process.**

| TYPE OF PROCESS | PANCREATIC NECROSIS | | | |
|---|---|---|---|---|
| | Sterile | | Infected | |
| | n | % | n | % |
| Systemic inflammatory response syndrome | 15 | 100 | 0 | 0 |
| Sepsis syndrome | 0 | 0 | 35 | 59,3 |
| Severe sepsis | 0 | 0 | 20 | 33,9 |
| Septic shock | 0 | 0 | 4 | 6,8 |

Pancreatogenic sepsis in patients with a sterile phase of pancreatic necrosis (subgroup I) was diagnosed in 39.5% of cases. Whereas among patients with the infected phase of pancreatic necrosis, it was diagnosed in 100% of cases. I would like to remind you that the III subgroup of patients was formed at the expense of patients with severe sepsis and septic shock. Accordingly, patients of subgroup II, who also had a generalized form of complication of the purulent-destructive process, were represented by the presence of sepsis syndrome or only by the syndrome of a systemic inflammatory response of the body.

In 74 (76.3%) patients, pancreatogenic sepsis was diagnosed on the day of hospitalization. Of these, almost half of the patients (47.3%) were diagnosed with sepsis syndrome, represented by patients with an infected phase of pancreatic necrosis. Patients who formed the III study subgroup (with the presence of severe sepsis and septic shock), who accounted for 32.4%, had an exclusively infected form of pancreatic necrosis. At the same time, if patients with severe sepsis were 27.0%, then patients with septic shock were 5.4%.

Despite the ranking of patients with systemic inflammatory response syndrome by the presence of organ dysfunction or septic shock, nevertheless, patients with sepsis syndrome on the day of hospitalization in the clinic turned out to be quite an impressive number (59.3%).

Systemic inflammatory response syndrome was diagnosed in the absence of a purulent focus and organ dysfunction in 20.3% of patients, and all of them were presented by patients with a sterile phase of pancreatic necrosis (Table 2).

The distribution of patients depending on the number of signs of systemic inflammatory response syndrome showed that there were 4 clinical and laboratory signs (27.8%). Only one patient was inferior to the number of patients with 3 clinical and laboratory signs. There were 21.6% of patients with 2 clinical and laboratory signs, and 23.7% of patients with 1 clinical and laboratory sign. It was the latter category of the nature of the manifestation of systemic inflammatory reaction syndrome that was represented in more than half of patients with a sterile phase of pancreatic necrosis (60.5%).

It should be noted that the nature of the change in the curve of the numerical value of patients with systemic inflammatory reaction syndrome among patients with the sterile phase of pancreatic necrosis had an inverse correlation with the number of patients (R = -0.847). In other words, as the number of clinical and laboratory signs of systemic inflammatory response syndrome increased, there was a progressive decrease in the number of patients with pancreatic necrosis in the sterile phase. At the same time, among patients with the infected phase of pancreatic necrosis, the correlation significance between the number of clinical and laboratory signs of systemic inflammatory response syndrome and the number of patients had a close direct correlation (R = 0.954).

**Table-2**
**Distribution of patients by the number of signs of systemic inflammatory response syndrome**

| NUMBER OF SIGNS OF SYSTEMIC INFLAMMATORY RESPONSE SYNDROME | PANCREATIC NECROSIS | | | |
|---|---|---|---|---|
| | Sterile | | Infected | |
| | n | % | n | % |
| $SIRS_{0-1}$ | 23 | 60,5 | 0 | 0 |
| $SIRS_2$ | 7 | 18,4 | 14 | 23,7 |
| $SIRS_3$ | 5 | 13,2 | 21 | 35,6 |
| $SIRS_4$ | 3 | 7,9 | 24 | 40,7 |

According to the records of the medical history, the presence of pancreatogenic sepsis was recorded among patients 405 times. On average, each patient accounted for 5.5 times. Such a high value of sepsis registration was probably due to the amount imposed on deceased patients. However, when subtracting deaths among patients with pancreatogenic sepsis, this figure decreased

by only 0.4 times. The ratio between patients with a sterile phase of pancreatic necrosis and with an infected one was 2.1 times in favour of the latter.

Mortality among patients with acute pancreatic necrosis was 30.9% (30 cases) and was distributed as follows in the dynamics of treatment.

On the day of admission, 1 (3.3%) patient died, by the end of the first day - another 1 (3.3%) patient. Subsequently, 2 (6.7%) patients died on 2-3 days of treatment, 7 (23.3%) patients died on days 3-7, 10 (33.3%) patients died on days 7-14 and another 9 (29.9%) patients died in the long term (over 14 days and up to 3 months). In general, 83.3% of patients died in the subgroup with acute infected pancreatic necrosis, and the remaining 16.7% with acute sterile pancreatic necrosis.

Thus, the first 14 days turned out to be the most dangerous, during which 21 (70.1%) out of 30 patients died. When comparing the total amount of complications for various systems in the victims, it was found that, on average, there were 5.6-6.5 complications for each death on days 1-7, and 4.9-6.6 complications associated with organ dysfunction on days 7-14. Of these, 4.5 – 5.8 complications on days 1-7 of admission of patients were associated with impaired activity of vital organs.

The combination of severe disorders of vital organs occurred in almost all patients who died in the first 3 days of observation - 3.6-3.8 such disorders per 1 patient, and later this figure decreased slightly.

The decrease in the frequency of septic shock in the first 3 days is fully consistent with the number of dead patients, which is associated with the initial severity of admitted patients. Starting from 3 days, there was a persistence of cases of septic shock due to the deterioration of the condition of patients and inadequate treatment with the development of complications (primarily purulent septic).

The most common causes of death among patients with pancreatic necrosis were generalization of infection (70.9%), peritonitis (59.6%), cardiovascular disorders (58.3%) and hepatic-renal disorders (45.4%) and other complications. Arrosive bleeding was less common (6.7%). Such a high percentage of multiple organ "interest" prompted us to retrospectively study pathomorphological changes in patients with a comparative analysis of lethal outcomes.

In 30 (30.9%) of the deceased, the data of pathomorphological examination of organs were studied. At the same time, it was found that fatty and granular degeneration of liver cells was observed in 29 (96.7%) cases, cirrhosis of the liver – 9 (30.0%), amyloidosis – in 4 (13.3%). Kidney changes in 23 (76.7%) of the deceased were characterized by granular dystrophy of the epithelium of the convoluted tubules and in 2 (6.7%) – by amyloidosis. Pathological changes in the heart muscle were found in 25 (83.3%) patients (no changes were found in 16.7%). These changes were characterized by granular dystrophy, combined in three patients with fragmentation of muscle fibers.

The revealed changes were characterized by granular dystrophy of internal organs, combined in one patient with damage to several organs at once and the development of multiple organ failures. Let's illustrate the observations.

Patient R.G., born in 1967, was transferred from the district branch of our regional center on 23.02.2015 with a diagnosis: "Acute infected pancreatic necrosis. Condition after laparotomy and drainage of the omentum bag. Phlegmon of the retroperitoneal space. Sepsis". From the anamnesis: acutely ill after eating fatty foods and alcohol, for which he was treated in one of the private clinics from 06.02.2015 Due to the deterioration of the condition and the appearance of peritoneal phenomena, the patient was transferred to the emergency surgical department of the central district hospital, where on 08.02.2015 the patient underwent laparotomy, cholecystectomy, drainage of the omentum bag and abdominal cavity. Active conservative therapy was started, which included active antibacterial and detoxification therapy. In dynamics, the patient's condition did not improve, the temperature reaction varied at the level of 38-39$^0$C, purulent-putrefactive discharge increased along the drainage. On 15.02.2015, on the control computed tomography of the abdominal organs, the patient was diagnosed with the presence of ongoing peritonitis and phlegmon of the retroperitoneal space. On 18.02.2015, relaparotomy and autopsy of the retroperitoneal phlegmon were performed. The patient's condition did not improve. Due to the failure of treatment, the patient was transferred to us. In the department: complaints about the presence of a wound with purulent discharge and pain around the drainage, hyperthermia up to 39$^0$C, chills, shortness of breath, palpitations, weakness, and dry mouth. Objectively: the patient's condition is severe, and the skin and visible mucous membranes are pale. The sclera is jaundiced. There is sweating of the body and blush of the cheeks. Locally: on the anterior and left lateral surfaces of the abdominal wall there are silicone drains with scanty purulent discharge. The laparotomy wound is supplemented with incisions and has a purulent-putrefactive discharge. Around drains up to 15 cm in diameter, the colour of the skin is not changed, however, its compaction, sharp soreness, swelling and crepitus of soft tissues are determined. When pressing on the area of swelling, purulent-putrefactive discharge with a fetid odour comes from the wound. Bacterioscopy of wound exudate revealed the presence of gram-positive cocci in the amount of 50 EPZ, and gram-negative bacilli in the amount of 59 EPZ. On multispiral computed tomography, complete destruction of the pancreas and cavity formations in the retroperitoneal space of various diameters. Immediately, in the lateral projection, drainage ectopic from the gland bag is determined. In blood tests: haemoglobin - 87 g/l, erythrocytes - 3.6x10$^{12}$/l, total protein - 52 g/l, urea - 14.2 mmol/l, creatinine - 125.3 mmol/l, alanine aminotransferase - 0.9 mmol/l.h, aspartate aminotransferase - 1.1 mmol/l.h, bilirubin - 36.1 mmol/l, amylase - 22.3 mmol/l. The diagnosis was established: "Acute infected pancreatic

necrosis. Condition after relaparotomy and drainage of the retroperitoneal space. Anaerobic phlegmon of the retroperitoneal space and anterior abdominal wall of the abdomen. Peritonitis. Severe sepsis. Multiple organ dysfunction". On 24.02.2015, under general intubation anesthesia, the following operation was performed: relaparotomy, revision of the omentum and abdominal cavity, necrsequestrectomy of the pancreas, extended opening, and drainage of phlegmon of the retroperitoneal space. The revision revealed the spread of the purulent-necrotic process to the right and left behind the colonic animal spaces with necrotic melting of tissues and purulent leakage of the omentum bursa to the right and left iliac regions. A laparostomy was imposed. In dynamics, the patient's condition remained extremely serious. 27.02.2015 Daily diuresis decreased to 20 ml/h. There was an increase in the liver + 5 cm, yellowness of the skin. In the analyzes haemoglobin – 90 g/l, erythrocytes – 3.2 x $10^{12}$/l, total protein – 48 g/l, urea – 31.2 mmol/l, creatinine – 175.3 mmol/l, alanine aminotransferase – 1.5 mmol/l.h, aspartate aminotransferase – 1.8 mmol/l.h, amylase – 32.1 mmol/l, bilirubin 56.1 mmol/l. The patient continued to receive intensive therapy in the intensive care unit with hemosorption sessions. Despite intensive therapy, including extracorporeal detoxification methods, the patient's condition progressively worsened, hemodynamics was unstable, rested solely on vasopressor drugs, and on 03.03.2015 cardiac arrest occurred. Resuscitation measures were unsuccessful. Postmortem diagnosis: "Acute infected total pancreatic necrosis. Condition after rere-laparotomy, necrsequestrectomy and drainage of a phlegmon of the retroperitoneal space and omentum bag. Ongoing purulent peritonitis. Septic shock. Multiple organ failure syndrome: acute renal-hepatic, cardiovascular and respiratory failure. A postmortem examination revealed purulent-necrotic destruction of the pancreas, necrotic melting of the parietal peritoneum posteriorly, phlegmon of the retroperitoneal space, granular dystrophy of the liver and convoluted tubules of the kidneys. Enlargement of the spleen and enlargement of the ventricles of the heart. Septic spleen and septic endocarditis.

As this case showed, an untimely diagnosis of pancreatitis led to the development of sterile pancreatic necrosis. At the same time, unjustified surgical intervention in the early stages of the development of the disease and inadequate drainage of the omentum bag, followed by ectopia of drainage, its untimely determination ended with the progression of infected pancreatic necrosis, the development of retroperitoneal phlegmon and sepsis, the progression of which, as well as multiple organ failure, led to death.

## DISCUSSION

A report on the registration of the presence or absence of pancreatogenic sepsis at the time of hospitalization of patients revealed that pancreatic necrosis in the infected phase is characterized by the predominance of sepsis without dysfunction of vital organs (more than half of the patients). At the same time, among patients with the sterile phase of pancreatic necrosis, this type of inflammatory complication was manifested only in 1/3 of patients without dysfunction of vital organs. However, in the dynamics of the treatment the picture regarding the manifestation of the development of the generalization of the inflammatory process has changed radically.

Unfortunately, even intensive complex treatment using active surgical tactics was not able to provide positive treatment results. At the same time, maintaining a high level of complication of pancreatic necrosis is accompanied by a violation of the natural mechanisms of the regressive course of the purulent-necrotic process. An example is the high level of septic complications identified by us because of a retrospective analysis of patients with pancreatic necrosis [15].

As mentioned above, underestimation of the severity of the patient's condition, even with the use of modern diagnostic methods, as well as the use of screening methods in the assessment of septic complications, contributed to the progression of the purulent-inflammatory process with the involvement of more and more new areas of the pancreas and more and more intensive involvement of vital organs in the process. At the same time, the initial state of the body becomes important. In the presence of concomitant diseases of internal organs (diabetes mellitus, cirrhosis of the liver, etc.), this process is inevitable. The basis for such an assumption can be the given numerical data [27].

The main causes of mortality in the midst of the disease are disorders of vital organs. In subsequent terms, along with mortality due to the initially serious condition of patients, liver disorders begin to come to the fore, while maintaining a high frequency of generalization of infection. An increase in the high frequency of purulent-septic complications in the midst of the disease, especially their most severe types – severe sepsis and septic shock, leads to changes in thanatogenesis with the dominance of this group of complications [16].

A very significant circumstance in the cause of death of patients is the frequent combination of disorders of the activity of 2-3 or more vital organs and systems (multiple organ failure syndrome), especially pronounced during the height of the disease [26].

The presented data, on the one hand, confirm the well-known scheme of the course of acute pancreatic necrosis: the inflammatory process in the pancreas with the enzymatic breakdown of the parenchyma leads to the development of systemic inflammatory reaction syndrome, which is a response of the macroorganism to the ongoing process of endotoxicosis. In patients with infected pancreatic necrosis, this process is aggravated due to bacterial invasion and uncontrolled cytokine discharge

into the systemic circulation. All this leads to a progressive disruption of the microvasculature, which, under conditions of enzymatic destruction of the pancreas, may take the form of the initial stage of a vicious circle, the exit from which becomes impossible by performing active surgical intervention in the early stages of the disease [18].

Infection in the pancreas and breakthrough of the capillary barrier leads, together with toxins and microbes, to an increase in the functional load on vital organs with the development of their multiple organ failure and subsequent generalization of the pathological process with the development of septic shock [25].

On the other hand, a certain relationship between the pathomorphological structural transformations of the pancreas and vital organs in the midst of the disease may indicate an increasing endothelial interdependence between these organs in this disease. This pathological process is known today as endothelial dysfunction [19].

At first glance, it is enough to understand that the endothelial system functionally regulates not only local processes, but also systemic ones. Unfortunately, this is not enough to prove the mandatory presence of such relationships in patients with sterile and infected forms of pancreatic necrosis. It is required to conduct targeted additional studies with an accurate assessment of the state of the endothelial system under normal conditions, and then in the dynamics of the development of pancreatic necrosis. Moreover, an important aspect in such a study is the assessment of the endothelial system in various variants of inflammatory lesions of the pancreas (acute pancreatitis, sterile pancreatic necrosis, infected pancreatic necrosis and infected pancreatic necrosis complicated by sepsis).

And from the internal organs, the liver underwent the most profound changes, which, as is known, is exposed to purulent intoxication to a greater extent than other organs [1].

As shown by the presented clinical cases, the performance of early laparotomies against the background of ineffective conservative therapy, in most cases, does not allow to solve the problem in a positive way. In such cases, pancreatic necrosis did not stop, which, accordingly, ended with the progression of sepsis, multiple organ failure and death. In such cases, the operation acquired a very traumatic character, aggravating the already critical condition of the patient. This indicates the need for a radical revision of active surgical tactics in pancreatic necrosis, especially in its sterile form. We also believe that it is advisable to introduce new minimally invasive technologies, since the use of traditional methods of surgical treatment of pancreatic necrosis against the background of a serious condition of patients initially predetermines high mortality. However, questions related to objective criteria for choosing a method of surgical intervention or conservative treatment should not be based only on morphostructural changes in the pancreas [28].

## CONCLUSION

The high mortality rate among patients who have undergone repeated relaparotomy indicates that it is necessary to delay their implementation in infected pancreatic necrosis. They are acceptable when they are performed in conditions of delimitation of processes. Delimited necrosis of the pancreas is lysed and sequestered. In such cases, the sanitizing and draining goal of surgery is easily achievable. This is what allows you to perform surgical intervention in more than  favorable background, as it will be less traumatic.

**Ethics approval and consent to participate** - All patients gave written informed consent to participate in the study.

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

**Availability of data and material** – Available.

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## PANCREONEKROZI KASALLIKLARIDA ASORAT VA ULIM SABABLARI

**Xamdamov B.Z., Musoev T.Yu., Xaydarov F.N., Ganiev A.A., Boboev Q.X., Abduraxmanov F.M., Korixonov D.N., Xamdamov Sh.A., Bobokulova Sh.A.**
**Buxoro davlat tibbiyot instituti**
**Respublika shoshilinch tibbiy yordam ilmiy markazi Buxoro mintaqaviy filiali**
**Toshkent tibbiyot academiyasi**

### ABSTRAKT

**Dozarbligi.** Oshqozon nekrozini diagnostikasi va jarrohlik taktikasi shoshilinch qorincha operatsiyasida hal qilingan muammolardan uzoq bo'lgan agregatda bizning davrimizda qolmoqda. Hech shubha yo'qki, bu muammo o'tkir pankreatitning destruktiv shakllarini bashorat qilish va erta tashxislash qiyinchiliklari bilan bog'liq.

**Material.** Oshqozon nekroziga chalingan 97 nafar bemorni kompleks tekshirish va davolash natijalarini retrospektiv kohort o'rganish o'tkazildi. 2013-2017 yillarda Respublika shoshilinch tibbiy yordam ilmiy-amaliy tibbiyot markazi Buxoro viloyat filialida barcha bemorlar davolanib, tekshirildi.

**Xulosa.** Takroriyrelaparotomiyadan o‘tgan bemorlar orasida o‘lim darajasi yuqori bo‘lishi kasallangan oshqozon nekrozida ularning amalga oshirilishini kechiktirish zarurligini ko‘rsatadi. jarayonlarni delimitatsiya sharoitida amalga oshirilganda ma'qul bo'ladi. Oshqozonning delimidli nekrozi lizlanadi va ketma-ket bo'ladi. Bunday hollarda operatsiyaning sanitariya va drenaj maqsadiga osonlik bilan erishish mumkin. Bu narsadan ko'ra ko'proq miqdorda jarrohlik aralashuvini amalga oshirish imkonini beradi ijobiy fon, chunki bu kamroq travmatik bo'ladi.

**Tayanch iboralar:** Pankreatik nekroz, pankreatogen sepsis, tizimli yallig'lanishga javob berish sindromi, og'ir sepsis, septik shok, o'lim

## ОСЛОЖНЕНИЯ И ЛЕТАЛЬНОСТЬ ПРИ ПАНКРЕОНЕКРОЗЕ

**Хамдамов Б.З., Мусоев Т.Ю., Хайдаров Ф.Н., Ганиев А.А., Бобоев К.Х., Абдурахманов Ф.М., Корихонов Д.Н., Хамдамов Ш.А., Бобокулова Ш.А.**
**Бухарский государственный медицинский институт**
**Бухарский областной филиал Республиканского центра экстренной медицинской помощи**
**Ташкентская Медицинская Академия**

### АБСТРАКТ

**Актуальность.** 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.

**Материал.** Проведен ретроспективное когортное исследование результатов комплексного обследования и лечения 97 больных с панкреонекрозом. Все больные находились на лечении и обследовании в Бухарском областном филиале Республиканского научно-практического медицинского центра экстренной медицинской помощи с 2013 по 2017 годы.

**Заключение.** Высокая летальность среди больных, у которых были выполнены неоднократные релапаротомии, свидетельствует о том, что необходимо отсрочивать их выполнении при инфицированном панкреонекрозе. Они приемлемы при их выполнении в условиях отграничения процессов. Отграниченный некроз поджелудочной железы лизируется и секвестрируется. В таких случаях санирующая и дренирующая цель хирургического вмешательства легко достижимы. Именно этом позволяет выполнять хирургическое вмешательство в более благоприятном фоне, так как будет менее травматичным.

**Ключевые слова:** Панкреонекроз, панкреатогенный сепсис, синдром системной воспалительной ответной реакции, тяжелый сепсис, септический шок, летальность