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Dynamics of Clinical, Laboratory, and Morphostructural Parameters in Patients with Infected Pancreatic Necrosis Associated with Diabetes Mellitus

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Динамика клинико-лабораторных и морфоструктурных показателей у больных с инфицированным панкреанекрозом на фоне сахарного диабета

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Abstract: In this article, the authors analyze the dynamics of clinical, laboratory, and morphostructural parameters in patients with infected pancreatic necrosis (IPN) associated with diabetes mellitus (DM). Clinical studies were conducted in 62 patients with IPN and concomitant DM. The significance of clinical signs, laboratory parameters, instrumental diagnostic methods, and morphostructural indicators in patients with IPN associated with DM was evaluated. Overall, the authors conclude that a comprehensive assessment of clinical, laboratory, and morphostructural changes in patients with IPN and concomitant DM is accompanied by an increasing systemic inflammatory response, pronounced organ dysfunction, and progression of destructive foci during the first 1-3 days of the disease, followed by partial stabilization of the patients' condition by the 10th day as a result of ongoing comprehensive treatment. The study demonstrated that conventional therapeutic approaches do not ensure timely prevention of disease progression in patients with IPN associated with DM.
Keywords: infected pancreatic necrosis, diabetes mellitus, clinical, laboratory, and morphostructural parameters.
Резюме: В статье авторы анализируют динамику клинико-лабораторных и морфоструктурных показателей у больных с инфицированным панкреанекрозом (ИПН) на фоне сахарного диабета (СД). Клинические исследования проводились у 62 больных с ИПН на фоне СД. Оценивается значимость клинических признаков, лабораторных показателей, инструментальных методов диагностики, а также морфоструктурных показателей у больных с ИПН на фоне СД. В целом, авторы делают итоги о том, что комплексная оценка клинических, лабораторных и морфо- структурных изменений у больных с ИПН на фоне СД сопровождаются нарастанием системной воспалительной реакции, выраженной органной дисфункцией и прогрессированием очагов деструкции в 1-3 суток заболевания и несколькой стабилизацией состояния к 10-дню в результате проводимого комплексного лечения. Исследования показали, что традиционные подходы не обеспечивают своевременное предотвращение прогрессирования процесса у больных с ИПН на фоне СД.
Ключевые слова: инфицированный панкреанекроз, сахарный диабет, клинические, лабораторные и морфоструктурные показатели.

Currentity. Acute pancreatitis (AP) is currently one of the most common acute surgical diseases of the abdominal organs [1, 7]. Since the last decade of the 20th century, pancreatologists around the world have established working groups several times (1992, 2012, 2020), attempting to reach consensus on the terminology, classification, diagnostic algorithms, and treatment of various variants of OP - this severe,

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"multifaceted," and often unpredictable disease [2,8,11]. The 2012 revision of the classification adopted in Atlanta (USA, 1992) allowed for consensus on many issues of diagnosis and treatment (definition of pancreatic necrosis, identification of two forms of severe OP—severe and moderate—division of the disease course into two phases, etc.). Severe acute pancreatitis (hereinafter referred to as severe acute pancreatitis) is diagnosed when the primary disease is accompanied by multi-organ failure lasting more than 48 hours [3,12,13]. OP develops in 15-20% of patients with OP of various etiologies [5,6]. Currently, thanks to the rapid development of new technologies, the large-scale introduction into clinical practice of various variants of minimally invasive percutaneous, retroperitoneal, and transmural treatment (surgical and endoscopic) techniques (and their combinations), as well as the development of a "stage-by-stage" or "enhancing" interdisciplinary approach to treating TOP, in a significant number of cases, clinical recovery is achieved without "open" surgical interventions [4,]. Nevertheless, in 10–30% of cases (according to various authors), open surgical interventions for OP remain relevant [10]. Severe acute pancreatitis, in accordance with both the international classification revised in 2012 [9], and the domestic classification adopted within the framework of national clinical recommendations for the diagnosis and treatment of acute pancreatitis [2], is a form of acute pancreatitis accompanied by persistent (48 hours or more) organ failure. OP - a severe, "multifaceted" clinical picture with diverse pathomorphological manifestations and often unpredictable disease - develops in 15-20% of patients with OP of various etiologies [5], and mortality in this disease, which remains high over the last few decades, ranges from 17 to 42%; the addition of purulent-destructive complications increases this figure to 46-80% [10]. Currently, various examination methods allow a significant portion of patients to promptly diagnose PID, more or less accurately predict its course, and monitor the dynamics of the inflammatory process and disease outcomes [14].

The relevance of the problem is confirmed by the complexity of each specific case, where standard approaches are impossible, and also requires the use of the latest technologies in diagnosis and treatment.

The aim of the study was: To establish the pathogenetic relationships between clinical-laboratory, morphostructural, and metabolic indicators that determine the intensity of the pancreatic necrobiotic process in infected pancreatic necrosis in patients with diabetes mellitus.

RESEARCH MATERIALS AND METHODS

The clinical material of the study was formed based on observations of 62 patients with IPN against the background of diabetes mellitus who underwent treatment from 2012 to 2018. The study was conducted at the Andijan branch of the Republican Scientific Center for Emergency Medical Care of the Ministry of Health of the Republic of Uzbekistan.

In the structure of the examined patients, a moderate predominance of men was noted in all studied groups (Table 1).

In patients with IPN associated with diabetes mellitus, men accounted for 62.9% and women for 37.1% of the total number of observations. The age structure of the patients was characterized by a predominance of middle-aged and elderly patients. The largest share falls on the age intervals of 45-59 and 60-74 years. The proportion of young patients aged 18-44 remained relatively low and did not exceed 15-18%. Elderly individuals aged 75-89 years accounted for 7 (11.3%) patients.

Table 1

Characteristics of patient distribution by gender and age (WHO classification)

GENDER / AGE	NUMBER OF PATIENTS
	(n=62)
Men, n (%)	39 (62.9%)
Women, n (%)	23 (37.1%)
18-44 years, n (%)	10 (16.1%)
45-59 years, n (%)	21 (33.9%)
60-74 years, n (%)	24 (38.7%)
75-89 years, n (%)	7 (11.3%)

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In the structure of diabetes in patients with IPN, type 2 diabetes predominated, which was diagnosed in the vast majority of patients. The etiological structure in patients with IPN against the background of diabetes mellitus is shown in Table 2. In patients with IPN against the background of diabetes mellitus, biliary stone disease was the leading etiological factor, observed in 26 (41.9%) patients. The dietary factor fluctuated within the range of 20-23%. The contribution of metabolic causes, including hypertriglyceridemia, deserves special attention, which was significantly more frequent in patients with IPN against the background of diabetes mellitus, accounting for 11.3%. The alcohol factor, on the contrary, occupied a secondary place in the overall structure of IPI etiology and was equal to (3.2%).

Table 2

Etiological structure of infected pancreatic necrosis in the examined patients

EFFECTIVE FACTOR	Number of patients (n=62)
Gallstone disease, n (%)	26 (41.9%)
Nutritional factor, n (%)	14 (22.6%)
Hypertriglyceridemia and metabolic causes, n (%)	7 (11.3%)
Alcohol factor, n (%)	2 (3.2%)
Post-manipulative IPN, n (%)	6 (9.7%)
Traumatic injuries to the pancreas, n (%)	4 (6.5%)
Idiopathic and other causes, n (%)	3 (4.8%)

Post-manipulative and traumatic causes were found in 9.7% and 6.5%, respectively. The clinical presentation in most observations included signs of a severe course of the disease, such as severe pain, intoxication, organ dysfunction, and the need for stage-by-stage surgical interventions. General clinical laboratory research methods, biochemical, and hemostasis system status were conducted; to assess the severity of endotoxemia, systemic inflammatory reaction (SIR), and tissue hypoperfusion, the determination of biochemical and immunological blood markers was additionally performed. Instrumental research methods were used to confirm the diagnosis of IPI, to clarify the extent and prevalence of damage to the pancreas, its retro- and parapancreatic tissues, and to dynamically assess the course of the pathological process. Primary instrumental examination included abdominal ultrasound, esophagogastroduodenoscopy, and multispiral computed tomography (MSCT), which were used in various combinations depending on the clinical situation. Magnetic resonance imaging (MRI) was used as indicated, primarily in cases of diagnostic difficulties or the need to clarify the nature of changes.

MRI was primarily used to clarify the nature of fluid formations, their degree of organization, and to differentiate inflammatory and necrotic changes in complex diagnostic cases. The obtained data were analyzed in conjunction with MSCT and ultrasound results.

The comprehensive application of these instrumental methods ensured an objective assessment of the depth and prevalence of IPI, allowed for monitoring the dynamics of the pathological process, and served as a basis for comparing instrumental data with clinical and laboratory indicators at various stages of treatment.

Results and their discussion.

Dynamic observation of the clinical condition of patients with IPN against the background of diabetes mellitus allowed for the establishment of a sequence of increases and changes in the severity of the systemic inflammatory response (SIR) against the background of standard therapy. Upon admission, the majority of patients exhibited signs of a pronounced inflammatory response. The proportion of patients with ≥2 criteria for systemic inflammatory response syndrome (SIRS) was 64.5%, and nearly 1/3 of patients exhibited 3 or 4

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signs of SIRS (29%). By the 1st day of treatment, the structure of indicators shifted toward further strengthening of the systemic reaction, which was reflected in an increase in the proportion of patients with ≥2 signs of CVSR to 74.2% (46 patients) and an increase in severe CVSR variants to 38.7% (Table 3).
On the 3rd day of treatment, inflammatory activity reached its maximum, with the number of patients with 3 or 4 signs of CVRS increasing to 48.4% (30 cases). This time period coincided with the period of the most frequent appeals from the duty brigade regarding the change of therapeutic tactics. For example, in patient M., 58, by the 3rd day, pronounced tachycardia, high fever, pain syndrome exceeding 7 points on the visual-analog scale (VAS), and signs of respiratory insufficiency persisted, requiring enhanced antibacterial therapy. Observations of this type were noted in a significant portion of patients and were reflected in quantitative data. Temperature indicators were characterized by similar dynamics, and at the time of admission to the clinic, febrile fever ≥38.5°C was identified in 58.1% of patients, and on the 1st day, this indicator increased to 64.5%, with a subsequent increase to 69.4% on the 3rd day of treatment. Only after 5 days did a gradual decrease in the proportion of patients with such a level of hyperthermia occur, indicating a delayed clinical response despite the measures taken. Temperature normalization within the range of ≤37.5°C was observed in only 12.9% of patients upon admission and remained a rare phenomenon until the 5th day. Only by the 10th day did the proportion of patients with normal temperature values reach 45.2%, indicating the gradual entry of a portion of patients into the stabilization phase. Pain syndrome also tended to persist in the early stages, with 67.7% of patients at the time of admission to the clinic evaluating pain as ≥7 points on the VASH scale. On the 1st day, the value remained almost unchanged, remaining above 70%. On the 3rd day, the proportion of patients with pronounced pain began to decrease, but the indicator remained high at 64.5%. The absence of a noticeable reduction in pain compared to the previous day was observed in 74.2% of patients on the 1st day and 80.6% on the 3rd day. This ratio reflected the slow regression of pain syndrome characteristic of IPN, especially in patients with diabetes mellitus. In patient N., 61, despite increased analgesic therapy, the severity of pain remained at 7-8 points for 4 days, which indirectly indicated ongoing inflammatory activity.

Table 3

Dynamics of clinical signs in the intensity of infected pancreatic necrosis in patients with diabetes

INDEX	TREATMENT DYNAMICS (days)					
	Before treatment	1	3.	5	7	10
$SSVR_{≥2}$	40 (64.5%)	46 (74.2%)	51 (82.3%)	48 (77.4%)	39 (62.9%)	28 (45.2%)
$SSVR_{3-4}$	18 (29%)	24 (38.7%)	30 (48.4%)	26 (41.9%)	19 (30.6%)	12 (19.4%)
Body temperature ≥38.5 °C	36 (58.1%)	40 (64.5%)	43 (69.4%)	38 (61.3%)	29 (46.8%)	18 (29%)
Body temperature ≤37.5 °C	8 (12.9%)	6 (9.7%)	5 (8.1%)	9 (14.5%)	16 (25.8%)	28 (45.2%)
Pain syndrome ≥7 points according to VASH	42 (67.7%)	44 (71%)	40 (64.5%)	32 (51.6%)	22 (35.5%)	12 (19.4%)
Absence of pain relief ≥2 points according to VAS	42 (67.7%)	46 (74.2%)	50 (80.6%)	38 (61.3%)	24 (38.7%)	14 (22.6%)
Episodes of hypotension (SAB <90 mm Hg)	12 (19.4%)	15 (24.2%)	19 (30.6%)	18 (29%)	14 (22.6%)	9 (14.5%)
Tachycardia >100 beats/min	34 (54.8%)	38 (61.3%)	41 (66.1%)	36 (58.1%)	27 (43.5%)	16 (25.8%)
Oliguria <0.5 ml/kg/h in the last 6 hours	16 (25.8%)	18 (29%)	21 (33.9%)	20 (32.3%)	15 (24.2%)	10 (16.1%)
Clinical signs of ODN	20 (32.3%)	24 (38.7%)	27 (43.5%)	25 (40.3%)	19 (30.6%)	11 (17.7%)

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Impairment of consciousness	6 (9.7%)	8 (12.9%)	10 (16.1%)	9 (14.5%)	7 (11.3%)	5 (8.1%)
Unsatisfactory clinical response to ABT	34 (54.8%)	40 (64.5%)	44 (71%)	39 (62.9%)	28 (45.2%)	16 (25.8%)

No noticeable reduction in pain compared to the previous day was observed in 74.2% of patients on the 1st day and 80.6% on the 3rd day. This ratio reflected the slow regression of pain syndrome characteristic of IPN, especially in patients with diabetes mellitus. In patient N., 61, despite increasing analgesic therapy, the severity of pain remained at 7-8 points for 4 days, which indirectly indicated ongoing inflammatory activity.

Hemodynamic indicators showed more moderate but stable dynamics, among which hypotension was noted in 19.4% of patients upon admission and reached a maximum on the 3rd day, accounting for 30.6%. By the 5th day, the proportion of hypotensive patients gradually decreased; similarly, the tachycardia rate exceeded 100 bpm in 54.8% of patients upon admission and increased to 66.1% by the 3rd day, which corresponded to the severity of the systemic reaction at this stage.

Perfusion indicators also showed a deterioration in the early stages, among which oliguria was recorded in 1⁄4 of patients as early as the 1st day of treatment and reached 33.9% by the 3rd day, after which it gradually decreased. In several patients, such a decrease in diuresis served as one of the criteria for transfer to the reanimation and intensive care unit (ORIT). For example, in patient S., 63 years old, who was initially stable, a decrease in diuresis to less than 0.5 ml/kg/h was observed by the 3rd day against the background of an increase in inflammatory markers.

Respiratory failure symptoms also showed an increase by the 3rd day, and while they were recorded in 32.3% of patients upon admission, this figure reached 43.5% by the 3rd day. Such changes were often combined with fever, tachycardia, and increased pain syndrome, forming a clinical picture that requires closer observation. At the same time, consciousness disorders occurred less frequently, but they retained a tendency to increase by the 3rd day, reaching 16.1%. The specified sign was usually accompanied by a combination of hemodynamic instability and a pronounced inflammatory response.

Assessment of the response to antibacterial therapy (ABT) in dynamics showed a slow regression of clinical manifestations. The absence of a satisfactory response in the form of persistent pain syndrome and fever was noted in 54.8% of patients upon admission, and on days 1–3, the indicator increased to 64.5% and 71%, respectively. Only by the 10th day did the proportion of such observations decrease to 1⁄4 of patients.

Dynamic observation of laboratory parameters in patients with IPN against the background of diabetes mellitus allowed for an assessment of the depth of IVR, the degree of organ dysfunction, and the influence of hyperglycemia on the course of IPN. At the time of admission to the clinic, the main markers of SVR were at high levels, which corresponded to the pronounced clinical symptoms. Leukocytosis reached 15.2±3.1 x109/L, and the leukocyte index of intoxication (LII) exceeded 5 units. (5.1±1.0 units), indicating an active process of gastric tissue destruction. As early as the 1st day of therapy, a further increase in the intensity of the inflammatory response occurred. Leukocytes increased to 16.3±3.4 x109/L, and the LII reached 5.6±1.1 units, reflecting the continued involvement of systemic mechanisms of inflammatory response (Table 4).

The maximum severity of the SVR laboratory profile occurred on the 3rd day of conservative treatment. During this period, the concentration of C-reactive protein (CRP) increased to 255.1±57.2 mg/l, and the level of procalcitonin (PCT) averaged 5.6±2.1 ng/ml. The obtained values were in good agreement with the clinical picture, in which the highest indicators of hyperthermia, tachycardia, and the number of signs of CVS were observed. For example, in patient P., 60 years old, an increase in PCT to 7.2 ng/ml was recorded on the 3rd day with persistent fever and pronounced pain syndrome, which confirmed the ongoing infection of necrotic masses of the pancreas. Such observations were characteristic of a significant portion of the patients and coincided with the dynamics of laboratory markers of SVR.

Table 4

Dynamics of laboratory indicators in patients with infected pancreatic necrosis during diabetes mellitus

INDEX	TREATMENT DYNAMICS (days)

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	Before treatment	1	3.	5	7	10
Leukocytes, x10^9/L	15.2 ±3.1	16.3 ±3.4	17.5 ±3.6	16.8 ±3.5	14.2 ±3	11.8 ±2.6
Thrombocytes, x10^9/L	260.3 ±70.1	248.5 ±68.2	230.4 ±64.1	224.9 ±62.5	236.8 ±60.3	248.4 ±58.3
Total protein, g/l	60.3 ±5.1	59.5 ±5.6	58.7 ±6.1	57.8 ±6.4	58.9 ±5.6	60.3 ±5.4
Albumin, g/l	30.7 ±4.2	29.5 ±4.1	28.4 ±4.1	27.8 ±4.5	29.9 ±4.3	31.7 ±4.5
Glucose, mmol/L	11.8 ±2.4	12.4 ±2.6	12.8 ±2.8	11.6 ±2.5	10.4 ±2.2	9.6 ±2
Creatinine, µmol/L	112.6 ±28.1	126.5 ±32.7	138.9 ±36.1	132.7 ±34.5	120.6 ±30.9	110.3 ±28.6
Total bilirubin, µmol/L	24.3 ±8.1	28.7 ±10.2	32.4 ±11.3	30.6 ±10.5	26.3 ±9.1	22.9 ±8.7
ALT, IU/L	64.3 ±22.1	70.8 ±14.2	78.7 ±16.4	74.5 ±25.2	66.8 ±23.9	58.4 ±20.7
AST, U/L	58.1 ±20.4	64.5 ±22.8	72.5 ±24.3	68.3 ±23.8	60.1 ±21.7	52.9 ±19.4
IF, U/L	180.1 ±40.5	190.3 ±42.1	205.4 ±45.7	198.5 ±44.8	184.5 ±41.3	170.1 ±38.2
Amilase, IU/L	580.7± 160,2	543.5± 138.7	511.3± 128.4	428.1± 107.5	365.9± 96.4	300,2± 86.7
Lipase, IU/L	780.5± 67.4	740± 190	690± 180	580± 170	480± 150	380.4± 42.8
Lactate, mmol/L	2.4±0.7	2.8±0.8	3.1±0.9	2.9±0.8	2.3±0.7	1.9±0.6
Fibrinogen, g/l	5.4±1.0	5.8±1.1	6.2±1.1	5.9±1	5.1±0.9	4.4±0.8
MNO	1.26± 0.18	1.3± 0.2	1.34± 0.21	1.32± 0.2	1.28± 0.18	1.22± 0.16
LII, units.	5.1±1.1	5.6±1.1	6±1.2	5.7±1.1	4.8±0.9	4±0.8
CRP, mg/l	210.5 ±48.6	230.9 ±52.3	255.1 ±57.2	243.2 ±49.7	190.7 ±45.2	150.6 ±40.4
PCT, ng/ml	4.2±1.6	4.9±1.8	5.6±2.1	5.1±1.9	3.9±1.5	3±1.2
MSM, units.	0.68± 0.14	0.74± 0.15	0.8± 0.16	0.76± 0.15	0.64± 0.13	0.54± 0.12

By the 5th day, most patients experienced moderate stabilization of inflammatory parameters, among which leukocytes decreased from 17.5±3.6 x10^9/L to 16.8±3.5 x10^9/L, CRP decreased to 243.2±49.7 mg/L, and PCT levels began to regress to 5.1±1.9 ng/ml. Despite the gradual improvement, these figures remained high, reflecting stable inflammatory activity in IPN in patients with DM. Only after the 7th and especially the 10th day was a more pronounced decrease in the main markers of inflammation noted, which coincided with a decrease in the frequency of febrile fever and a reduction in pain syndrome.

Carbohydrate metabolism indicators demonstrated their own dynamics, where at the time of patients' admission to the clinic, the average glucose level was 11.8±2.4 mmol/L; on the 1st and 3rd days, an upward trend persisted, reaching 12.8±2.8 mmol/L. Such hyperglycemic resistance was observed in the majority of patients and was often combined with the need to intensify insulin therapy.

Kidney functional indicators also changed in parallel with the inflammatory phase of the disease, with creatinine levels at admission (on day zero) being 112±28 µmol/L and rising to 138±36 µmol/L by day 3, which aligned with the incidence of oliguria episodes presented above. On the 5th and subsequent days, a

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gradual decrease in creatinine levels was noted, reflecting the onset of kidney function restoration in most patients.

Liver functional status indicators in the early stages of patient treatment showed a moderate deterioration, among which total bilirubin increased from 24.3±8.1 to 32.4±11.3 μmol/l by day 3, while AST and ALT showed similar dynamics with a peak by day 3, reflecting the response of the hepatobiliary system to systemic inflammation and decreasing only by day 10. At the same time, PBJ enzymes showed a gradual decrease, specifically amylase decreased from 580.7±160.2 to 300.2±86.7 U/L, and lipase from 780.5±67.4 to 380.4±42.8 U/L by the 10th day. This dynamic characterizes a natural process of decreasing enzymatic activity against the background of gastric tissue destruction and is not a specific diagnostic marker in the late period of the disease in IPN. Fibrinogen reached its maximum on day 3 and then decreased, while lactate increased to 3.1±0.9 mmol/l on day 3, after which it began to decrease. The endotoxemia indicator remained steadily elevated until day 5 and regressed only by day 10. Overall, coagulation indicators and endotoxemia markers demonstrated a characteristic profile.

Overall, the dynamics of laboratory indicators emphasize the two-phase nature of IPN in patients with diabetes mellitus. The first phase was characterized by a pronounced increase in inflammatory markers and organ dysfunction between the 1st and 3rd days of the disease. The second phase manifested as a slower decrease in these indicators, which was observed only after 7 days. Such a structure of changes confirms the stability of the early inflammatory response and the slow formation of a tendency toward clinical improvement, which is important for the subsequent analysis of morphological changes and the justification of the need for a personalized approach.

Dynamic assessment of morphological indicators revealed a pronounced instability in the structural changes of the pancreas in patients with diabetes mellitus during the early period of IPN (Table 5). Upon admission, a significant portion of patients exhibited large areas of destruction in the form of a predominance of patients (54.8%) with a volume of necrotic damage exceeding 50%.

Table 5

Dynamics of changes in the morphostructural changes of the pancreas in patients with infected pancreatic necrosis during diabetes mellitus

INDEX	TREATMENT DYNAMICS (days)					
	Before treatment	**1**	**3.**	**5**	**7**	**10**
Pancreatic necrosis volume >50%	34 (54.8%)	36 (58.1%)	38 (61.3%)	37 (59.7%)	35 (56.5%)	30 (48.4%)
Pancreatic necrosis volume ≤50%	18 (29%)	19 (30.6%)	18 (29%)	17 (27.4%)	16 (25.8%)	14 (22.6%)
Emergence of new destruction zones in dynamics	-	14 (22.6%)	20 (32.3%)	18 (29%)	10 (16.1%)	4 (6.5%)
Absence of focus encapsulation	38 (61.3%)	40 (64.5%)	42 (67.7%)	40 (64.5%)	34 (54.8%)	24 (38.7%)
Partial encapsulation of the focus	8 (12.9%)	10 (16.1%)	12 (19.4%)	14 (22.6%)	18 (29%)	22 (35.5%)
Free fluid in the abdomen	32 (51.6%)	34 (54.8%)	36 (58.1%)	35 (56.5%)	30 (48.4%)	24 (38.7%)
Presence of gas in the IPN area	12 (19.4%)	14 (22.6%)	16 (25.8%)	16 (25.8%)	14 (22.6%)	10 (16.1%)
Thickness of peripancreatic infiltrate ≥10 mm	39 (62.9%)	41 (66.1%)	42 (67.7%)	40 (64.5%)	34 (54.8%)	26 (41.9%)
Retroperitoneal flegmona	20 (32.3%)	22 (35.5%)	24 (38.7%)	23 (37.1%)	19 (30.6%)	14 (22.6%)

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Total defeat	22 (35.5%)	24 (38.7%)	26 (41.9%)	26 (41.9%)	24 (38.7%)	20 (32.3%)
Compression of stomach or intestines	24 (38.7%)	25 (40.3%)	26 (41.9%)	25 (40.3%)	22 (35.5%)	18 (29%)

On the next day, this indicator increased to 58.1%, reaching its maximum by the 3rd day (61.3%). In practice, such an increase most often reflected the appearance of new areas of gastric tissue death. For example, in patient R., 62 years old, upon repeated examination on the 3rd day, the pancreas necrosis zone involving the body of the gland expanded to the head with the formation of additional fluid elements, which corresponded to the most active phase of the disease.

Simultaneously, the dynamics of the emergence of new destruction zones were observed.

On the 1st day, this sign was identified in 22.6% of patients, and by the 3rd day, it was already present in 32.3%. This sequence aligns well with the laboratory inflammation peak noted above. On the 5th day, the proportion of patients with new foci remained high at 29%, after which the frequency of this sign gradually decreased.

The formation of the necrotic focus's capsule demonstrated a reverse pattern, and upon admission, the absence of the capsule was noted in 61.3% of patients. On the 1st and 3rd days, the proportion of such observations increased to 64.5% and 67.7%, respectively, which reflected the slow formation of the focus boundaries in patients with DM. Only by the 7th and 10th days of treatment did a gradual decrease in this indicator indicate the transition of a portion of the foci to a more stable phase. In the early stages, a formed capsule was encountered rarely (no more than 12.9% of patients) upon admission. By the 10th day, this indicator increased to 35.5%, which coincided with a decrease in SVR markers and clinical stabilization in a portion of patients.

The thickness of the peripancreatic infiltrate remained high throughout the entire observation period, with ≥10 mm recorded in 62.9% of patients upon admission, slightly increasing on days 1 and 3 and remaining at about 60% until day 5. Only after the 7th day of treatment was there a trend toward a reduction in infiltrate thickness, which reflected a slow resolution of the perifocal inflammation. In patient T., 66 years old, the infiltrate in the projection of the pancreas body retained a thickness of more than 10 mm during the first 5 days, despite treatment and draining surgical interventions, including necrossectomy.

The presence of free fluid in the abdominal cavity demonstrated stable dynamics; at the time of admission, it was detected in 51.6% of patients, and on the 1st and 3rd days, in 54.8% and 58.1%, respectively. This trend reflected the spread of the inflammatory process to the surrounding tissues. By the 7th and 10th days, the proportion of patients with fluid decreased, which coincided with the gradual recovery of clinical parameters.

An important morphological sign was the presence of gas in the area of gastric necrosis or in the parapancreatic spaces. At the time of admission, this sign was observed in 19.4% of patients and reached its maximum by the 3rd day (25.8%). Such changes often served as a basis for discussing emergency surgical interventions. For example, in patient L., 58 years old, the appearance of gas in the projection of the buttock pouch on the 3rd day of treatment was accompanied by an increase in pain syndrome and an increase in body temperature, which necessitated a transition to open intervention on the 4th day.

Signs of retroperitoneal phlegmon also showed an increase in frequency by the 1st and 3rd days, reaching 38.7%. As inflammatory activity decreased, this sign occurred less frequently, reflecting the gradual limitation of the process.

An increase in the anatomical areas of the pancreas involved was noted in a similar sequence, i.e., if upon admission, damage to all 3 parts (head, body, and tail) was observed in 35.5% of patients, then by the 3rd day, this indicator increased to 41.9%, which aligned with the general trend toward the spread of destruction. Compression of the stomach or transverse colon was recorded in approximately 1/3 of patients at all stages of observation, showing only a moderate decrease by the 10th day of complex treatment.

In total, the sequence of described changes indicates two key features of morphological dynamics in patients with IPN against the background of diabetes mellitus. The first consists of a high frequency of progression of the necrotic process on days 1–3, accompanied by the formation of new foci and the absence of a tendency toward early capsule formation. The second reflects only the gradual appearance of stabilization signs after 7

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days, which aligns with clinical and laboratory data and emphasizes the complexity of the IPN course in this category of patients.

FINDINGS:

1. Infected pancreatic necrosis in patients withacute diabetes is characterized by a more severe and unstable course compared to patients without carbohydrate metabolism disorders, which is manifested by more frequent early progression of the necrobiotic process (58.1%), predominance of extensive forms of pancreatic damage with necrosis volume >50% (54.8%), absence of a formed capsule of the necrotic focus (61.3%), and high mortality during traditional surgical tactics (29%).
2. In patients with infected pancreatic necrosis against the background of sugar diabetes , the maximum severity of the systemic inflammatory response is observed between the 1st and 3rd days, and signs of stabilization become noticeable only after 7 days of treatment. This structure of changes emphasizes the slow onset of clinical improvement and the stability of the early phase of the inflammatory reaction, which creates an unfavorable background for making decisions on management tactics and forms the basis for analyzing laboratory and morphological data.
3. Assessment of clinical, laboratory, and morphostructural changes shows that in patients with infected pancreatic necrosis against a background of ahar, it was characterized by an early and steady increase in systemic inflammatory reaction, pronounced organ dysfunction, and the progression of destruction foci on the 1st–3rd day of the disease. The subsequent decrease in the intensity of inflammatory and morphological indicators occurred slowly and was accompanied by incomplete stabilization of the condition by the 10th day. This dynamic was formed against the backdrop of standard treatment tactics and emphasizes that traditional approaches do not ensure timely prevention of process progression in patients with acute diabetes mellitus, which creates prerequisites for the development of a personalized therapeutic and diagnostic algorithm.

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