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Nutritional support in patients with acute pancreatitis. Review of published studies

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Acute pancreatitis is a common disease that occurs in 5—10 % of patients with urgent pathology of the abdominal cavity. The most prevalent metabolic disorders affecting this group of patients are hypermetabolism and hypercatabolism syndromes, which are accompanied by excessive consumption of carbohydrates, fats, and amino acids, increased oxygen intake, and carbon dioxide production.

Objective — to analyse the current state of the problem of nutritional support for patients with acute pancreatitis.

The degree of nutritional disorders in patients with acute pancreatitis varies depending on the etiological factors and severity of the disease, necessitating a differential approach to their correction. Patients with acute pancreatitis experience disruption of the intestinal microflora due to the antibiotic therapy, nutrient and fiber deficiency, and lack of microbial antagonism. This disruption leads to excessive growth of bacteria, particularly gram-negative microflora. The effectiveness and safety of enteral tube feeding are determined by a complex of factors: the timing of recovery of peristalsis and the absorption function of the intestinal wall, the type of mixture, and the method of its administration. Restoration of intestinal absorption in patients with severe acute pancreatitis occurs on average 48 hours after the start of complex conservative therapy. The use of antiflatulents as part of a mixture for enteral nutrition allows to improve the laboratory indicators of blood serum and reduce the frequency of intestinal complications on the 7th day by 21.5 % (χ^2=4.88, 95 % CI 2.3—39.5, p=0.03). Nasogastric nutritional support in patients with severe acute pancreatitis is safe and leads to a 25.8 % reduction in the incidence of local infectious complications (χ^2=4.59, 95 % CI 2.43—45.53, p=0.03), length of hospital stay by 16 days (p=0.04), and deaths by 21.4 % (χ^2=4.13, 95 % CI 0.81—39.68, p=0.04) in comparison with parenteral nutrition. Nutritional support should be started with nasogastric administration of a food mixture, and in case of complications (intolerance, aspiration, etc.), nasojejunal administration. Parenteral nutrition should be used if enteral nutrition is impossible or not tolerated.

Keywords
acute pancreatitis, nutritional support, enteral nutrition, complications.

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Acute pancreatitis (AP) is a widespread disease that occurs in 5—10 % of patients with urgent pathology of the abdominal cavity [39]. According to the revised classification of AP (Atlanta, 2012), which was proposed by the «Acute Pancreatitis Classification Working Group», three degrees of severity of the course of the disease are distinguished: moderate (mild), moderate and severe, while in the general structure of the disease, severe AP occupies from 10 % to 20 %, accompanied by a high risk of complications (up to 50 %) and deaths (40—70 %) [9, 11]. The most prevalent metabolic disorders affecting patients with AP are hypermetabolism and hypercatabolism syndromes, which are accompanied by excessive consumption of carbohydrates, fats, and amino acids as energy substrates, a significant increase in oxygen intake and carbon dioxide production, and increased nitrogen loss with urine [21]. In turn, the goal of nutritional support in patients with AP is prevention of catabolic processes, correction of negative nitrogen balance, reduction of inflammation, and improvement of treatment results [61]. Despite the publication of a large number of studies devoted to the nutritional support of patients with AP, this topic remains debatable among practicing doctors, the vast majority of whom continue to adhere to the principle of the need for complete restriction of enteral nutrition (EN) (starvation) in this category of patients [13, 62].

Objective — to analyse the current state of the problem of nutritional support for patients with acute pancreatitis.

The review of articles on AP published up to November 2023 in the PubMed, Web of Science, and EMBASE databases was carried out using the specific search terms «acute pancreatitis», «nutritional support», «enteral nutrition», and «complications». Qualitative and quantitative data were obtained through iterative interpretation of each article. This prevented the loss of potentially valuable information.

Pathophysiology of metabolic disorders in patients with acute pancreatitis

The occurrence of hypermetabolism and hypercatabolism syndromes in patients with AP is primarily associated with the release of pro-inflammatory cytokines, which leads to an increase in energy consumption and basic metabolism, which depend on both the severity of the course and the duration of the disease [21, 50]. Thus, in case of sepsis, as a complication of AP, in 80 % of patients there is an increase in protein catabolism and an increase in nutritional needs [47]. In turn, a long-term negative nitrogen balance leads to the deterioration of the course of AP and increases the risk of fatal cases [45].

Focusing on glucose metabolism in patients with AP, it should be noted that it is associated with both increased energy needs and damage to the cells of the islets of Langerhans. As a result of a complex metabolic reaction to the inflammatory process in the pancreas and surrounding tissues, endogenous glyconeogenesis increases [18]. In turn, exogenous glucose is an important source of energy, but it can only partially counteract the increase in glyconeogenesis and the destruction of proteins in response to inflammation. At the same time, the introduction of a large amount of glucose can cause an increase in oxygen consumption and carbon dioxide production and be one of the causes of acute respiratory failure. In addition, the occurrence of hyperglycemia is possible, which deepens metabolic disorders and is an aggravating factor in the risk of infectious complications [16, 34].

According to literature data, with the development of hypermetabolism syndrome in patients with AP, energy consumption can increase by 77—158 % [1]. When calculating nutritional support, it is recommended to take into account the following consumption norms [2]:

- energy consumption in the amount of 25—35 kcal/kg of body weight/day;
- consumption of carbohydrates — 3—6 g/kg of body weight/day (blood serum glucose concentration should not exceed 10 mmol/L);
- protein consumption — from 1.2 to 1.5 g/kg of body weight/day (requires correction in the development of acute kidney or liver failure);
- fat consumption — 2 g/kg of body weight/day (the blood serum triglyceride level should not exceed 12 mmol/L).

Pathogenetic features of the occurrence of purulent-septic complications in patients with acute pancreatitis and their connection with enteral nutrition

Special consideration should be given to the latest studies on the problem of infected necrotic foci and the development of the systemic inflammatory response syndrome (SIRS) in acute aseptic necrotic pancreatitis. Thus, microbial translocation from the lumen of the small intestine is considered to be one of the causes of infection of necrotic foci. This phenomenon was first described by Durwandiring in 1881 [41]. In turn, R. Berg defines translocation as «the passage of viable bacteria and their toxins from the gastrointestinal tract through the mucous membrane to the surrounding tissues of the body (in the mesenteric lymph nodes, circulatory system, liver, and spleen)» [59]. There is also research devoted to the study of microbial translocation from the lumen of the small intestine in the etiopathogenesis of multiple organ failure syndrome (MODS) in critical conditions [29].

The main factors that ensure the barrier properties of the intestinal mucosa include an intact epithelial layer, the presence of lymphocytes, macrophages, and neutrophils produced in «Peyer's plaques» and located in the submucosal layer, and a normally functioning lymphoid tissue system associated with the intestine (gut-associated lymphoid tissue, or GALT) [65].

The barrier properties of the intestinal mucosa can be compromised due to the following factors [60, 66]:

- inadequate perfusion (microcirculation) and oxygenation of organs of the gastrointestinal tract;
- excessive bacterial growth;
- long-term lack of nutrients in the lumen of the gastrointestinal tract (enterocytes are fed directly from the chyme);
- impairment of local and general immunity.

These factors lead to the infection of necrotic foci and the development of purulent-septic complications, SIRS, and MODS [12]. The main causes of intestinal ischemic disorders in AP are: systemic toxic effects of inflammatory mediators (free peroxide radicals, cytokines, activated neutrophils), systemic intestinal blood supply disorders (arterial hypotension, centralization of blood circulation,

decreased cardiac output), microcirculation disorders due to excessive release of cytokines and vasoactive substances, and elastase activation (Fig. 1) [59]. Ischemia leads to anaerobic metabolism, acidosis, and a decrease in the energy reserves of cells, which leads to irreversible changes. These changes occur quickly, and on the 4th day after the onset of the disease, atrophy of the intestinal mucosa and lymphoid tissue develops [49]. Morphological examination of the intestinal mucosa reveals exfoliation of necrotic enterocytes and the formation of erosions and ulcers, i.e., inflammation of the mucosa, which aggravates the phenomenon of bacterial translocation. This is also facilitated by general immunosuppression, microcirculation disorders, and excessive bacterial contamination as a result of dysbacteriosis, which may worsen with the use of antibiotic therapy [56].

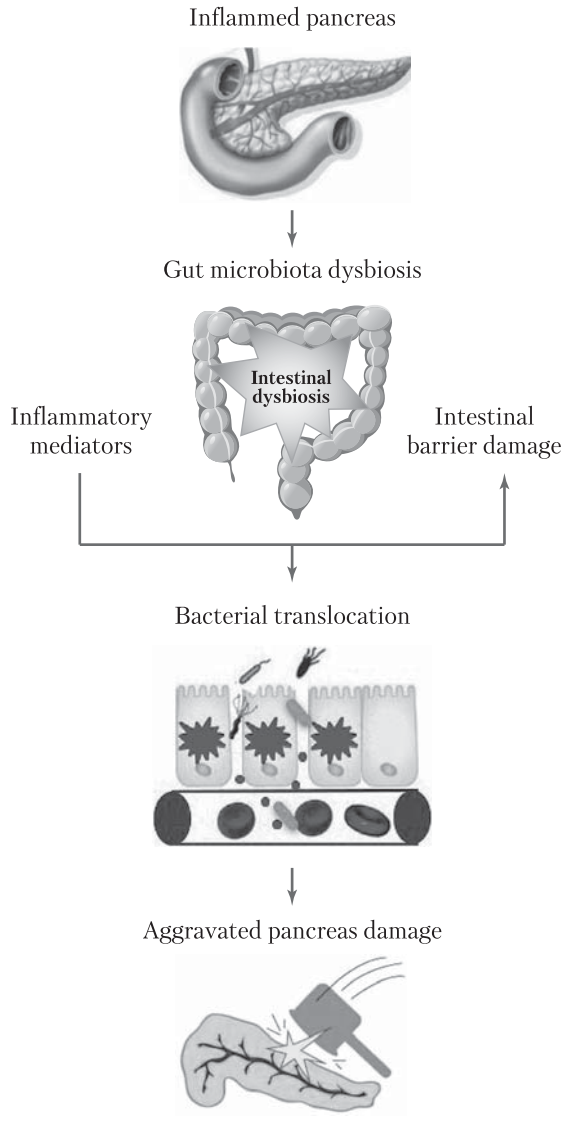

Figure 1. **The relationship between acute pancreatitis and gut microbiota [28]**

In inflammatory bowel diseases, there is an immunological imbalance characterised by increased production of pro-inflammatory cytokines (IL-6, IL-12) and decreased synthesis of anti-inflammatory cytokines (IL-4, IL-10, IL-11), resulting in an aggressive immune response to normal intestinal microflora [63].

The migration of bacteria through the intestinal wall is influenced by the number of microorganisms and the stability and competitiveness of the microflora. Normally, intestinal bacteria, together with the epithelium, form a natural protective barrier against exogenous microorganisms. With AP, the stable balance of the intestinal microflora is disrupted due to antibiotic therapy, a deficit of nutrients and fibre, and a lack of inter-microbial antagonism, leading to excessive bacterial growth and an advantage in the microbial population of gram-negative microflora [62]. This increases the risk of microbial translocation and the occurrence of purulent-septic complications [51].

In modern literature, there are many clinical studies according to which EN prevents degenerative changes in the intestinal mucosa, contributes to the normalisation of the immune response, and improves the state of the immune system as a whole [35]. As previously stated, fluid sequestration causes a reduction in the volume of circulating blood in patients with AP. In this case, intestinal ischemia occurs, which leads to a decrease in the energy potential of enterocytes and the activation of lipid peroxidation. The cells of the intestinal mucosa are deprived of energy substrates and the ability to maintain intercellular contacts. In turn, the entry of the nutrient mixture into the intestines increases both mesenteric blood circulation and the energy potential of enterocytes [36].

Thus, EN effectively restores the body's energy and plastic needs, is physiological, prevents atrophic processes, and improves the barrier characteristics of the intestinal mucosa.

Peculiarities of enteral nutrition in patients with acute pancreatitis

Although the administration of glucose, proteins, and lipids is a necessity for patients with AP, EN has long been considered dangerous because of possible stimulation and increased secretory activity of pancreatic cells. However, it has now been proven that the introduction of glucose or food mixtures containing amino acids and lipids into the small intestine is well tolerated by patients with AP and causes weak stimulation of pancreatic cells [44, 54].

It should be noted that the intravenous administration of carbohydrates, proteins, and fats also does not cause an increase in the secretory activity of pancreatic

cells, and in the case of the administration of protein hydrolyzate, the effect of their suppression occurs. However, with intravenous administration of glucose in patients with a severe course of AP, there is a risk of hyperglycemia due to insulin resistance, which is observed in patients with critical conditions [64].

The disadvantages of using parenteral nutrition in patients with AP are [26]:

- the need to transfuse large volumes of fluid, which is not always possible in elderly patients and in patients with severe concomitant (cardiovascular and pulmonary) pathology;
- patient's hypersensitivity to different components in the solutions for parenteral nutrition;
- the risk of phlebitis, thrombosis, embolism, and angiogenic sepsis;
- the risk of developing hyper- and hypoosmolar conditions and aggravation of acid-alkaline balance disorders;
- trophic and degenerative changes in the intestines, suppression of normal flora, bacterial contamination.

Notwithstanding the aforementioned challenges in facilitating access for EN, the introduction of a food mixture into the gastrointestinal tract is currently regarded as the most justifiable course of action.

The effectiveness and safety of EN in patients with AP depend on a complex of factors: the type of mixture, the method of administration of the mixture, the timing of recovery of peristalsis, and the absorption function of the intestinal wall.

Depending on their composition and energy value, mixtures for EN are classified as [30]:

- standard isocaloric mixtures (correspond to daily needs under normal conditions with the preserved function of the gastrointestinal tract);
- hypercaloric mixtures (contain an increased number of proteins and energy with a limited amount of liquid);
- mixtures with increased protein content, enriched with trace elements, glutamine, arginine, and omega-3 fatty acids (indications for use are critical and immunodeficient states);
- mixtures with a low content of fats and carbohydrates, containing dietary fibres (prescribed for patients with diabetes);
- mixtures with high fat content and low carbohydrate content (prescribed for lung function disorders at the stage of decompensation);
- mixtures with a low content of aromatic amino acids and an increased content of amino acids with a branched chain (prescribed for liver failure);
- oligomeric mixtures containing dipeptides, tripeptides, and several amino acids (prescribed for disorders of the gastrointestinal tract);
- immunostimulating hypocaloric mixtures with increased glutamine content (prescribed in critical conditions).

Depending on their formula, polymer mixtures (containing elements of whole protein, partially split starch, triglycerides, vitamins, macro- and microelements) are distinguished from semi-elemental and elemental mixtures (containing short peptides, crystalline amino acids, dextrose, oligosaccharides, essential fatty acids, and medium-chain triglycerides) [46]. It should be noted that for the absorption of polymer mixtures, their enzymatic processing in the intestines is necessary, while semi-elemental and elemental ones are already pre-hydrolyzed. Thus, polymer mixtures include *Nutrilan* (Germany), *Nutrison* (Holland), *Nutren 1.0* (Switzerland), *Isocal* (Netherlands), and others (the osmolarity of the mixtures is 380 mosm/L, pH 6, 8). Semi-elemental and elemental include *Peptamen* (Switzerland), *Reabilan* (USA), *Alfare2* (Switzerland), *Pepli-2000* (Netherlands), and others (the osmolarity of the mixtures is 315 mosm/L, pH 7.6) [3].

Numerous studies are currently comparing the effects of elemental, semi-elemental, and polymer mixtures used in the enteral nutrition of patients with AP [43]. As a rule, the authors prefer semi-elemental and elemental mixtures, explaining this by the fact that these mixtures cause less stimulation of the pancreas. This is due to the low fat content and the presence of free amino acids instead of intact proteins, which bind to free trypsin in the intestine and cause both a decreased level of trypsin and acidity in the stomach. However, according to the results of other studies, any type of food mixture introduced into the gastrointestinal tract causes a certain stimulation of the pancreas [37]. However, according to other publications, using polymer combinations rather than semi-elemental or elemental mixtures does not raise the risk of infectious complications or mortality in patients with AP [3, 4].

Some attention is paid to the gastrointestinal tract, where the food mixture is introduced. EN can be done through a tube inserted into the stomach, duodenum, or small intestine [7, 13]. There are also operative techniques for applying a gasto- or jejunostomy by an open or laparoscopic approach [53, 62]. It should be noted that probes with a large diameter can create significant discomfort for the patient, while probes with a small diameter often become impenetrable. Endoscopic techniques for inserting food probes have become popular in recent years. However, in the first 24 hours after endoscopic nasointestinal probe fitting, 15—25 % of patients experience dislocation of the latter in the shunt [5]. There is also conflicting evidence regarding the

feasibility of nasogastric tube feeding. There are a number of contraindications to the nasogastric administration of food mixtures: the presence of large gastric residual volumes (over 500 mL/6 hours), abdominal pain or vomiting due to delayed gastric emptying, and the inability to meet the patient's energy needs within 72 hours from the start of enteral nutrition (< 70 % based on 25 kcal/kg of body weight during hospitalisation) [42, 43]. Some authors indicate that the introduction of a mixture for enteral nutrition into the stomach or duodenum leads to stimulation of pancreatic secretion and increases the risk of aspiration pneumonia (observed in 6—8 % of patients) [33] and respiratory failure (observed in 25.8 % of patients. The assessment was carried out according to the Marshall scale: $PaO_2/FiO_2 < 300$ mm Hg, score ≥ 2) [31].

Traditionally, it is believed that the introduction of the mixture into the cavity of the small intestine 20—120 cm distal to the ligament of Treitz does not cause a stimulating effect on the pancreas (it is possible to avoid the cerebral, gastric, and intestinal phases of secretion, but the synthesis of secretin and cholecystokinin-pancreozymin is inhibited [37, 38]. At the same time, the statement about creating «rest for the pancreas» in AP is currently being revised [52]. It has been suggested that despite inflammation and/or necrotic changes, the pancreas continues to produce enzymes in response to stimulation. However, animal studies in the experimental modelling of AP showed that the exocrine secretion of the pancreas is inhibited when inflammation occurs, even when stimulated by cholecystokinin [20]. Other studies indicate inhibition of trypsin synthesis in patients with AP, especially in the case of acute necrotizing pancreatitis, but the rate of appearance of newly synthesised trypsin remains unchanged [37, 38, 48].

Hence, the lack of deterioration in the health status of individuals with AP who are administered EN can be explained by the decreased pancreatic response to food mixtures and the reduced secretory response to basal indicators.

At the same time, there are studies that prove the absence of a reliable difference in the frequency of complications and deaths when using the nasogastric or nasojejunal method of introducing a mixture for nutrition in patients with AP [27].

In our clinic, a study was conducted comparing the effectiveness and safety of nasogastric feeding mixtures in patients with a severe course of AP [22]. According to the results of the study, it was established that the provision of nasogastric nutritional support in patients with a severe course of AP leads to a decrease in the frequency of local infectious complications by 25.8 % ($\chi^2 = 4.59$; 95 % CI 2.43—45.53; $p = 0.03$), the duration of multiple organ failure by 3.1 days ($p < 0.001$), the duration of hospital stay by 16 days ($p = 0.04$), and deaths by 21.4 % ($\chi^2 = 4.13$; 95 % CI 0.81—39.68; $p = 0.04$) in comparison with parenteral nutrition. In addition, no significant difference was found between the frequency of occurrence of local infectious complications and deaths, the duration of MODS, and the length of stay of patients in the hospital when comparing nasogastric and enteral administration of food mixtures in patients with a severe course of AP.

There are also many studies devoted to the timing of the introduction of food mixtures in patients with AP. Thus, according to modern views, enteral nutrition should be started as early as possible (24—72 hours from the moment of hospitalisation) compared to parenteral nutrition [19]. Early onset of EN (up to 48 hours from the moment of hospitalisation) in patients with a severe course of AP is associated with a decrease in the frequency of infectious complications by 24 % and mortality by 32 % [18, 55]. However, today there are no clear criteria for the onset of EN in this category of patients. Thus, some authors emphasise that factors that indicate the possibility of starting the administration of enteral mixtures are the appearance of peristalsis and a decrease in intra-abdominal pressure below 10 mm Hg [10]. At the same time, it should be noted that restoration of intestinal absorption can occur at a later time, and this is dangerous due to the occurrence of a number of complications [40, 55]:

- disorders of the gastrointestinal tract in the form of nausea, vomiting, occurrence of gastroesophageal reflux, gastrointestinal bleeding, and increased pain (occurs in 30—38 % of patients);
- the occurrence of injuries of the upper parts of the gastrointestinal tract and respiratory tract with the development of rhinitis, pharyngitis, esophagitis, pulmonary aspiration, erosions and perforation of the esophagus, displacement, and obliteration of the feeding probe (2—10 %);
- occurrence of infectious complications in the form of parotitis, otitis, sinusitis, aspiration *pneumonia*, and microbial contamination (6—8 %);
- aggravation of metabolic disorders in the form of disturbances in the metabolism of calcium, magnesium, phosphorus, fluid balance, occurrence of hyperosmolar states, hyper- or hypoglycemia (10—15 %);
- occurrence of intestinal complications in the form of large residual volumes — 39 %, diarrhea — 14.7 %, flatulence — 13.2 %, vomiting — 12.2 %, regurgitation — 5.5 %.

Early use of EN in patients with AP can also cause the emergence of digestive and dynamic types of increased gas formation in the gastrointestinal tract

[58]. This is explained by the fact that the introduction of a mixture for nutrition can cause an imbalance between the bacteria that participate in the production of gases and their absorption. In turn, the combination of maldigestion and malabsorption syndromes and reflex suppression of intestinal motility against the background of AP lead to a disorder of gas transportation and absorption [15]. Liquid fecal masses, which are located in the intestines and contain various organic compounds consisting of proteins, fats, and carbohydrates, contribute to the formation of a large number of bubbles of various diameters, surrounded by mucus (foam). The bubbles cover the epithelial layer of the intestinal wall and lead to disorders of parietal digestion and the absorption of nutrients [8]. Therefore, determining and assessing the recovery of intestinal absorption can be one of the main criteria for the initiation of enteral tube feeding in patients with AP. There are known methods of assessing the recovery of intestinal absorption using labelled radioisotopes (Cu67-ceruloplasmin, Cr51-albumin) or by the level of absorption and secretion of disaccharides (lactulose/mannitol) that are not metabolised (in this case, the state of the urinary system should also be taken into account) [6]. The test using disaccharides is carried out as follows: 5.0 g of mannitol and 5.0 g of lactulose, dissolved in 100 ml of distilled water, are introduced into the probe. Urine is collected within 6 hours after administration of the mannitol and lactulose solutions. The analysis is carried out by the method of ion chromatography with a pulsed amperometric detector. Mannitol passes through the intestinal epithelium by passive transport. The average absorption rate is 14 %. Lactulose, being a larger molecule, is, on the contrary, poorly absorbed in the intestines. The degree of its absorption is less than 1 %. Therefore, the lactulose/mannitol ratio in urine is normally less than 0.03 [17].

There is also a way to assess the restoration of intestinal absorption by determining the fasting glucose level two hours after exercise (introduction of glucose into a feeding tube at a dose of 1 g/kg of the patient's body weight). An increase in the level of glucose in blood serum after 2 hours is a sign of restoration of intestinal absorption (restoration of the enzyme activity of intestinal disaccharidases) [14]. However, the use of this method is limited in the case of fluctuations in the level of glucose, which can be observed against the background of the course of AP and other accompanying pathologies (Itsenko-Cushing syndrome, diabetes, hyperthyroidism, acromegaly, etc.).

Due to the significant limitations and low efficiency of the aforementioned tests, which can be attributed to a variety of factors, the search for objective criteria to initiate enteral tube feeding in patients with AP should be aimed at assessing the restoration of intestinal absorption.

We conducted a study, the purpose of which was to determine the timing of the recovery of intestinal absorption as one of the main criteria for the initiation of EN in patients with AP and to improve the results of the complex treatment of patients by preventing its complications [23]. It was found that in the vast majority of patients with AP (70.6 % of patients in the main group and 69.7 % of patients in the comparison group), recovery of intestinal absorption occurs on average 48 hours after the start of complex conservative therapy, so this time is optimal for the beginning of EN (Table).

To determine the beginning of intestinal absorption, we developed our own method using a 3 % potassium iodide solution (the sensitivity is 87.36 %, and the specificity is 81.5 %). The method consists of determining the timing of restoration of intestinal absorption by recording excretion with saliva of potassium iodide 10 minutes after its enteral probe administration (20 ml of 3 % solution). The transparent secretion taken into a test tube changes its colour to blue when the indicator — starch (2 ml of 10 % solution) is added to it, in case of restoration of intestinal absorption.

As an alternative method for determining the beginning of intestinal absorption, a sample with disaccharides (lactulose or mannitol) that are not metabolised was used (introduction of disaccharides was carried out at the beginning of treatment, after 12, 24, 36, and 48 hours). When comparing the mean levels of lactulose/mannitol in the urine and their standard deviation in the main group and the comparison group at the beginning of treatment (0.042 ± 0.001 and 0.041 ± 0.001; $p = 0.64$ respectively), after 12 hours (0.040 ± 0.002

Table. **Terms of recovery of intestinal absorption in patients with severe acute pancreatitis depending on the duration of treatment**

Duration of treatment of the patient in the hospital	Restoration of intestinal absorption processes	
	Main group (n = 34)	Comparison group (n = 33)
12 hours	–	–
24 hours	3 (8.8 %)	4 (12.1 %)
36 hours	10 (29.4 %)	9 (27.3 %)
48 hours	24 (70.6 %)	23 (69.7 %)

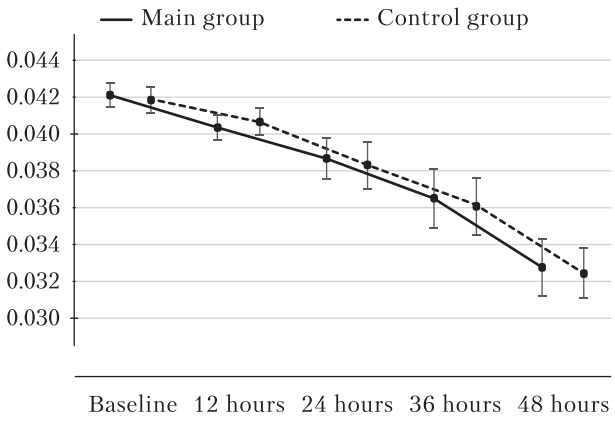

Figure 2. **Histogram of the ratio of lactulose/ mannitol in the urine in the study groups**

and 0.041 ± 0.002; p = 0.27 respectively), 24 hours (0.039 ± 0.002 and 0.039 ± 0.003; p = 0.92 respectively), 36 hours (0.036 ± 0.003 and 0.037 ± 0.004; p = 0.9 respectively), 48 hours (0.033 ± 0.004 and 0.033 ± 0.004; p = 0.9 respectively), no significant difference was obtained (Fig. 2).

It should be noted that the protocols for providing assistance to patients with intestinal digestive disorders arising from the use of EN in the treatment of AP, as well as their prevention, are insufficiently developed. Activated charcoal, loperamide, etc. are used to reduce gas formation in the intestines [57]. However, the therapeutic effect of these drugs is insignificant, so this issue needs further study.

Therefore, nutritional support is an important component of therapy in patients with AP, the purpose of which is to ensure adequate caloric intake, modulate the response to oxidative stress, and counteract catabolic effects during the course of the disease. It should be noted that the degree of nutritional status disorders in patients with AP varies depending on the etiological factors and the severity of the disease and requires a differentiated approach to their correction. Currently, it is believed that with a mild course of AP, fasting does not affect the course and outcome of the disease, and this category of patients does not require the prescription of active nutritional support. Whereas with severe AP, adequate protein and energy supply is one of the key points of intensive therapy, the completeness of which affects the frequency of the development of complications, the duration of hospitalisation, and mortality [26]. In patients with AP, the optimal time to start nutritional support is 48 hours after hospitalisation, whereas one of the main criteria for initiating EN is the restoration of intestinal absorption. Nutritional support should be started with nasogastric administration of a food mixture, and in case of complications (intolerance, aspiration, etc.), nasojejunal administration. At the same time, it is possible to use polymer, semi-elemental, and elemental mixtures. Parenteral nutrition should be used if enteral nutrition is impossible or not tolerated.

Conclusions

In the treatment of acute pancreatitis, nutritional support is an important component. It ensures adequate caloric intake, modulates the response to oxidative stress, and counteracts catabolic effects during the course of the disease.

The degree of nutritional disorders in patients with acute pancreatitis varies depending on the etiological factors and severity of the disease, necessitating a differential approach to their correction.

Restoration of intestinal absorption in patients with severe acute pancreatitis occurs on average 48 hours after the start of complex conservative therapy, which is the optimal time to initiate enteral nutrition.

Nasogastric nutritional support in patients with severe acute pancreatitis is safe and leads to a 25.8 % reduction in the incidence of local infectious complications ($\chi^2 = 4.59$; 95 % CI 2.43—45.53; p = 0.03), the duration of multiple organ failure by 3.1 days (p < 0.001), the length of stay in the hospital by 16 days (p = 0.04), and mortality by 21.4 % ($\chi^2 = 4.13$; 95 % CI 0.81—39.68; p = 0.04) compared to parenteral nutrition.

Declaration of interests

The authors declare that they have no conflicts of interest.

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Authors contributions

I. V. Kolosovych: conception and design; I. V. Hanol: collection, analysis and interpretation of data, drafting and revision of the manuscript.

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Нутритивна підтримка в пацієнтів із гострим панкреатитом. Огляд опублікованих досліджень

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Гострий панкреатит — поширене захворювання, що трапляється у 5—10 % пацієнтів з ургентною патологією органів черевної порожнини. В основі розладів обмінних процесів, які виникають у цієї категорії пацієнтів, лежать синдроми гіперметаболізму та гіперкатаболізму, що супроводжуються підвищеними витратами вуглеводів, жирів і амінокислот, зростанням споживання кисню та продукції вуглекислого газу.

Мета — дослідити сучасний стан проблеми нутритивної підтримки пацієнтів із гострим панкреатитом.

Ступінь розладів харчового статусу в пацієнтів із гострим панкреатитом варіює залежно від етіологічних чинників і тяжкості захворювання та потребує диференційованого підходу до їхньої корекції. У хворих на гострий панкреатит спостерігається порушення балансу кишкової мікрофлори внаслідок прийому антибіотиків, нестачі поживних речовин, клітковини, а також відсутності мікробного антагонізму, що призводить до надлишкового бактеріального росту з переважанням грамнегативної мікрофлори в мікробній популяції. Ефективність і безпечність ентерального зондового харчування зумовлена комплексом чинників: термінами відновлення перистальтики і всмоктувальної функції кишкової стінки, типом суміші, способом її введення. Відновлення кишкової абсорбції у хворих на тяжкий гострий панкреатит відбувається в середньому через 48 год від початку комплексної консервативної терапії. Використання антифлатулентів у складі суміші для ентерального харчування дало змогу поліпшити лабораторні показники сироватки крові, зменшити частоту розвитку кишкових ускладнень на 7-му добу на 21,5 % (χ^2=4,88, 95 % довірчий інтервал (ДІ) 2,3—39,5; p=0,03). Проведення назогастральної нутритивної підтримки в пацієнтів із тяжким перебігом гострого панкреатиту було безпечним та сприяло зниженню частоти виникнення локальних інфікованих ускладнень на 25,8 % (χ^2=4,59; 95 % ДІ 2,43—45,53; p=0,03), тривалості перебування в стаціонарі на 16 діб (p=0,04) і рівня летальності на 21,4 % (χ^2=4,13; 95 % ДІ 0,81—39,68; p=0,04) порівняно з парентеральним харчуванням. Нутритивну підтримку слід розпочинати з назогастрального введення харчової суміші, у разі виникнення ускладнень (непереносність, аспірація тощо) — з назоєюнального введення. Парентеральне харчування слід використовувати, якщо ентеральне харчування неможливе або не переноситься.

Ключові слова: гострий панкреатит, нутритивна підтримка, ентеральне харчування, ускладнення.

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