

PATHOGENETIC MECHANISMS OF ENTERIC INSUFFICIENCY DEVELOPMENT IN DISSEMINATED PERITONITIS

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RESUME

Disseminated purulent peritonitis is one of the main causes of high mortality in emergency surgery which, with the development of abdominal sepsis, can reach 35–41 %. Secondary peritonitis initiates a cascade of pathophysiological changes in the intestinal system, including intestinal barrier dysfunction, impaired neuroendocrine regulation, and disruption of microbiotic homeostasis. These changes in 85–100 % of cases naturally lead to the development of enteric insufficiency syndrome (EIS), which is a key pathogenetic factor in the development of abdominal sepsis and multiple organ dysfunction via the “intestine – target organ” mechanism.

In this review article, we analyzed recent open-source studies on the development of enteric insufficiency in secondary peritonitis. We searched the databases of the Scientific Library of Russia (eLibrary.ru, Cyberleninka.ru) and the US National Library of Medicine (PubMed.org, Wiley.com) over the past decade (2014–2024) in Russian and English. The following keywords were used for the search: “peritonitis”, “enteral insufficiency”, “etiology of enteral insufficiency”, “pathogenesis of enteral insufficiency”, and “abdominal sepsis”. The inclusion criteria for the review included studies on the development of enteral insufficiency in inflammatory abdominal diseases and peritonitis. Exclusion criteria for the review: articles containing information on the development of enteral failure in patients with septic, metabolic, and complex nutritional complications typically occurring in patients with inflammatory bowel disease requiring multidisciplinary intervention with metabolic and nutritional support for recovery, as well as enteral failure requiring long-term nutritional support.

This literature review aims are to systematize the current understanding of the pathogenesis of enteral failure syndrome associated with secondary purulent peritonitis, taking into account existing gaps in research. The results of this analysis have practical value for surgeons and specialists in related fields.

Key words: peritonitis, enteral failure, etiology of enteral failure, pathogenesis of enteral failure development

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ПАТОГЕНЕТИЧЕСКИЕ МЕХАНИЗМЫ РАЗВИТИЯ ЭНТЕРАЛЬНОЙ НЕДОСТАТОЧНОСТИ ПРИ РАСПРОСТРАНЁННОМ ПЕРИТОНИТЕ

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РЕЗЮМЕ

Распространённый гнойный перитонит является одной из основных причин высокой летальности в urgentной хирургии, которая, при развитии абдоминального сепсиса, может достигать 35–41 %. Вторичный перитонит инициирует каскад патофизиологических изменений в интестинальной системе, включающий дисфункцию кишечного барьера, нарушение нейроэндокринной регуляции, расстройство микробиотического гомеостаза. Эти изменения в 85–100 % случаев закономерно приводят к развитию синдрома энтеральной недостаточности (СЭН), который является ключевым патогенетическим звеном в развитии абдоминального сепсиса и полиорганной дисфункции по механизму «кишечник – органы-мишени».

В обзорной статье мы проанализировали последние исследования, представленные в открытых источниках, посвящённые проблематике развития энтеральной недостаточности при вторичном перитоните. Поиск информации проводился по базам данных Научной библиотеки России (eLibrary.ru, Cyberleninka.ru) и Национальной медицинской библиотеки США (PubMed.org, Wiley.com) за последнее десятилетие (2014–2024 гг.) на русском и английском языках. В качестве поисковых запросов использовались следующие ключевые слова: «перитонит», «энтеральная недостаточность», «этиология энтеральной недостаточности», «патогенез развития энтеральной недостаточности», «абдоминальный сепсис». Критерии включения в обзор: работы, посвящённые развитию энтеральной недостаточности при воспалительных заболеваниях брюшной полости и перитоните. Критерии исключения из обзора: статьи, содержащие информацию о развитии энтеральной недостаточности у пациентов с септическими, метаболическими и сложными нутритивными осложнениями, обычно возникающими у пациентов с воспалительными заболеваниями кишечника, требующими многопрофильного вмешательства с метаболической и питательной поддержкой для восстановления, а также энтеральная недостаточность, требующая длительной питательной поддержки.

Настоящий литературный обзор направлен на систематизацию современных представлений о патогенезе синдрома энтеральной недостаточности, ассоциированной с вторичным гнойным перитонитом, в контексте существующих пробелов в его изучении. Результаты проведенного анализа представляют практическую ценность для врачей-хирургов и специалистов смежных областей.

Ключевые слова: перитонит, энтеральная недостаточность, этиология энтеральной недостаточности, патогенез развития энтеральной недостаточности

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INTRODUCTION

Secondary peritonitis (SP) is a clinically significant surgical pathology occupying a leading position in the structure of urgent abdominal morbidity [1]. Epidemiological studies demonstrate that SP accounts for about 1 % of all cases of hospitalization in general surgical hospitals [2, 3]. According to international registries, this nosology is the second most significant etiological cause of septic conditions among patients in critical care units [2, 3, 4].

Prognostic parameters in SP are characterized by significant variability: overall mortality is about 6 % while in cohorts of patients with complicated forms (sepsis, septic shock) this parameter reaches 35–41 % [2, 5, 6]. The pathogenetic concept proposed by M. Schein et al. in 1996 describes a two-component model of peritonitis integrating infectious and inflammatory cascades [7]. The key position of this hypothesis is that even with adequate surgical debridement and optimal antimicrobial therapy, a significant risk of lethal outcome persists, caused by systemic inflammatory response syndrome (SIRS) and progressive multiple organ dysfunction (MODS) [2].

At the cellular and molecular level, SP initiates a cascade of pathophysiological changes in the intestinal system, including intestinal barrier dysfunction, impaired neuroendocrine regulation, and disruption of microbiotic homeostasis [8, 9]. In 85–100 % of cases, these processes naturally lead to the development of enteral insufficiency syndrome (EIS) [10], which acts as a key pathogenetic link in the formation of abdominal sepsis and multiple organ dysfunction of the “intestine – target organs” type [10, 11].

The modern concept considers EIS as a system of interrelated pathogenetic processes, the primary link of which is dysregulation and destabilization of the membrane homeostasis of tissue structures of the intestinal wall, especially its mucous layer, which causes profound disorders of the functional and metabolic status of the intestine against the background of acute abdominal pathology [10].

At the same time, EIS acts as a connecting link between reflex intestinal paresis and multiple organ dysfunction. EIS aggravates the course of the underlying disease, increases the probability of complications, and increases the risk of unfavorable outcomes.

An analysis of existing publications reveals significant gaps in the understanding of key aspects of the pathogenesis of enteral insufficiency. In particular, there is insufficient fundamental research on the study of pathogenetic mechanisms of enteral disorders at the level of cell membranes of enterocytes, underlying the dysregulation of the barrier function of the intestine. In addition, the pathogenesis of motor-evacuation disorders and the role of damage to the mucous membrane in diffuse purulent peritonitis require systematic study to establish their universality and pathogenetic significance.

It is precisely therefore that a deeper study of the pathogenetic mechanisms of the development of enteral insufficiency will allow creating the basis for the development of therapeutic strategies aimed at correcting key links of pathogenesis and, as a consequence, at the severity of the course of peritonitis. The introduction of a stratified approach that takes into account the features of the development of EIS in each specific clinical case determines the potential possibility of introducing important changes into the complex of treatment and reducing mortality in this group of patients.

AIM

To analyze literature data and present a review of current concepts of the pathogenetic mechanisms of the development of enteral insufficiency syndrome in secondary diffuse peritonitis.

MATERIALS AND METHODS

Using the databases of the Scientific Library of Russia (eLibrary.ru, Cyberleninka.ru) and the US National Library of Medicine (PubMed.org, Wiley.com) over the past decade (2014–2024) in Russian and English, an analysis of studies devoted to the problems of the development of enteral insufficiency in secondary peritonitis was conducted. The following keywords were used as search queries: “peritonitis”, “enteral insufficiency”, “etiology of enteral insufficiency”, “pathogenesis of enteral insufficiency”, “abdominal sepsis”, “peritonitis”, “enteral failure”, “etiology of enteral failure”, “pathogenesis of enteral failure development”, “abdominal sepsis”.

Inclusion criteria for the review: works devoted to the development of enteral insufficiency in inflammatory diseases of the abdominal cavity and peritonitis.

Exclusion criteria: articles containing information on the development of enteral insufficiency in patients with septic, metabolic, and complex nutritional complications that usually occur in patients with inflammatory bowel diseases requiring multidisciplinary intervention with metabolic and nutritional support for recovery, as well as information on the development of enteral insufficiency requiring long-term nutritional support.

In the analysis of the literature for the specified period, a predominance of sources from foreign search systems was established.

RESULTS AND DISCUSSION

For three decades, the hypothesis that the intestine plays a key role in the development of multiple organ dysfunction syndrome has dominated. According to this concept, inflammatory processes in the abdominal cavity increase the permeability of the intestinal barrier,

which promotes the translocation of viable bacteria into the systemic bloodstream, causing the phenomenon of endotoxin aggression and leading to abdominal sepsis and multiple organ failure. However, further studies of pathogenetic mechanisms have shown that intestinal dysfunction is formed as a result of more complex and multicomponent processes [12, 13].

The intestine is a finely tuned dynamic ecosystem consisting of approximately 40 trillion microorganisms, a single-layer intestinal epithelium that separates the host from the microbiome and its products, as well as the immune system of the mucous membrane, which actively interacts with them in both directions. Normally, all elements maintain a balanced homeostasis regulated by hormones, dietary components, inflammatory mediators, and the intestinal nervous system [14], which benefits both the host and its internal microbial community [12, 15].

In secondary peritonitis, against the background of inflammatory changes in the abdominal cavity, disruption of the function and damage to each component of the intestine occurs, which leads to damage to both local and distant organs. In addition, the interaction between the components of the intestine and distant organs as a result of inflammation aggravates cell and organ damage [12, 16].

The complexity of the functioning of the intestine lies in the need to simultaneously perform two opposite tasks: ensuring selective permeability for the absorption of nutrients into the bloodstream and the internal environment of the body, and at the same time – an effective barrier against the penetration of pathogens, antigens, proinflammatory factors and other harmful substances from the intestinal lumen [17].

In critically ill patients, the protective mechanisms supporting the intestinal barrier cease to work due to impaired blood circulation and neuroendocrine regulation. Pathogenic processes include hypoperfusion of the mesentery, impaired intestinal motility, and disruption of the intestinal barrier with subsequent translocation of substances from the intestinal lumen [8, 16].

IMPAIRMENT OF GASTROINTESTINAL MOTILITY IN PERITONITIS

The development of purulent inflammation in the abdominal cavity in peritonitis is accompanied by an imbalance of sympathetic and parasympathetic regulation, which serves as a key pathogenetic factor in the impairment of the motor function of the gastrointestinal tract [18].

The enteric nervous system consists of many small ganglia that work independently of each other but retain the ability to interact with each other, forming myenteric and submucosal nerve plexuses, and are crucial for the proper functioning of the intestine. Thus, the myenteric plexus (Auerbach's) regulates the contractile activity of smooth muscles, ensuring the passage

of chyme, while the submucosal plexus (Meissner's) modulates the processes of secretion and absorption. Synchronization of their functions is critically important for effective digestion and assimilation of nutrients [19].

In the initial stages of diffuse peritonitis, the disorder of motility, especially in the small intestine, acts as the primary link in the cascade of pathological changes leading to the progression of intestinal insufficiency syndrome. Suppression of gastrointestinal motility during the development of inflammation in the abdominal cavity by the central nervous system occurs in response to intense afferent impulses from peritoneal receptors. Local metasympathetic innervation of the intestinal wall, controlled by pacemaker cells of the duodenum, generates the migrating myoelectric complex (MMC) [20]. In peritonitis, inflammation of the visceral peritoneum reflexively suppresses peristalsis mediated by central neuroendocrine mechanisms. The dominance of sympathetic regulation causes persistent intestinal hypokinesia, accompanied by suppression of the MMC. This leads to parietic dilation of intestinal loops, wall ischemia, and hypoxic damage to the metasympathetic nervous system [21].

Intestinal neurons, in response to inflammation, express Toll-like receptors (TLRs), such as TLR2 and TLR4, which suggests that bacterial metabolites affect the enteric nervous system [18]. Components of the cell walls of gram-negative and gram-positive bacteria under conditions of peritonitis activate TLR4 and TLR2, which leads to a "cytokine storm" – the release of proinflammatory mediators. All this affects the progression of changes in the intestinal wall and contributes to an increase in its permeability [18, 22].

Subsequent impairment of neurotransmission is caused by dysmetabolic changes, including the accumulation of acid hydrolases and intermediate metabolites, which disrupts cholinergic transmission in neuromuscular synapses of the intestinal wall and intermuscular plexuses [21].

Impairment of the motor function of the intestine leads to the cessation of the process of evacuation of contents, its accumulation in the lumen – all this causes overstretching of intestinal loops, impaired microcirculation in the wall, and its ischemia. Ischemia in this case comes to the fore as an endogenous cause of damage to the intestinal wall, which becomes permeable to the pathogenic microflora contained in its lumen.

ROLE OF THE INTESTINAL EPITHELIUM IN THE DEVELOPMENT OF ENTERAL INSUFFICIENCY

The intestinal epithelium functions, as the main barrier between the host organism and the complex microbial community in the intestinal lumen [23], also performs many key functions that ensure homeostasis in the body, such as the absorption of nutrients,

barrier protection against pathogens, secretion of hormones and cytokines, synthesis of antimicrobial peptides, and constant interaction with both the gut-associated lymphoid tissue (the largest lymphoid organ) and the microbiota of the intestinal lumen [24].

The human host receives almost all nutrients and is simultaneously protected from trillions of potential microbial invaders by a single-layer intestinal epithelium. At the base of the intestinal crypt lie pluripotent stem cells that actively proliferate and give rise to daughter cells. These cells differentiate as they migrate upward along the villi into absorbing enterocytes, goblet cells producing mucus, enteroendocrine cells that trigger the immunity of the mucous membrane against parasitic infections, and Paneth cells, which play an antimicrobial role and regulate the microbiota through the secretion of lysozyme, defensin, and other immunoregulatory proteins [25]. All epithelial cells gradually move to the tops of the villi, while Paneth cells migrate to the base of the crypt. At the same time, epithelial cells are either completely displaced into the intestinal lumen or die as a result of apoptosis, which ensures continuous turnover and renewal of the intestinal epithelium. The entire path from the birth of a cell to migration/differentiation and death takes less than a week [12, 15].

In the development of secondary peritonitis, the integrity of the intestine is disrupted, apoptosis of the epithelium is accelerated, cell proliferation decreases, and their migration slows down. At the subcellular level, in peritonitis, a disruption of redox processes occurs, including the oxidation of glutathione and glutathione disulfide [26]. At the cellular level, a decrease in the proliferative activity of crypts is observed against the background of enhanced apoptosis in both crypts and villi [27]. In critical states, the migration of epithelial cells slows down due to the TLR4-dependent mechanism [11]. However, suppression of proliferation and enhanced apoptosis neutralize this slowdown, leading to a significant shortening of the villi [28], which leads to a decrease in the absorptive capacity of the intestine, changes in systemic inflammation, and an increase in paracellular permeability [12]. Also, pronounced changes occur in the structure of the mucous layer: the structure of the intestinal mucosa is destroyed, the number of LGR5⁺ (Leucine-rich repeat-containing G-protein coupled receptor 5⁺) cells and goblet cells decreases significantly, and the secretory function of goblet cells weakens [28, 29], which disrupts the barrier function of the intestine [28]. Normally, LGR5⁺ is a marker of adult intestinal stem cells, and the high rate of renewal of the intestinal mucosa directly depends on its concentration. Consequently, with a decrease in the concentration of LGR5⁺, the differentiation and renewal of small intestine cells are disrupted, which leads to morphofunctional disorders in the intestinal wall [30].

In an experimental study by D.S. Parshin (2020), it was shown that in the development of enteral insufficiency

in peritonitis, in micropreparations of the small intestine, shortening and thinning of intestinal villi are observed, Paneth cells disappear, while massive desquamation of epithelial cells is noted in the area of the tops of the villi with exposure of the connective tissue base. A statistically significant decrease in CD3⁺ and an almost complete absence of Bcl-2⁺ cell expression were noted, which serves as additional evidence of irreversible apoptotic changes in enterocytes [31].

Also, D.V. Volkov, et al. (2016) studied the morphofunctional state of the small intestine of experimental animals with EIS against the background of experimental fecal peritonitis. It was established that already after 48 hours, pronounced heteromorphism of the structure of the small intestine villi is observed. The epithelium covering the villi is flattened, significantly infiltrated with lymphocytes, has a basophilic cytoplasm, and a high content of ribonucleoproteins. The number of goblet cells increases, especially at the bottom of the crypts, while epithelial cells have signs of dystrophic changes (karyopyknosis, karyolysis, vesiculation of the cytoplasm). Local desquamation of epitheliocytes is observed. Damage to the mucous membrane can penetrate to the submucosal and serous membranes [32].

According to I.A. Shurygina et al. (2021), the morphological picture of damage to the small intestine associated with enteral insufficiency is formed during the first 12 hours of the development of experimental peritonitis. It was established that the most informative and statistically significant criteria reflecting the dynamics of damage to the intestinal wall were shortening of villi and disruption of the epithelial cover of the villi [33].

In a study by E.E. Chepurnykh, et al. (2024), where the severity of morphological damage to the intestinal wall was considered as an equivalent of EIS, a direct dependence of the severity of damage on the degree of morphological changes was established. A progressively increasing intensification of correlations is observed: on day 3, a strong correlation with the length and state of the villus stroma was revealed ($r_s = 0.84$); by day 7, the extent of damage to the intestinal wall was included in the model ($r_s = 0.82$); and by day 14, an integral correlation with the entire complex of morphological disorders was recorded ($r_s = 0.98$) [34, 35].

Thus, all the presented experimental studies confirm the study by Tang A.L., et al. (2022), which states that in peritonitis and abdominal sepsis, the mucous membrane of the intestine suffers more severely than the serous and muscular membranes, and this is associated with the features of its blood supply [36]. The capillaries of the villi, which have a physiologically determined "spiral-shaped structure", create uneven blood supply to their tops [37], which makes microcirculation in this zone especially vulnerable. As experimental studies by D.V. Volkov, et al. (2016) show, the blood vessels of the microcirculatory bed sharply dilate and become plethoric with signs of diapedesis of formed blood

elements [32]. Significant edema of the stromal elements of the vessel walls is combined with sludging of blood cellular elements and stasis in hemocapillaries, and thrombosis of small blood vessels. The data obtained by the researchers indicate an early disruption of microcirculation against the background of intensification of inflammation in the wall of the small intestine of experimental animals under simulated conditions with EIS, which leads to aggravation of dystrophic and necrobiotic lesions of epitheliocytes [32].

Simultaneously, an increase in the concentration of the adhesion molecule PECAM-1 (CD31) in the blood plasma of experimental animals occurs, while the concentration of CD31 is statistically significantly higher ($p < 0.05$) in animals with peritonitis. The data obtained by the authors indicate severe damage to the vascular endothelium and disruption of the tone of the microcirculatory bed [38].

Emerging hypoperfusion and ischemia damage the mucous barrier, intensify the immunoinflammatory response, and contribute to the release of biologically active substances into the blood and mesenteric lymphatic vessels [39].

According to B.P. Yoseph, et al., already one hour after the onset of the inflammatory process and abdominal sepsis, hyperpermeability of the intestinal barrier is noted [40]. An increase in the permeability of the intestinal wall and the development of endotoxemia are also associated with the weakening of tight junctions and the proteins associated with them, which serve as a kind of entrance gate for paracellular transport of various substances. Tight junctions in their structure represent a complex of occludin and claudin proteins connected to each other by desmosomes and Zonula occludens [41]. The permeability of the intestinal epithelium directly depends on the state of claudins and occludins and vice versa. In a functional state, the proteins are in a state of "waiting", however, in inflammation or damage to the intestinal wall, the expression of the gene encoding claudin increases. Thus, in the development of sepsis, the level of claudin-2 and intercellular adhesion molecules (JAM)-A increases, while the level of claudin-5 and occludin decreases [19]. Increased permeability caused by contraction of the actin-myosin ring after incubation with IFN- γ and TNF- α provokes the synthesis of IL-13 in enterocytes, which specifically enhances the expression of claudin-2 and increases the cation current [19]. In addition, bacterial infection activates myosin light chain kinase (MLCK), which phosphorylates the regulatory light chain of myosin, which leads to contraction of the actin-myosin ring and an increase in paracellular permeability [42]. This is associated with the action of inflammatory mediators such as TNF- α and IL-13, which induce the formation of tight junction proteins [42].

In an experimental study by T. Yumoto, et al., it was shown that in animals with occludin knockout, intestinal permeability significantly increases, and an increase in the level of systemic cytokines IL-6 and MCP-1

and bacteremia are also observed. In addition, in mice with occludin knockout, the level of IL-1 β and MCP-1 in the jejunum was increased, as well as the level of MCP-1 and IL-17A in the peritoneal fluid, although the number of bacteria in the peritoneal fluid did not change. The authors note that 7-day mortality was significantly higher in animals with occludin knockout. Thus, occludin plays a crucial role in maintaining the barrier function of the intestine and ensuring survival in sepsis associated with inflammation and bacteremia [43].

INTERACTION OF THE MUCOUS MEMBRANE AND THE INTESTINAL MICROBIOTA IN PERITONITIS

The mucous membrane of the intestine and the mucus produced by it prevent contact of bacteria with the intestinal epithelium. In inflammation, the mucous layer is damaged, its hydrophobicity changes, which, in turn, leads to dysfunction of epithelial cells and a change in intestinal permeability [42].

Normally, the homeostasis of the intestinal mucosa depends on physical and molecular interactions between three components: the resident microbiota, the epithelial layer, and the local immune system [44]. In a study by F. Schreiber, et al., the ability of the cytokine IL-22 to influence this three-way interaction and stimulate the restoration of intestinal tissues was shown. IL-22 is produced by immune cells located under the epithelium and is induced by bacteria present in the intestine. IL-22 stimulates epithelial cells through the receptor complex IL-22RA1-IL-10R2, causing changes in the expression of genes involved in maintaining the integrity of the epithelial barrier, as well as in various functions associated with resistance to pathogens, such as modification and moistening of the mucous layer, strengthening of tight junctions, and the production of a wide range of bactericidal compounds. These mechanisms of resistance to pathogens, in turn, affect the composition of the microbiota and create an environment that excludes pathogens [44]. In the development of peritonitis, the level of IL-18 increases, which reduces the level of the IL-22 binding protein, thereby, suppressing the production of antimicrobial proteins [14].

Thus, the production of intestinal mucus directly depends on the presence of the microbiota [44, 45].

Of interest are fundamental studies showing that individual bacteria produce structural appendages and soluble exoproducts that, interacting with pathogen receptors on the intestinal epithelium, serve as a trigger for launching a number of processes. For the initiation of subsequent cascade processes, bacteria do not require active movement, since the receptors of epithelial cells and antigen-presenting cells are accessible to luminal pathogens directly on the apical surface of the epithelial barrier.

In peritonitis, bacteria and their products (pathogen-associated molecular patterns, PAMPs), including lipopolysaccharides, phospholipids, and bacterial DNA, can be recognized by paracrine regulation receptors (for example, TLR2 and TLR4) on the surface of macrophages, neutrophils, dendritic cells, and even enterocytes [46]. Thus, intestinal macrophages and dendritic cells, which are part of the intestinal microbiota, can detect pathogenic microorganisms in the intestinal lumen using transepithelial dendrites (TED) [47]. Consequently, pathogenic microorganisms cause a "cytokine storm" with the release of proinflammatory mediators that lead to local and systemic inflammation in the intestine [46]. The released mediators can lead to increased expression of endothelial adhesion molecules (for example, ICAM, VCAM, E- and P-selectin), which leads to increased influx of neutrophils and monocytes and, in turn, to an increase in the level of proinflammatory cytokines and reactive oxygen species [48]. These cellular reactions enhance vasodilation and cause a high degree of capillary permeability with the development of interstitial edema. The developing local DIC syndrome leads to hypoxia, which, in turn, leads to increased apoptosis and necrosis of enterocytes, and the regeneration of these enterocytes is suppressed. The integrity of enterocytes is disrupted, which can also contribute to bacterial translocation [46].

The consequences of this interaction include impaired permeability of tight junctions and effects on the functions of the immune system both locally and at the systemic level [44, 49].

The predisposition to the realization of such pathological mechanisms is formed with a critical decrease in the number of protective microbiota, which can be induced by inflammation. It has been proven, that already 6 hours after the onset of the inflammatory reaction, the death of up to 90 % of the normal flora represented by anaerobic microorganisms occurs, which as a result leads to the activation of specific pathogens producing proteases that initiate the destruction of the mucous barrier of the intestinal epithelium [49, 50]. According to J.C. Alverdy, et al. (2017), the mechanism of this effect has not been fully studied and requires further research [49].

Consequently, it can be concluded, that the microbiota colonizing the layer of intestinal mucus acts as a key regulator of the functional state of both epitheliocytes and populations of immunocompetent cells of the intestinal wall, thereby maintaining non-inflammatory homeostasis. Numerous experimental data indicate that a violation of the integrity of the mucous barrier in combination with a decrease in the number of protective autochthonous microbiota, a decrease in the synthesis of antimicrobial peptides such as lysozyme, defensins and immunoglobulin A [24, 51], create the prerequisites for the development of severe complications caused by facilitated adhesion and subsequent translocation of pathogens into

the abdominal cavity and systemic hemo- and lymph flow [21, 52, 53].

Thus, the development of purulent inflammation of the peritoneum induces a pronounced imbalance of microbial populations, characterized by a change in their quantitative ratio and atypical distribution along the parts of the gastrointestinal tract. In the context of enteral insufficiency associated with abdominal sepsis, the dynamics of these disorders is divided into four successive phases. Initially, a sharp decrease in the number of obligate symbionts in their typical biotopes occurs. The second phase is characterized by a dysbiotic shift. A change in autochthonous microflora occurs with a dominant increase in populations of opportunistic bacteria such as *Escherichia* spp., *Klebsiella* spp., *Lactobacillus* spp., *Campylobacter* spp., and *Enterococcus* spp. Subsequently, the migration of autochthonous microflora to the proximal parts of the intestine is noted, which are normally characterized by low microbial density, which is defined as the phenomenon of proximal microbial contamination. In conclusion, in microbial associations that have colonized unusual biotopes, pathogenicity signs are formed and expressed, potentiating the maintenance of the septic state [21]. The pathological process is characterized by the development of pronounced dysbiosis, manifested by a decrease in the diversity of autochthonous microbiota, the dominance of opportunistic species and an increase in their virulent potential [12, 54]. These changes lead to a violation of immune homeostasis and dysfunction of interaction between the epithelium and the microbiome [12]. The mucous layer exfoliates from the wall of the colon, which leads to direct contact of pathogenic microflora with the intestinal epithelium [55].

It is at this stage that the intestinal microbiota or its products, such as molecular damage markers (DAMPs) in the lymphatic ducts and endotoxins in the portal blood, can cause dysfunction of distant organs in patients with impaired or dysfunctional gastrointestinal tract and lead to the development of multiple organ failure and abdominal sepsis [56].

D.S Arshin, et al. (2021) analyzed the results of treatment of 233 patients with diffuse secondary purulent peritonitis complicated by EIS. It was established that as EIS progresses in patients with diffuse peritonitis, the enteral pathobiome changes: against the background of a decrease in the growth of saprophytic microflora (*Staphylococcus saprophyticus* and *Escherichia coli* by more than 2 times), a significant increase in pathogenic isolates (*Klebsiella* spp., *Candida*, *Clostridium* spp., and *Citrobacter* ($p \leq 0.05$)) is noted, especially *Staphylococcus aureus*, and the number of antibiotic-resistant forms increases exponentially [57].

In our opinion, in decompensated EIS under conditions of diffuse peritonitis, one cannot underestimate the features of the enteral pathobiome and it is necessary to take this into account when choosing therapy for such a severe category of patients.

CONCLUSION

Purulent peritonitis leads to complex structural and functional disorders in the intestinal wall, contributing in most cases to the development of enteral insufficiency. The intestine becomes the “engine of thanatogenesis” of abdominal sepsis and multiple organ failure. Against the background of increased apoptosis of enterocytes, suppression of the proliferative activity of crypts occurs, the absorption function is disrupted, and a systemic inflammatory response develops with an increase in paracellular permeability. Simultaneously, there is a disintegration of functional interaction in the system “intestinal epithelium-microbiota-immune system”, where each of the components makes a significant contribution to the damage of the intestinal barrier, forming a vicious circle of progression of the pathological process.

In recent years, significant progress has been achieved in understanding the complex mechanisms of regulation of the intestinal barrier and intestinal permeability. The analysis of current literature data confirms the indisputable relevance of the problem of enteral insufficiency syndrome.

At the same time, no one doubts that the intestinal tract is one of the main causes of the development of abdominal sepsis. However, to establish complex pathogenetic relationships between immune reactions, inflammation, intestinal microbiota and the integrity of the intestinal barrier, further research is needed, including experimental and translational studies. The fact of an increase in the permeability of the intestinal wall preceding the manifestation of pathological processes in the intestine is fundamentally important. In this regard, the identification of agents capable of preventing the dysfunction of the intestinal barrier and reducing its permeability opens prospects for the development of new therapeutic strategies aimed at strengthening the barrier function of the intestine.

Conflicts of interest

The authors declare no conflicts of interest.

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