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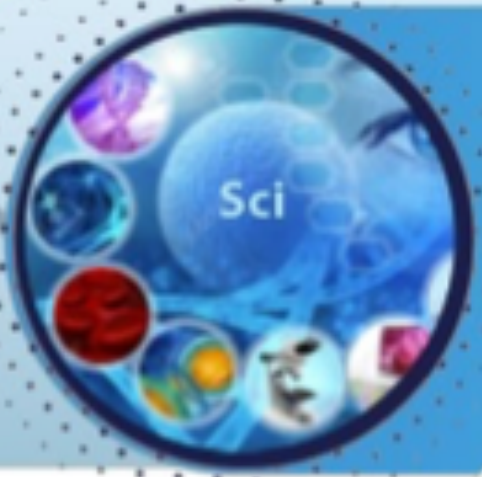

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ABDOMINAL SEPSIS – Problems That Need to Be Addressed

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ABSTRACT

This review scientific article presents current information on the etiology, pathogenesis, diagnosis, clinical manifestation and treatment of abdominal sepsis. Common etiologies of abdominal sepsis in developed countries include appendicular process perforation, cholecystitis, perforated gastrointestinal cancer, and diverticulitis. Peritonitis is one of the leading causes of abdominal sepsis. Despite the fact that there are many studies on peritonitis, in general, this problem is still global, since peritonitis can provoke the development of abdominal sepsis.

Keywords: abdominal sepsis, peritonitis, diagnosis, treatment

Epidemiology and etiology

Abdominal sepsis is egalitarian because it remains a potential health threat to all people of all ages, races, and socioeconomic groups, regardless of how healthy. Worldwide, the combined burden of all pathologies that cause abdominal sepsis is enormous. Abdominal sepsis, which affects both developing and developed countries, is a huge source of lost lives, livelihoods and resources. Using data from the Global Prevalence Survey [8, 21].

B. Stewart et al. reported 896,000 deaths, 20 million years of life lost, and 25 million disability-adjusted life years lost per year, associated with a total of 11 general surgical emergencies [23].

The magnitude of DALYs lost due to this disease is also staggering [46].

The overall incidence of abdominal sepsis is difficult to estimate, but large-scale epidemiological studies indicate that abdominal sepsis accounts for 1% of all hospital visits and is the second most important type of sepsis worldwide [27].

Diffuse peritonitis as a cause of abdominal sepsis in any form is a poor prognostic indicator, with mortality as high as 20% in some studies [35].

Since many patients with abdominal sepsis are present in extreme cases and require a long stay in the intensive care unit, the economic importance of this problem is very relevant.

At the beginning of the 20th century, sepsis was recognized as a dangerous disease, since it leads to dysfunction of vital organs, which is based on the mechanism of the unregulated reaction of the macroorganism to the introduced microorganisms.

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The treatment of patients with abdominal sepsis is a more complex problem, since the focus of the disease is located in the abdominal cavity, inside which there is an inflammatory process caused by the primary disease. At the same time, the "aggressive" methods used to treat peritonitis are often accompanied by the development of aggravated morphofunctional disorders, primarily of the entire gastrointestinal tract with an increase in intracavitary pressure, intestinal paresis, etc.

In addition to this, severe pathophysiological transformations of both substrate and hemolytic nature develop, with aggravation of the patient's condition. In addition, as the process of intestinal dysfunction is underway, the consequences of the primary disease, progressive intra-abdominal hypertension and systemic vasomotor changes, rapidly induce pathological intestinal microflora with multiple but still poorly understood consequences for the macroorganism. In such settings, the task of clinicians is to make the correct diagnosis and assess the extent of surgical and conservative treatments required not only to correct or mitigate the primary pathology (source control), but also to assess the patient's subsequent response, in particular appropriate support of organ function during the treatment of abdominal sepsis.

In addition to standard laparotomy, there are now many less invasive techniques for the potential treatment of primary pathology, so that great skill and experience are required for each unique patient. However, there are fewer options for treating the most severe cases of abdominal sepsis caused by secondary peritoneal pathologies without any pharmacological therapy, but only with the support of intensive care.

Leaving the abdomen open for better peritoneal debridement and lowering intra-abdominal pressure is a therapeutic adjunct that is increasingly used and applicable in any health care setting, even at the district level, especially in cases where intensive care is required. However, there is no evidence to unequivocally support this strategy, which forms the basis for research into the efficacy of treatment for abdominal sepsis currently being conducted on a global basis.

Peritonitis has been a life-threatening and ominous phenomenon throughout human history. Mentions of peritonitis can be found in the ancient Egyptians [6].

Today, the classification of peritonitis can be divided into primary, secondary, and tertiary forms. Each of these types of peritonitis has typical clinical manifestations and accompanying scenarios. However, in practice, there are many nuances to consider. One of the main points of this is the development of abdominal sepsis, which leads to multiple organ failure and death of the patient.

A conceptual framework for understanding the incredibly complex and rapidly changing aspects of the inflammatory response in abdominal sepsis includes the theory that the acute pro-inflammatory response is supplanted by a mixed anti-inflammatory response with balanced pro-inflammatory and anti-inflammatory cytokines. This is followed by a syndrome of anergic, compensatory anti-inflammatory response, as a result of which the patient becomes susceptible to secondary infectious complications [29].

Primary peritonitis is defined as spontaneous bacterial contamination (infection) of the peritoneal cavity. This type of peritonitis requires the presence of a certain bacterial environment in the abdominal cavity. In particular, ascites secondary to cirrhosis or peritoneal dialysate in end-stage renal disease provide a bacterial growth medium that can progress to disseminated infection after infection.

In hospitalized patients with cirrhosis, the overall prevalence of bacterial infections is 32–34%, a quarter of which are patients with abdominal infection [48].

After infection, the risk of recurrence within 1 year without prophylaxis is 20–24% [24].

Patients undergoing peritoneal dialysis suffer from spontaneous infection of the abdominal cavity on average once every 2 years [36].

The main mechanism of infection of ascitic fluid depends on the cause of the accumulation of fluid in the abdomen. This is reflected in the microbiology of the infected fluid.

Usually, the infection in primary peritonitis consists of a single, dominant type of bacteria. Cirrhotic ascites is most commonly infected by gram-negative or enterococcal species of microorganisms through bacterial translocation from the intestine.

The abdominal cavity of patients with permanent drains for peritoneal dialysis is more likely to become infected with staphylococcus, Pseudomonas aeruginosa, or pneumococcus, that is, as a result of direct infection through the drainage (catheter) itself by the patient's skin microflora [36].

The main methods of treatment of primary peritonitis are the early use of empiric antibacterial therapy, followed by transfer to a scheme for the identified sensitivity of pathogenic microflora to the antibacterial drug.

Secondary peritonitis is defined as lesions of the abdominal cavity caused by direct contact with the source of infection [39].

This is most often due to structural or functional impairment of the gastrointestinal tract, and therefore the bacterial landscape in secondary peritonitis is usually polymicrobial.

While perforation of a hollow organ in the gastrointestinal tract causes direct entry of contents into the abdominal cavity, secondary peritonitis can also be seen due to intestinal ischemia, or volvulus.

In general, although "peritonitis" encompasses a wide range of pathologies in the abdominal cavity, the actual significance and consequences for morbidity and mortality as a whole correlate with the potential for a provoking condition of abdominal sepsis.

Tertiary peritonitis, or "continuing peritonitis," is poorly defined, misunderstood, and possibly of historical origin.

As recently as 2005, it was defined as "peritonitis that persists or recurs within 48 hours of apparently successful treatment of primary or secondary peritonitis" [5].

It has been associated with the observed transition from gram-negative and gut bacteria to nosocomial microbes such as Enterobacter, Enterococcus, Acetinobacter, Citrobacter, Pseudomonas, and various fungal species [26].

The clinical consequences of tertiary peritonitis are severe and often fatal, with a mortality rate of 30–64% in some cases [28, 33].

Clinically, it is most often manifested by a long-term syndrome, a systemic inflammatory response, despite the effective therapy of the provoking pathology that causes secondary peritonitis. Often, the diagnosis is made after repeated trips to the operating room when ineffective treatment of secondary peritonitis is suspected.

Effective treatment of tertiary peritonitis is multifaceted, although it is described as the limit of surgical treatment of severe secondary peritonitis [4, 28].

Patients suffering from tertiary peritonitis are often comorbid, malnourished, and with profound and marked metabolic disorders.

Treatment measures often include admitting the patient to an intensive care unit, prescribing broad-spectrum antibiotics, and ensuring that the source is controlled. However, pathogens cultured from the abdomen may be signs rather than the cause of critical illness [28].

Computed tomography imaging should confirm the absence of an intra-abdominal abscess, anastomosis leakage, or the impossibility of primary correction that can be managed surgically. Unfortunately, by definition, there is no clear direction. As a rule, only serous-hemorrhagic exudate is found during repeated surgery, in which the isolated microorganisms can be cultured [28].

It should be noted that the classical descriptions of tertiary peritonitis date back long before the critical importance of the human microbiota and the consequences of their pathological translocation in the development of abdominal sepsis were understood.

To the best of our knowledge, the observations and theories associated with tertiary peritonitis have not been updated to include either the current understanding of dysbiosis or the concept of systemic inflammatory response syndrome.

Dysbiosis determines quantitative and functional changes in the intestinal microflora that alter immune responses, destabilize intestinal homeostasis, and are associated with the overgrowth of pathogenic microorganisms [15].

During the acute purulent-inflammatory process of the abdominal cavity organs, there is a catastrophic loss of microbial diversity and induction of a state of severe dysbacteriosis [30].

The loss of the normal microbial landscape is accompanied by an over-representation of potentially pathogenic organisms, which, combined with the loss of integrity of the intestinal barrier, leads to a greater potential for microbial translocation to extraintestinal sites [21].

It goes without saying that the risk factors and clinical conditions in which tertiary peritonitis was previously described will almost certainly be valid, in which the critically ill patient will exhibit radically pathological dysbiosis and probably systemic inflammatory response syndrome. In this case, further non-targeted broad-spectrum antibiotic therapy can have catastrophic consequences.

This view remains speculative, as there is still no reliable data to support it, but it is a field of knowledge that, in our opinion, deserves urgent study and a comprehensive review of various theoretical models.

With prompt access to elective surgical services, screening programs, and prophylactic medications (e.g., proton pump inhibitors), treatment outcomes for patients with abdominal sepsis are steadily improving in developed countries [3].

However, despite significant advances in many areas of global health, the provision of surgery worldwide in low- and middle-income countries has stagnated and sometimes even regressed. Case fatality rates remain

high for common, easily treatable diseases, including appendicitis and hernia [25].

The Lancet Commission on Global Surgery concluded that surgery is "an indivisible, indispensable part of health care and that surgical and anesthesiological care should be an integral component of the national health system in countries at all levels of development" [25].

Thus, the treatment of abdominal sepsis, which is applicable in all parts of the globe, deserves special attention.

This consideration is crucial. Even in developed countries, a large proportion of the population lives far from surgical care. Time to intervention is a proven predictor of outcome in secondary peritonitis [37].

Studies conducted in developed countries with relatively appropriate access to surgical services show that the mortality rate for abdominal sepsis is 10.5% [3].

Patients with long-term abdominal sepsis are more likely to experience severe metabolic abnormalities and wasting. This results in a long stay in the intensive care unit and overall adverse outcomes. This is exacerbated by an increase in the incidence of predisposing pathologies such as H. pylori, tuberculosis, and other infectious etiologies [23, 34, 37].

On the Pathogenesis of Abdominal Sepsis

From a patient-centered practical perspective, peritonitis is most deserving of consideration as a marker of impending abdominal sepsis. Intra-abdominal infection is the most common cause of abdominal sepsis [37].

The high incidence of abdominal sepsis is accompanied by a high mortality rate, which can range from 7.6% to 36.0% [3].

A number of studies have shown that the worsening prognosis in secondary peritonitis is due to some direct factors. Candida infection, severe organ dysfunction (SOFA≥7), severe pre-existing comorbidities, inadequate source control, and inappropriate antibiotic prescription play a role [1, 49].

Once a patient meets the criteria for septic shock, cardiovascular instability, sepsis-associated coagulopathy, and worsening organ failure, the mortality rate reaches more than 50% or even 80% [16].

In 2016, the definitions of sepsis and septic shock were revised. A third international consensus defines sepsis as a life-threatening organ dysfunction caused by an unregulated host response to infection, emphasizing a critical concept to be evaluated, which is host self-destruction initiated by primary pathology [44].

Organ dysfunction was defined by an increase of 2 or more in the Consistent Organ Failure Rating (SOFA) score.

Previous definitions of sepsis, based on criteria for systemic inflammatory response syndrome in the presence of an infectious source, have been rejected as too focused on the patient's inflammatory response.

Septic shock is defined as "a subgroup of sepsis in which there are particularly profound microcirculation disorders and metabolic disorders associated with a greater risk of death" [42, 44].

This is supported by the need for vasopressors to maintain adequate mean arterial pressure and serum lactate levels of >2 mmol/L after adequate resuscitation.

The patient's body reaction, manifested by sepsis and septic shock, largely dictates the treatment of abdominal infection. Decisions about surgery for stoma formation, for example, depend on the patient's metabolic/physiological status. Thus, a "deeper dive" into the underlying mechanisms of this unregulated, systematic self-destruction is justified.

The relatively recent recognition of the microbial environmental shift in critical disease settings has led to a better understanding of the mechanisms by which multiple organ failure develops. In addition, multidrug-resistant microorganisms have become commonplace around the world.

A study on complicated intra-abdominal infections worldwide has revealed a growing incidence of resistant microorganisms [37].

Between 2002 and 2008, the incidence of extended-spectrum E. coli-producing β-lactamases nearly tripled worldwide [39].

Resistance to Klebsiella pneumoniae is about 20%. Enterococci species, which are considered to be the most common pathogens isolated in nosocomial sepsis, have also shown increasing resistance. Pseudomonas aeruginosa has been identified as an independent risk factor for mortality [50].

Candida infection has also been shown to dramatically increase mortality in patients with abdominal sepsis [2].

As resistance grows, the role of resistant microorganisms plays a larger and larger role in the outcomes of critically ill patients with abdominal sepsis.

An unregulated immune response is a pathophysiological factor that leads to the effects of sepsis on target organs. In the presence of infection, microbial pathogen-associated molecular patterns are formed. In the case of trauma, pancreatitis, or other non-infectious lesions, sys-

temic inflammatory responses may be triggered by the recognition of molecular patterns associated with the injury. These inflammatory mediators activate toll-like receptors on the signaling cells of the immune system. These macrophages and dendritic cells initiate the inflammatory cascade responsible for the adverse effects of sepsis on target organs.

By activating neutrophils, platelets perform a variety of immune functions in a variety of immune pathways, including inducing the release of extracellular neutrophil traps, stimulating degranulation, releasing leukocyte-activating cytokines (CD40L), increasing leukocyte adhesion, and even directly killing invading pathogens [17].

Secondary peritonitis, being a surgical disease, prepares the infected, physiologically exhausted patient for a massive systemic inflammatory response with a combination of massive intraperitoneal bacterial load and invasive surgery to control the source.

TLRs are pattern recognition receptors expressed on endothelial and immune cells that play an important role in the inflammatory response. In these cells, cascades of protein kinases are activated, promoting the production of pro-inflammatory and anti-inflammatory cytokines. Specifically, IL-6, IL-8, IL-1/β, IL-10, MCP-1, TNF-α, thromboxane A2, HMGB1, and thrombin are among the known effectors and cytokines produced by TLR pathways.

Intra-abdominal contamination and secondary peritonitis are a constant source of pathogen-associated molecular patterns (through spillage of intestinal contents) and through direct injury to internal and abdominal organs. This "multisystem organ failure motor" provides continuous cytokine fuel for the raging systemic response [18].

For example, TNF-α and IL-1 are important pro-inflammatory cytokines. Each has been shown to induce vascular permeability, leading to pulmonary edema and bleeding [31]. IL-6 is a key molecule in initiating a febrile response, activating lymphocytes, and also plays a role in hematopoiesis. It has also been shown to cause myocardial depression [31].

IL-12, interferon-γ, and macrophage migration inhibitor play a role in boosting the regulation of the immune system and also describe deleterious effects on target organs in sepsis. This demonstrates once again that septic shock is more than just a severe infection. Host response is of paramount importance in the response of every patient facing sepsis, with marked differences in the patient's response, based in part on sex, age, and especially genetics.

In the presence of secondary peritonitis, the abdominal cavity is a rich reservoir of inflammatory cytokines. Visceral abdominal injury, peritoneal irritation, and intra-abdominal contamination are potent triggers of the systemic cytokine response. IL-6, IL-8, TNF-α, and IL-1β have been shown to occur in high concentrations in inflammatory ascites following abdominal visceral injury [53].

It has been shown that the translocation of inflammatory cytokine from ascitic fluid to the systemic circulation occurs through the mesenteric lymphatic tracts [7].

The lymphatic capillary network on the diaphragmatic surface is also responsible for the reabsorption of up to 70–80% of fluid from the abdominal cavity [43].

The mesenteric and phrenic lymphatic channels eventually drain into the thoracic duct and into the systemic circulation. Disruption of this inflammatory flow can blunt the systemic inflammatory response and reduce the likelihood of developing acute respiratory distress syndrome and multiple organ dysfunction syndrome [13, 47].

The abdomen and inflammatory exudate become the target for intervention to blunt the systemic effects of intraperitoneal involvement. Many studies have been conducted that have tested the clinical effects of removing or diluting inflammatory exudate in a metabolically depleted septic patient. The premise of these studies is that the removal of inflammatory cytokine from the abdomen inhibits its lymphatic uptake and subsequent systemic circulation.

Diagnosing Abdominal Sepsis

The success of therapeutic measures for abdominal sepsis depends, first of all, on the correct understanding by the clinician of both anatomical, morphological and functional transformations of the pathological process.

In the era of advanced imaging, clinical examination remains important. A patient with diffuse peritonitis requires immediate surgical intervention. However, in the context of the development of a limited purulent-inflammatory process in the abdominal cavity, the clinician has the opportunity to perform diagnostic imaging of the abdominal organs.

Each hollow organ of the abdomen can cause secondary peritonitis, with a variety of pathologies listed for each organ. But there are nuances that are as complex and diverse as the patients themselves. An example is microperforation of sigmoid diverticulitis. At the same time, macroperforation causing massive peritoneal irrita-

tion and a deep systemic reaction with obvious vasomotor changes is clinically obvious and requires urgent laparotomy without further investigation.

However, the same anatomical perforation in an anergic patient with a minor systemic response may require advanced imaging to detect a focus of peritonitis. Microperforation of the same organ with a profound response may require both diagnostic imaging to make a diagnosis and multiple biochemical/hematological tests to assess the body's response to the pathology and make treatment decisions.

Patients may present with various stages of hemodynamic instability, ranging from normal hemodynamics to decompensated shock. Patients may present with leukocytosis, acidosis, and high lactate levels, but this is not necessary for diagnosis.

Although the physical examination is an integral part of the surgical patient's examination, the generally accepted signs may not be present. Less than half of patients with acute abdomen have diffuse peritonitis [27]. Limited peritonitis is much more common.

Physical examination may be unreliable in a steroid-dependent, malnourished, or paralyzed patient. From a biochemical point of view, a complete blood count is probably the most common laboratory test. However, leukocytosis is a non-sensitive (53.5%) and relatively nonspecific (73.7%) finding in abdominal sepsis. In combination with relative lymphopenia, specificity is increased (89.2%), but sensitivity is affected (47.8%) [32].

C-reactive protein, which is largely considered an oversensitive test, also does not correlate with positive intra-abdominal pathology on abdominal computed tomography [32]. However, because of this sensitivity, if the symptoms last more than 24 hours and the C-reactive protein is normal, peritonitis is very unlikely to be the cause of the symptoms.

Ultrasound imaging plays a significant role in the diagnosis of peritonitis, especially in pathologies of the biliary tract, ovaries and uterus. It is often the primary diagnostic test of choice in children and pregnant women. Ultrasound is playing an increasingly prominent role in the diagnosis of appendicitis, while the sensitivity of the method is low (59–78%) and specificity (73–88%) can help improve the physical examination and avoid ionizing radiation [14, 27].

Although ultrasound is by no means an alternative to computed tomography, it can be effectively applied by clinicians at the bedside to supplement a conclusive history or physical exam findings, and we believe it should be adopted by practicing surgeons.

Enhanced CT scans have largely become the diagnostic workhorse in diagnosing peritonitis. In a stable patient with peritonitis, computed tomography interpreted by a consultant radiologist is able to make the correct diagnosis in >90% of cases [52].

This is useful not only when deciding on surgery, but also when planning a surgical approach.

Treatment of abdominal sepsis

Similar to the dual responsibility of diagnosis, the optimal treatment of abdominal sepsis involves both addressing the primary anatomical cause and treating or supporting the affected organs. Ideal results typically require a multidisciplinary effort involving surgeons, radiologists, and the recognition that these are surgical diseases, and the team must be led by a surgeon.

It is critical to ensure the earliest possible control of the sources or management of what is causing abdominal sepsis. Failure to achieve adequate control of sources of sepsis is an independent predictor of mortality [49].

The primary goals of surgery for secondary peritonitis remain the same: stopping bleeding, controlling infection, and making decisions about reconstruction or control of lesions are the main components of emergency laparotomy. Perforated or damaged internal organs must be removed or repaired. Abscesses should be drained. If the decision to reconstruct is made, the well-perfused ends of the intestine should be sutured together with airtight anastomoses.

Numerous questions still remain in the treatment of secondary peritonitis. Stoma formation has long been considered the standard of destructive colon pathology in critically ill patients. However, more recent retrospective literature has shown that anastomosis formation is safe even in the most severely injured patient.

Drainage of the abdomen after laparotomy is a common practice among emergency surgeons. There is a lot of evidence in favor of this practice, but it shows an increase in the duration of hospitalization, the duration of surgery, the incidence of wound infection, and the overall complication rate [19].

Most surgical solutions for secondary peritonitis evolve from etiology to more reflective of the body's physiology and response. Although the disease begins due to a disruption of integrity in the gastrointestinal tract, progressive organ failure is the ultimate cause of

death. Thus, the question of how best to stop or mitigate this progressive organ dysfunction is crucial.

Initial steps in the management of patients with abdominal sepsis include the full range of resuscitation and intensive care options. Fortunately, mass crystalloid resuscitation has fallen out of fashion, giving way to pergulative hypotension and the use of vasoactive agents. Despite the lack of conclusive scientific evidence, the modulation of the "salt tsunami" that has characterized gross over-resuscitation in the recent past appears to be, in our opinion, one of the most profound evolutions in the treatment of abdominal sepsis.

In addition to general supportive care, it is recommended that consideration be given to blocking or removing mediators that contribute to progressive organ damage. The increased recognition of inflammatory cytokines as a driver of organ dysfunction in sepsis has opened the door to new potential treatments for the systemic inflammatory response in sepsis. For example, immunological monoclonal antibody therapy has been developed against TNF-α, IL-1, and MIF. However, antibodies against these cytokines did not show any significant mortality outcomes [10, 54].

Similarly, there have been 100 unsuccessful attempts to manipulate or block single neurotransmitter molecules, but without success [20].

Currently, no human trials of these treatments have been conducted, and their use remains a possibility in the future.

Thus, it appears that other methods will be needed to respond more effectively to the systemic consequences of abdominal sepsis.

Another option to potentially mitigate biotransmitter leakage from the abdomen into the systemic circulation is to leave the abdomen open with a peritoneal negative pressure device. Such a method in severe sepsis has been proposed for early detection and enhanced drainage of any residual infection, control of any persistent source of infection, more effective removal of biotransmitter-rich peritoneal fluid, effective avoidance of abdominal sepsis, and safe provision of delayed gastrointestinal anastomosis [20].

Despite the lack of conclusive evidence of efficacy, the use of laparostomy in abdominal sepsis is increasingly recommended [11, 38, 40].

This includes consensus recommendations from recognized societies such as the World Society of Abdominal Compartment Syndrome and the World Society of Emergency Surgery, which stated that despite the lack of high-quality data, the use of laparostomy may be an important treatment option for severe peritonitis and abdominal sepsis [38], and this position was confirmed in 2018, although the lack of evidence was again highlighted [9].

Kirkpatrick et al. [22] demonstrated in a randomized controlled trial that intraperitoneal negative pressure therapy is associated with mortality compared to a less effective homemade system in patients with abdominal sepsis. However, they did not show any significant differences in biotransmitter levels.

Peritoneal lavage is used in an attempt to "wash out" not only peritoneal impurities, but also to dilute and remove peritoneal cytokines. While most laparotomies will be irrigated at some point, interest is again focused on continuous intraperitoneal lavage, which can be combined with negative peritoneal pressure wound treatment systems. The most recent and largest (though not randomized) experience with this method using isotonic fluid infusion has revealed an increase in complications during the treatment period, but no differences in mortality, entero-atmospheric fistula, or time of opening.

Direct peritoneal debridement is a related method in which hypertonic dialysis fluid is continuously injected into the abdominal cavity [51].

The perceived mechanism of action is related to fluid hypertonicity. Hyperosmolarity is thought to dilate intestinal arterioles and improve visceral blood flow by counteracting intestinal ischemia [51].

A randomized clinical trial conducted by Smith et al. [45] in traumatized patients undergoing injury repair surgery with massive transfusions showed improved fascial closure, fewer days in the intensive care unit, shorter ventilation, and insignificant trends in 30-day mortality. Despite these findings, the technique of direct peritoneal debridement does not seem to have taken root.

Cytokines in the abdomen have a clear effect on the systemic response of abdominal sepsis. Disruption of the transmission of these intraperitoneal cytokines into the systemic circulation may be beneficial, although the ideal mechanism to achieve this goal remains to be elucidated.

Although unexplained, the significantly improved survival with more effective treatment of abdominal sepsis using safer devices to temporarily close the abdomen appears to be worthy of further research, especially since there appears to be a large clinical application without conclusive scientific evidence based on it. In this regard, a multicenter, multinational, prospective, randomized trial has recently been launched worldwide to address

this issue in patients requiring source-controlled laparotomy for severe complicated abdominal sepsis [20].

Despite the fact that intensive care services are critically limited worldwide, the use of laparostomy from a logistical point of view is possible even in rudimentary intensive care settings [12, 41].

So, if this method does address systemic disorders, sepsis, and multiple organ failure, then it could be a truly effective surgical strategy. Therefore, research in this area of treatment of abdominal sepsis, the source of which is secondary peritonitis, can lay the foundation for solving critical questions for improving the treatment outcomes of patients with abdominal sepsis.

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

Availability of data and material - Available

Competing interests - No

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ABDOMINAL SEPSIS – BARTARAF ETILISHI KERAK BO'LGAN MUAMMOLAR

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ABSTRAKT

Ilmiy maqolada abdominal sepsisning etiologiyasi, patogenezi, diagnostikasi, klinik ko'rinishi va davosi bo'yicha joriy ma'lumotlar keltirilgan. Rivojlangan mamlakatlarda abdominal sepsisning umumiy etiologiyalariga appendikulyar usimta perforatsiyasi, xoletsistit, oshqozon yara perforatsiyasi, divertikulitlar kiradi. Peritonit - abdominal sepsisning asosiy sabablaridan biri hisoblanadi. Peritonit bo'yicha ko'plab tadqiqotlar mavjud bo'lishiga qaramay, umuman olganda bu muammo hali ham globaldir, chunki peritonit abdominal sepsis rivojlanishiga sabab bo'lishi mumkin.

Tayanch iboralar: abdominal sepsis, peritonit, diagnostika, davolash

АБДОМИНАЛЬНЫЙ СЕПСИС – ПРОБЛЕМЫ, ТРЕБУЮЩИЕ РЕШЕНИЯ

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АБСТРАКТ

В данной обзорной научной статье представляются современные сведения относительно Этиологии, патогенеза, диагностики, клинического проявления и лечения абдоминального сепсиса. Общая этиология абдоминального сепсиса в развитых странах включает в себя перфорацию аппендикулярного отростка, холецистит, перфоративный рак желудочно-кишечного тракта и дивертикулит. Перитонит – является одной из ведущих причиной развития абдоминального сепсиса. Несмотря на то, что существуют множество исследований по перитониту, в целом данная проблема все еще остается глобальной, поскольку перитонит может провоцировать развитие абдоминального сепсиса.

Ключевые слова: Абдоминальный сепсис, перитонит, диагностика, лечение