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A MODERN VIEW OF THE CAUSES AND MECHANISMS OF THE DEVELOPMENT OF ACUTE DISSEMINATED PERITONITIS

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Abstact

Despite the fact that the diagnosis of acute peritonitis has improved in recent years, a wide range of antibiotics is available, and minimally invasive methods of surgical treatment have been implemented, the mortality rate for this disease is high and ranges from 12.5 % to 39.2 %.

The purpose of the work was to analyze data from literary sources regarding the classification of acute peritonitis, to summarize the causes and mechanisms of its development among the adult population.

Analytical and bibliosemantic research methods were used in the work.

It is known that according to the nature of penetration of microflora into the abdominal cavity, peritonitis is divided into primary, secondary and tertiary. According to the clinical course, peritonitis is divided into acute, subacute and chronic. According to the nature of the exudate in the abdominal cavity, serous, serous-purulent, fibrinous, fibrinous-purulent, purulent, hemorrhagic, fecal and purulent peritonitis are distinguished. To date, the existing classifications are constantly being improved and have both practical and academic significance.

It has long been known that the cause of unsatisfactory results of surgical treatment of patients with acute disseminated peritonitis can be the development of intra-abdominal hypertension with subsequent development of compartment syndrome and multiple organ failure. The development of multiple organ failure is the leading cause of death in patients with acute surgical pathology. Abdominal compartment syndrome plays a big role in this, the mortality in which reaches very significant figures — 42-68 % and without treatment approaches 100 %.

Therefore, the development of acute peritonitis is due to numerous reasons (in particular, acute destructive appendicitis, cholecystitis, perforated ulcer of the stomach or duodenum, intestinal obstruction, tumor processes, gynecological pathology, abdominal injuries, etc.), which in turn causes the absence of a generally recognized justified classification.

Key words: acute generalized peritonitis; classification of acute peritonitis; multiple organ failure; intra-abdominal hypertension; compartment syndrome.

Introduction. Peritonitis is an inflammation of the parietal and visceral leaves of the peritoneum, in most cases caused by bacterial, fungal, viral infection, foreign bodies, accompanied by a specific symptom complex, which complicates its differential diagnosis with other diseases [1].

Despite the fact that diagnostics have improved in recent years, a wide range of antibiotics is available, and minimally invasive methods of surgical treatment have been implemented, the mortality rate of this disease is high and ranges from 12.5% to 39.2% [1-4]. It is worth noting that the mortality rates for acute disseminated peritonitis in children reach 22 % [5-6].

The aim of the study: to analyze data from literary sources regarding the classification of acute peritonitis, to summarize the causes and mechanisms of its development among the adult population.

Materials and methods. An analysis of literary sources on the classification of acute peritonitis was carried out, the causes and mechanisms of its development among the adult population were summarized. Analytical and bibliosemantic research methods were used.

Research results and their discussion. The first classification of peritonitis was developed in 1886 by Yohan Mikulich. Since that time, scientists have proposed a number of classifications of peritonitis, but the attempt to create a single unified classification has not been successful. It should be understood that any classification is conditional and cannot fully reflect all aspects of the development of acute peritonitis.

According to the nature of penetration of microflora into the abdominal cavity, peritonitis is divided into primary, secondary and tertiary [7-8]. With primary peritonitis, there is no visible focus of infection in the abdominal cavity [9-10]. Microflora penetrates into the abdominal cavity mostly by hematogenous and lymphogenous routes. In a number of cases, the source of primary peritonitis is the coccal flora of the female genital tract, in the case of liver cirrhosis, the flora of the colon, which enters the peritoneal cavity by bacterial translocation [11-12]. Primary peritonitis occurs in approximately 1-3 % of all observations in patients undergoing chronic peritoneal dialysis [13]. Secondary peritonitis occurs from foci of infection located in the abdominal cavity [14-15]. The most frequent causes of secondary peritonitis are acute destructive appendicitis (12.7-66.8 %), cholecystitis (1.9-2.1 %), perforated ulcer of the stomach or duodenum (8.7-6.2 %), intestinal obstruction (4.3 %), tumor processes (4.6-6.7%), gynecological pathology (2.3-17 %), abdominal injuries (3.6-46 %) [16-19].

The term tertiary peritonitis refers to inflammation of the peritoneum, which has a "recurrent" nature, is characterized by few symptoms and the difficulty of diagnosis [20]. Tertiary peritonitis is considered one of the most complex forms of abdominal sepsis with high mortality rates [21]. It develops against the background of long-term treatment of patients with secondary intoxication and often immunodeficiency of various etiologies [22].

According to the clinical course, peritonitis is divided into acute, subacute and chronic. According to the nature of the exudate in the abdominal cavity, serous, serous-purulent, fibrinous, fibrinous-purulent, purulent, hemorrhagic, fecal and purulent peritonitis are distinguished.

In 1971, K.S. Simonyan proposed to classify peritonitis based on the factor of the affected area of the peritoneum into local (demarcated, non-demarcated) and widespread (diffuse, spilled) [23]. Diffuse peritonitis localized directly near the source of infection of one anatomical part of the abdominal cavity is called local. Diffuse peritonitis, which spreads to more than several anatomical sites, is called widespread.

To characterize the degree of severity of peritonitis, it is advisable to preserve the principle of staging of the inflammatory process of the peritoneum, where is distinguished the

early, late and final stages of peritonitis [24]. According to the classification of Simonyan K.S. (1971), distinguish three stages in the course of acute peritonitis: reactive (first 24 hours), toxemic (24-72 hours), terminal (more than 72 hours).

To date, the existing classifications are constantly being improved and have both practical and academic significance.

There is an opinion that the cause of peritonitis can be the entry of any microorganism into the abdominal cavity, but the conditions necessary for the manifestation of peritonitis for each pathogen are individual [25].

Currently, evolutionary changes are taking place in the understanding of the pathogenesis of acute disseminated peritonitis. The key links naturally attract the attention of researchers and clinicians: oxidative stress (OS), systemic inflammatory response syndrome (SIRS), endothelial dysfunction, intestinal (enteric) failure and intra-abdominal hypertension [26-29].

It has long been known that the cause of unsatisfactory results of surgical treatment of patients with acute disseminated peritonitis can be the development of intra-abdominal hypertension with subsequent development of compartment syndrome and multiple organ failure.

The development of multiple organ failure is the leading cause of death in patients with acute surgical pathology. Abdominal compartment syndrome plays a big role in this, the mortality in which reaches very significant figures -42-68% and without treatment approaches 100%.

An increase in intra-abdominal pressure above 10 mm Hg within 1-2 days leads to a fatal outcome in 3-7 % of cases, and when this value increases to more than 35 mm Hg within 6-7 hours leads to a fatal outcome in 100 % of cases. Factors contributing to the development of abdominal compartment syndrome are hypothermia below 33 °C, acidosis with blood pH less than 7.2, large volumes of hemotransfusion (more than 10 doses per day), coagulopathy, sepsis [30-31]. The classification of intra-abdominal hypertension, based on the levels of intra-abdominal pressure, currently has the following form: I degree – 12-15 mm Hg, II degree – 16-20 mm Hg, III degree – 21-25 mm Hg, IV degree – more than 25 mm Hg [32]. Abdominal compartment syndrome is currently defined as a persistent increase in intra-abdominal pressure to a level of more than 20 mm Hg, which is associated with the manifestation of organ failure / dysfunction.

It is important that, unlike the phenomenon of intra-abdominal hypertension, abdominal compartment syndrome does not require classification according to the level of

intra-abdominal hypertension, since this syndrome in modern literature is represented by the phenomenon of "all or nothing" (that is, with the development of abdominal compartment syndrome at any the degree of intra-abdominal hypertension, further increase of intra-abdominal pressure has no significance) [33]. It is very important to measure the perfusion pressure of the abdominal cavity, which determines the severity and prognosis in the syndrome of intra-abdominal hypertension. It is the difference between mean arterial pressure and intra-abdominal pressure. The level of perfusion pressure is less than 60 mm Hg correlates with patient survival [34].

Conclusions. The development of acute peritonitis is due to numerous reasons (in particular, acute destructive appendicitis, cholecystitis, perforated ulcer of the stomach or duodenum, intestinal obstruction, tumor processes, gynecological pathology, abdominal injuries, etc.), which in turn causes the absence of a generally recognized justified classification.

Further, deeper study of the causes and mechanisms of the development of acute peritonitis will make it possible to develop unified approaches to the treatment of this pathology, as well as to develop effective preventive measures for the treatment of complications of acute peritonitis.

References

- 1. Capobianco A, Cottone L, Monno A, Manfredi AA, Rovere-Querini. The peritoneum: healing, immunity, and diseases. P. J Pathol. 2017;243(2):137-147.
- 2. Ross JT, Matthay MA, Harris HW. Secondary peritonitis: principles of diagnosis and intervention. BMJ. 2018;361:k1407.
- 3. Pörner D, Von Vietinghoff S, Nattermann J, Strassburg CP, Lutz P.Advances in the pharmacological management of bacterial peritonitis. Expert Opin Pharmacother. 2021;22(12):1567-1578.
- 4. Li PK, Chow KM, Cho Y, Fan S, Figueiredo AE, [et al.]. ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. Perit Dial Int. 2022;42(2):110-153.
 - 5. Hervieux E. Acute peritonitis in children. Rev Prat. 2020;70(5):e177-e182.
- 6. Suzuki R, Sato M, Murakoshi M, Kamae C, Kanamori T, [et al.]. Eosinophilic peritonitis in children on chronic peritoneal dialysis. Pediatr Nephrol. 2021;36(6):1571-1577.
- 7. Kedra B. Acute peritonitis--current clinical classification. Przegl Lek. 1987;44(8):608-10.

- 8. Bassetti M, Eckmann C, Giacobbe DR, Sartelli M, Montravers P. Post-operative abdominal infections: epidemiology, operational definitions, and outcomes. Intensive Care Med. 2020;46(2):163-172.
- 9. Diaconescu B, Uranues S, Fingerhut A, Vartic M, Zago M, [et al.]. The Bucharest ESTES consensus statement on peritonitis. Eur J Trauma Emerg Surg. 2020;46(5):1005-1023.
- 10. Wittmann DH, Schein M, Condon RE. Management of secondary peritonitis. Ann Surg. 1996;224(1):10-8.
- 11. Capobianco A, Cottone L, Monno A, Manfredi AA, Rovere-Querini P. The peritoneum: healing, immunity, and diseases. J Pathol. 2017;243(2):137-147.
- 12. Montravers P, Assadi M, Gouel-Cheron A. Priorities in peritonitis. Curr Opin Crit Care. 2021;27(2):201-207.
- 13. Banno T, Shima H, Kawahara K, Okada K, Minakuchi J. Risk factors for peritoneal dialysis withdrawal due to peritoneal dialysis-related peritonitis. Nephrol Ther. 2021;17(2):108-113.
- 14. Unusual peritoneal dialysis fluid culture: Think secondary peritonitis. Cheema RS, Manandhar AK, Willis AP, David MD, Kamesh L. Perit Dial Int. 2021 Jan;41(1):127-128.
- 15. Biocompatible dialysis fluids for peritoneal dialysis. Htay H, Johnson DW, Wiggins KJ, Badve SV, Craig JC, Strippoli GF, Cho Y. Cochrane Database Syst Rev. 2018 Oct 26;10(10):CD007554.
- 16. Szeto CC, Li PK. Peritoneal Dialysis-Associated Peritonitis. Clin J Am Soc Nephrol. 2019;14(7):1100-1105.
- 17. Spalding DR, Williamson RC. Peritonitis. Br J Hosp Med (Lond). 2008;69(1):M12-5.
- 18. Culp WT, Holt DE. Septic peritonitis. Compend Contin Educ Vet. 2010;32(10):E1-14.
- 19. Lippi G, Danese E, Cervellin G, Montagnana M. Laboratory diagnostics of spontaneous bacterial peritonitis. Clin Chim Acta. 2014;430:164-70.
 - 20. Germer CT, Eckmann C. Peritonitis. Chirurg. 2016;87(1):3-4.
- 21. Vega-Pérez A, Villarrubia LH, Godio C, Gutiérrez-González A, Feo-Lucas L, [et al.]. Resident macrophage-dependent immune cell scaffolds drive anti-bacterial defense in the peritoneal cavity. Immunity. 2021;54(11):2578-2594.e5.
- 22. Stocker F, Reim D, Hartmann D, Novotny A, Friess H. Clinical Manifestations and Therapeutic Implications of Peritonitis. Ther Umsch. 2020;77(4):171-176.
 - 23. Simonyan KS. Peritonitis. M.: Medicine. 1971: 296 pp.

- 24. Gostischev VK, Sashyn VP, Avdovenko AL. Peritonitis. M.: Medicine. 2002:224 pp.
- 25. Primary bacterial peritonitis in dogs and cats: 24 cases (1990-2006). Culp WT, Zeldis TE, Reese MS, Drobatz KJ. J Am Vet Med Assoc. 2009 Apr 1;234(7):906-13.
- 26. Pathophysiological changes in peritonitis. Delibegovic S. Med Arh. 2007;61(2):109-13.
- 27. Song DS. Spontaneous Bacterial Peritonitis. Korean J Gastroenterol. 2018;72(2):56-63.
- 28. Dever JB, Sheikh MY. Review article: spontaneous bacterial peritonitis -bacteriology, diagnosis, treatment, risk factors and prevention. Aliment Pharmacol Ther. 2015;41(11):1116-31.
- 29. Velkey B, Vitális E, Vitális Z. Spontaneous bacterial peritonitis. Orv Hetil. 2017;158(2):50-57.
- 30. Töns C, Schachtrupp A, Rau M, Mumme T, Schumpelick V. Abdominal compartment syndrome: prevention and treatment. Chirurg. 2000;71(8):918-26.
- 31. Surace A, Ferrarese A, Marola S, Cumbo J, Valentina G, [et at.]. Abdominal compartment syndrome and open abdomen management with negative pressure devices. Ann Ital Chir. 2015;86(1):46-50.
- 32. Dietz UA, Baur J, Piso RJ, Willms A, Schwab R, [et al.]. Laparostoma-Avoidance and treatment of complications. Chirurg. 2021;92(3):283-296.
- 33. Perova-Sharonova VM, Albokrinov AA, Fesenko UA, Gutor TG. Effect of intraabdominal hypertension on splanchnic blood flow in children with appendicular peritonitis. J Anaesthesiol Clin Pharmacol. 2021;37(3):360-365.
- 34. Sakka SG. The patient with intra-abdominal hypertension. Anasthesiol Intensivmed Notfallmed Schmerzther. 2016;51(1):8-16.