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Increased Intra-Abdominal Pressure as One of the Links in the Pathogenesis of Secondary Diffuse Purulent Peritonitis

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Aim: to present literature data on the importance of intra-abdominal hypertension in the pathogenesis of peritonitis.
Key points. Secondary purulent peritonitis is a severe and fairly common form of abdominal infection with high mortality. According to statistics, hospitalization of patients with signs of local or diffuse peritonitis is more than 15 % among patients with acute surgical pathologies. One of the factors in the development of complications and an unfavorable prognosis for this disease is increased intra-abdominal pressure. To measure this indicator, a Foley catheter inserted into the bladder is used in medical practice. According to reference values, the optimal level of intra-abdominal pressure does not exceed 5 mmHg. A persistent increase in pressure of 12 mmHg and above indicates the development of intra-abdominal hypertension. An increase in the level of intra-abdominal pressure indicates the progression of the inflammatory destructive process in the abdominal cavity and contributes to the development of multiple organ failure with subsequent fatal outcome with untimely treatment. With an indicator exceeding 20 mmHg, there is a risk of developing abdominal compartment syndrome. This condition is characterized by increased pressure in two or more anatomical areas, which leads to decreased blood flow and subsequent tissue hypoxia. Intra-abdominal hypertension also increases the risk of developing postoperative peritonitis in patients who have undergone laparotomy.
Conclusion. To predict the course of secondary diffuse purulent peritonitis, reduce the risk of complications and mortality, it is necessary to focus on measuring intra-abdominal pressure as a mandatory manipulation in surgical patients. Despite its general availability and ease of implementation, this technique allows assessing the severity of organ dysfunctions.
Keywords: intra-abdominal hypertension, diffuse purulent peritonitis, intra-abdominal pressure, abdominal compartment syndrome
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Повышенное внутрибрюшное давление как одно из звеньев в патогенезе вторичного распространенного гнойного перитонита

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Цель: представить данные литературы о значении внутрибрюшной гипертензии в патогенезе перитонита.
Основные положения. Вторичный гнойный перитонит — тяжелая и довольно частая форма абдоминальной инфекции с высокой летальностью. Согласно статистическим данным госпитализация пациентов с признаками местного или разлитого перитонита составляет более 15 % среди пациентов с острыми хирургическими заболеваниями. Одним из факторов возникновения осложнений и неблагоприятного прогноза при данном заболевании служит повышенное внутрибрюшное давление. Для измерения данного показателя в медицинской практике применяется катетер Фолея, установленный в мочевой пузырь. Согласно референсным значениям оптимальный уровень интраабдоминального давления не превышает 5 мм рт. ст. Стойкое повышение давления в 12 мм рт. ст. и выше свидетельствует о развитии внутрибрюшной гипертензии. Нарастание уровня внутрибрюшного давления указывает на прогрессирование воспалительного деструктивного процесса в брюшной полости, а также способствует развитию полиорганной недостаточности с последующим летальным исходом при несвоевременном лечении. При показателе, превышающем 20 мм рт. ст., существует ве-

роятность развития абдоминального компартмент-синдрома. Данное состояние характеризуется повышенным давлением в двух и более анатомических отделах, что приводит к снижению кровотока и последующей гипоксии тканей. Также внутрибрюшная гипертензия повышает риск развития послеоперационного перитонита у пациентов, перенесших лапаротомию.

Заключение. Для прогноза течения вторичного распространенного гнойного перитонита, уменьшения риска возникновения осложнений и летальности необходимо акцентировать внимание на измерении внутрибрюшного давления как обязательной манипуляции у хирургических больных. Несмотря на общедоступность и простоту в исполнении, данная методика позволяет произвести оценку тяжести органных дисфункций.

Ключевые слова: интраабдоминальная гипертензия, распространенный гнойный перитонит, внутрибрюшное давление, абдоминальный компартмент-синдром

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Introduction

Recent studies demonstrate high morbidity and, as a result, mortality among critically ill patients with acute abdominal pathology. Intra-abdominal infections, which include peritonitis, rank second among infectious diseases. Mortality among this group of patients ranges from 5 to 50 % [1]. It was found that in 2019, out of 1,300 autopsy reports, there were 80 (6.15 %) cases with a pathological diagnosis of the disease complicated by peritonitis. According to the classification of secondary peritonitis, it is customary to distinguish serous, fibrinous, purulent and other types, depending on the nature of the contents of the abdominal cavity. Based on statistical data, purulent peritonitis accounts for 35.28 % of cases [2]. It is important that when patients with diffuse peritonitis and abdominal sepsis are admitted, increased intra-abdominal pressure is observed in all cases, which is one of the key links in the pathogenesis of this disease.

Intra-abdominal pressure is the pressure inside the abdominal cavity, which normally does not exceed 5 mmHg. However, under some physiological conditions (cough, heavy physical exertion, etc.), it can reach 80 mmHg. Data from the World Society of the Abdominal Compartment Syndrome (WSACS) indicate that adult patients in critical condition already have increased intra-abdominal pressure [3, 4]. If this indicator is 12 mmHg or higher in two consecutive measurements, intra-abdominal hypertension may develop within 46 hours, which increases the risk of mortality by 11 times. In addition, rapid progression of intra-abdominal hypertension leads to the development of abdominal compartment syndrome, which is defined as intra-abdominal pressure exceeding 20 mmHg [4]. This syndrome is characterized by damage to two or more anatomical sections (compartments), which leads to impaired blood flow, against which tissue hypoxia develops. Unlike intra-abdominal hypertension, abdominal compartment syndrome is not classified by the level of intra-abdominal pressure (IAP), since with the development of this syndrome, a further increase in IAP is not significant [5].

The World Society of Abdominal Compartment Syndrome (WSACS) proposed 4 degrees of intra-abdominal hypertension depending on the intra-abdominal pressure indicator (Table) [6].

This classification is proposed to assess the patient's condition for the purpose of further selection of treatment tactics for surgical patients with intra-abdominal hypertension [7].

Despite modern and rapid diagnostics, as well as timely adequate surgical interventions, mortality in this syndrome remains extremely high and amounts to 38–71 % [6]. Moreover, against the background of intra-abdominal hypertension syndrome, abdominal sepsis and multiple organ failure may develop, which also increases the risk of death. The overall mortality among patients with abdominal sepsis varies from 58.06 to 84 %, which is a high figure. At the same time, almost half of the patients (48.39 %) have intra-abdominal hypertension [8].

Pathophysiology of intra-abdominal hypertension

A persistent increase in intra-abdominal pressure leads to disruption of most organ systems: cardiovascular, respiratory, nervous, and urinary. Also, increased IAP leads to disruption of the gastrointestinal tract [6]. Increased intra-abdominal pressure has no specific symptoms; it is characterized by various manifestations (Fig.).

Intra-abdominal hypertension has a significant impact on the cardiovascular system, which occurs due to compression of the abdominal blood vessels, especially the inferior vena cava and aorta. Obstruction of blood flow in the inferior vena cava leads to a decrease in venous return of blood to the heart. To increase venous return, compensatory mechanisms are activated in the form of an increase in heart rate. Thus, when the vessels are compressed, systemic vascular resistance increases, afterload on the heart increases, which causes myocardial overload and

Table. Grades of intra-abdominal hypertension
Таблица. Степени внутрибрюшной гипертензии

Grades / *Степени*	Pressure level, mmHg / *Уровень давления, мм рт. ст.*
Grade 1 / *1-я степень*	12–15
Grade 2 / *2-я степень*	16–20
Grade 3 / *3-я степень*	21–25
Grade 4 / *4-я степень*	>25

the development of heart failure. Acute heart failure contributes to a decrease in renal perfusion and arterial filling, activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system [9, 10].

In addition to the cardiovascular system, the respiratory system is also negatively affected. Due to the increase in IAP, the diaphragm is displaced upward into the chest cavity. This leads to an increase in intrathoracic pressure, and then to compression of the lungs, impaired breathing, gas exchange, a decrease in the indicators of external respiration function (respiratory volume, functional vital capacity of the lungs), a decrease in lymphatic drainage and, as a result, to an increased risk of pulmonary edema. Also, compression of the lungs and limitation of air flow into the alveoli lead to the development of alveolar atelectasis. All these processes contribute to the development of respiratory failure [2, 11].

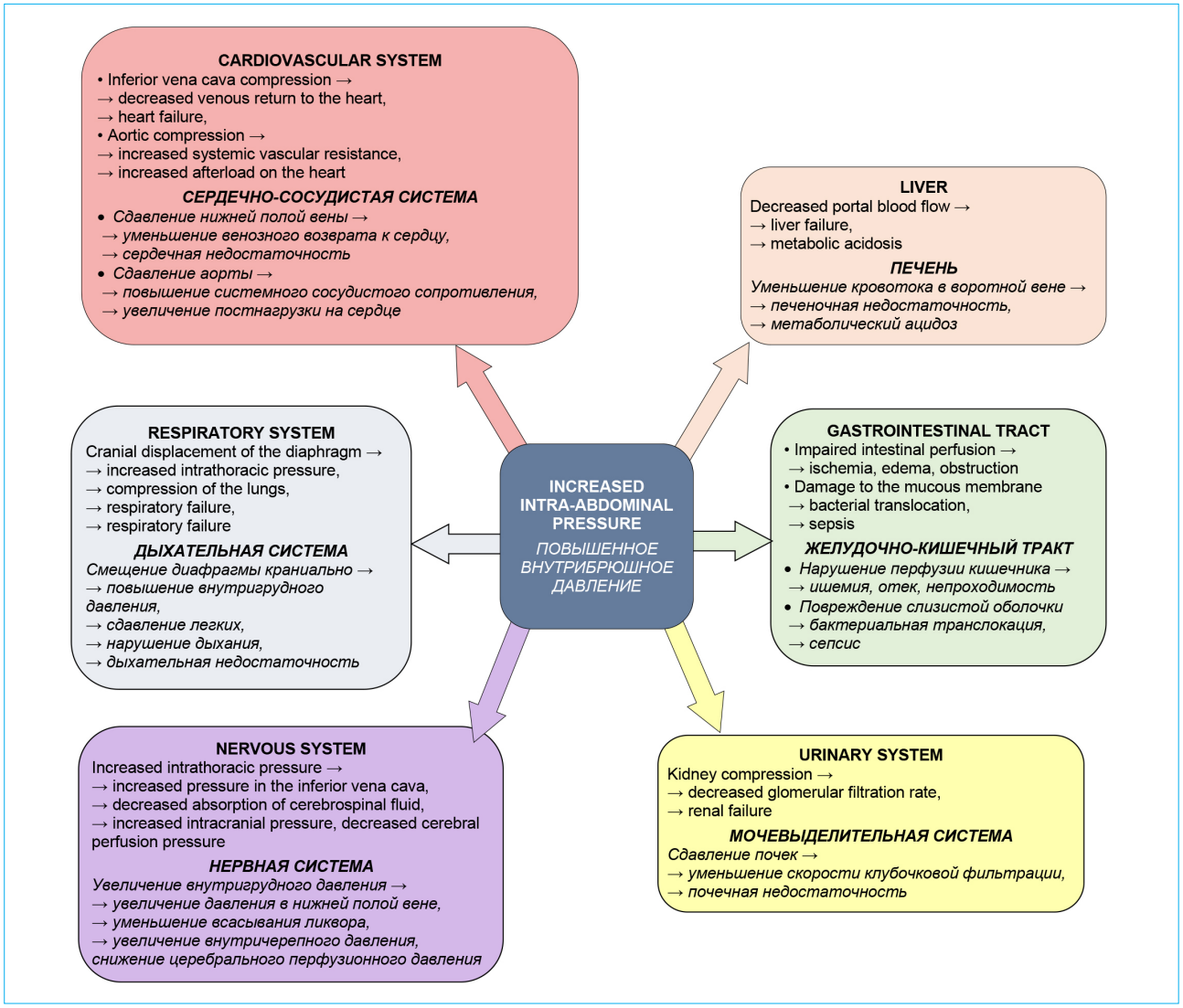

Figure. Pathophysiology of intra-abdominal hypertension

Рисунок. Патофизиология внутрибрюшной гипертензии

One of the earliest signs of intra-abdominal hypertension is oliguria and acute kidney injury. Due to compression of the renal parenchyma, the glomerular filtration rate decreases, and "renal compartment syndrome" appears, which is characterized by a decrease in renal arterial blood flow with increased renal venous pressure and vascular resistance, which leads to ischemia and, ultimately, to renal failure [9, 12, 13]. A study by J. Sun et al. showed that the incidence of acute kidney injury is higher in patients with intra-abdominal hypertension than without it [14]. S. Agrawal et al. established a link between intra-abdominal hypertension in patients with combined kidney diseases and an 11-fold increase in mortality, compared with patients without persistent increased intra-abdominal pressure [15]. Pre-existing chronic kidney disease is a negative prognostic factor in patients with peritonitis [16].

Intra-abdominal hypertension is characterized by a significant decrease in the blood supply to the abdominal organs, impaired intestinal perfusion, causing ischemia, then edema and intestinal obstruction [13]. A sustained increase in pressure of more than 25 mmHg for 60 minutes causes impaired blood flow in the mucous membrane even in successfully resuscitated patients. This leads to the translocation of bacteria through the damaged barrier of the intestinal mucosa, which worsens the patient's condition. The risk of sepsis and septic shock increases [2].

Also, intra-abdominal hypertension leads to an increase in pressure in the inferior vena cava, then in the lumbar venous plexus, which leads to a decrease in the absorption of cerebrospinal fluid, an increase in intracranial pressure and a decrease in cerebral perfusion pressure. There is a connection between intra-abdominal, intrathoracic and intracranial pressure — with an increase in IAP, intrathoracic, pleural and central venous pressure increases, which leads to a decrease in venous outflow from the brain, an increase in intracranial pressure and the development of edema [13].

A constant increase in IAP leads to a decrease in blood flow in the hepatic artery and portal vein, while the liver's ability to regulate blood flow is impaired, liver failure develops, which is manifested by a decrease in the excretion of lactate from plasma, leading to metabolic acidosis. In addition to hemodynamic changes, hepatocyte necrosis develops [2].

Due to the complex effect on various organ systems, the clinical manifestations of intra-abdominal hypertension are varied: abdominal pain and a feeling of heaviness or fullness in the abdomen; an increase in abdominal volume; tension of the anterior abdominal wall; oliguria or anuria; increased central venous pressure; hypo- or hypertension; tachypnea; orthopnea; dyspnea; hypercapnia; organ failure, etc. Without direct measurement of IAP, all this complicates diagnosis [2].

According to clinical examination, it is often difficult to diagnose intra-abdominal hypertension and determine its degree. Therefore, it is preferable to use the "gold standard" — measurement of IAP using a Foley catheter installed in the bladder. This method is performed through the bladder with the introduction of 25 mL of sterile saline. The units of measurement are millimetres of mercury (mmHg), the parameter is determined at the end of exhalation in the supine position. It is necessary to ensure the absence of abdominal muscle contractions, having previously zeroed the sensor at the level of the midaxillary line, which is the zero-reading level, according to WSACS standards [17].

Also, based on the WSACS recommendations, in critically ill patients with risk factors for intra-abdominal hypertension or abdominal compartment syndrome, IAP should be measured every 4–6 hours. Continuous monitoring of parameters is possible but is currently not standard practice [18].

Intra-abdominal hypertension and secondary diffuse purulent peritonitis

Some studies have established a relationship between an increase in IAP and the progression of the inflammatory process in the abdominal cavity, retroperitoneal space, and the development of signs of multiple organ failure. This relationship was confirmed by an analysis of the results of monitoring intra-abdominal and abdominal perfusion pressure in patients in the postoperative period by measuring with a Foley catheter. Additionally, clinical and laboratory indicators of peritonitis, intestinal paresis, and purulent-septic complications were determined [8, 19].

In the study by V.F. Zubritsky et al., a 100 % relationship was established between the presence of intra-abdominal hypertension in patients with diffuse peritonitis and abdominal sepsis. The authors determined a statistically significant direct positive correlation between the level of IAP and the prevalence of the inflammatory process in the abdominal cavity and retroperitoneal space ($p < 0.05$). In the first day after laparotomy, a reliable decrease in IAP and normalization of a number of indicators were noted: a decrease in pulmonary hypertension, diastolic load, as well as an increase in stroke volume and arterial pressure [8].

Increased IAP correlated with worsening of the integrated assessment of the severity of the condition according to the APACHE II and SOFA scales in 291 patients in the postoperative period. Such patients required urgent surgical intervention [19].

V.M. Timerbulatov et al. in a retrospective study assessed the effectiveness of treatment depending on the IAP indicators. The first stage was a retrospective analysis of the medical records of 201 patients. Based on this, the main group was formed, where the consideration of IAP indicators and the concentration of blood lactate were the main criteria for choosing a treatment method. The second stage was a retrospective analysis of the medical records

of 196 patients, who formed a comparison group. These patients were treated with traditional methods, without monitoring IAP. In the course of this study, it was found that monitoring the indicators allowed for timely surgical decompression of the abdominal cavity, and this ensured a decrease in mortality among patients [20].

In a prospective observational study, P.N. Kumar et al. found that among 40 patients with perforated peritonitis, 65 % had elevated IAP of 13.73 ± 4.30 mmHg upon admission, which corresponds to Grade 1 intra-abdominal hypertension. A directly proportional relationship was also noted between the increase in IAP and the deterioration of the SOFA (Sequential Organ Failure Assessment) score, which was developed to predict the outcome in diffuse purulent peritonitis [15, 21]. The use of the assessment scale is based on the analysis of the state of several organ systems. The level of blood oxygenation characterizes the state of the respiratory system, the number of platelets characterizes the blood coagulation system, the value of arterial pressure — the state of the cardiovascular system, the level of creatinine or diuresis — the state of the urinary system. A tendency of the sum of points towards the maximum value indicates a high probability of a fatal outcome [22].

A high frequency of intra-abdominal hypertension in peritonitis was identified by S.R. Jampani et al. In addition, its relationship with the progression of organ failure was noted, so the authors concluded that early detection of intra-abdominal hypertension and timely intervention are necessary to reduce morbidity and mortality rates [23].

R. Kidwai et al. observed an increase in the degree of intra-abdominal hypertension in most patients 6 hours after surgery for intestinal obstruction and gastrointestinal perforation, and a decrease in subsequent measurements after 12 and 24 hours. Patients with intra-abdominal hypertension had severe organ dysfunction, with the cardiovascular (40.7 %) and renal systems (39.3 %) suffering the most [24].

The development of increased IAP in the early postoperative period may increase the risk of postoperative peritonitis. As shown in the study by A. Basu et al., serial measurements of IAP in patients with secondary peritonitis revealed intra-abdominal hypertension in 41 % of cases in the postoperative period. Postoperative peritonitis developed in 20.5 % of patients and IAP was significantly increased ($p = 0.002$) during the immediate postoperative period. Thus, monitoring the dynamics of IAP changes in the postoperative period may help to make a timely decision on the need for early relaparotomy [25].

Intra-abdominal hypertension and abdominal compartment syndrome are serious complications of peritonitis, and, in this regard, B.Sh. Gogia et al. propose to include IAP measurement in the mandatory list of monitored parameters in surgical patients, primarily to reduce mortality, since in the absence of early diagnosis of abdominal compartment syndrome, which is based on IAP monitoring, mortality reaches 100 % [7].

Due to its systemic effects, intra-abdominal hypertension worsens the course of many diseases, including peritonitis. Sh.N. Kadirov, studying the dynamics of intra-abdominal pressure in acute small intestinal obstruction complicated by peritonitis, predicted the development of postoperative complications and the outcome of the disease. The study revealed that one of the signs of this obstruction was persistently increased IAP at the time of admission. Further, the author noted the relationship between intra-abdominal hypertension and the development of complications that required relaparotomy. When analyzing the dynamics of the indicators, two features were revealed — IAP decreases to normal on the second day of the postoperative period in the uncomplicated course of the disease and, conversely, increases with the development of complications [26].

In patients with secondary diffuse purulent peritonitis and abdominal compartment syndrome, a decrease in IAP promotes regression of disorders, but is accompanied by such a complication as ischemic reperfusion syndrome, in which the restoration of microcirculation in the intestinal wall promotes translocation of infection into the general bloodstream, which in turn leads to the development of generalized abdominal infection. Despite the reduction in manifestations of systemic disorders with normalization of IAP, a sudden decrease leads to the appearance of signs of multiple organ dysfunction and septic shock. Thus, monitoring the level of this indicator is necessary for timely and correctly selected treatment [27].

Conclusion

Intra-abdominal hypertension worsens the prognosis of secondary purulent peritonitis due to developing complications and increases the risk of mortality if assistance is not provided in a timely manner. As a result, the course of the disease is complicated by the development of multiple organ failure, primarily the cardiovascular, respiratory and urinary systems, gastrointestinal tract, and central nervous system. Thus, to predict the course of secondary diffuse purulent peritonitis, it is necessary to measure intra-abdominal pressure. Monitoring intra-abdominal pressure indicators allows for timely surgical decompression of the abdominal cavity, which reduces the time before the start of surgery, the time to eliminate organ dysfunction and improves the patient's recovery in the postoperative period. Monitoring the dynamics of intra-abdominal hypertension helps to decide on the need for repeated laparotomy to prevent complications. That is why in modern surgical practice in this category of patients it is important to consider measuring intra-abdominal pressure as a mandatory manipulation [28].

References / Литература

1. *Napolitano L.M.* Intra-abdominal infections. *Semin Respir Crit Care Med*. 2022;43(1):10–27. DOI: 10.1055/s-0041-1741053
2. *Попова Н.М., Кирьянов Н.А., Гончарук Б.Л., Старовойтов С.О.* Клинико-морфологическая характеристика заболеваний, осложненных перитонитом. *Modern Science*. 2020;12(2):204–6. [*Popova N.M., Kiryanov N.A., Goncharuk B.L., Starovoitov S.O.* Clinical and morphological characterist ics of diseases complicated by peritonitis. *Modern Science*. 2020;12(2);204–6. (In Russ.)].
3. *Łagosz P., Sokolski M., Biegus J., Tycinska A., Zymlinski R.* Elevated intra-abdominal pressure: A review of current knowledge. *World J Clin Cases*. 2022;6(10):3005–13. DOI: 10.12998/wjcc.v10.i10.3005
4. *Pereira B.M.* Abdominal compartment syndrome and intra-abdominal hypertension. *Curr Opin Crit Care*. 2019;25(6):688–96. DOI: 10.1097/MCC.0000000000000665
5. *Montalvo-Jave E.E., Espejel-Deloiza M., Chernitzky-Camaño J., Peña-Pérez C.A., Rivero-Sigarroa E., Ortega-León L.H.* Abdominal compartment syndrome: Current concepts and management. *Rev Gastroenterol Mex (Engl Ed)*. 2020;85(4):443–51. DOI: 10.1016/j.rgmx.2020.03.003
6. *Bodnar Z.* Polycompartment syndrome – intra-abdominal pressure measurement. *Anaesthesiol Intensive Ther*. 2019;51(4):316–22. DOI: 10.5114/ait.2019.87474
7. *Гогия Б.Ш., Аляутдинов Р.Р., Сизов В.А., Раевская М.Б., Мальцева Л.Г.* Абдоминальный компартмент синдром и интраабдоминальная гипертензия. *Высокотехнологическая медицина.* 2018;5(4):4–13. [*Gogia B.Sh., Alyautdinov R.R., Sizov V.A., Raevskaya M.B., Maltseva L.G.* Abdominal compartment syndrome and intraabdominal hypertension. *Hi-Technology Medicine*. 2018;5(4):4–13. (In Russ.)].
8. *Зубрицкий В.Ф., Забелин М.В., Айрапетян А.Т., Голубев И.В., Розберг Е.П., Коренев Д.Н.* Диагностика и лечение внутрибрюшной гипертензии и абдоминальный сепсис. *Вестник Медицинского института непрерывного образования.* 2022;2:14–7. [*Zubritsky V.F., Zabelin M.V., Hayrapetyan A.T., Golubev I.V., Rozberg E.P., Korenev D.N.* Diagnostics and treatment of intra-abdominal hypertension for patients suffering from abdominal sepsis. *Bulletin of the Medical Institute of Continuing Education*. 2022;2:14–7. (In Russ.)]. DOI: 10.46393/27821714_2022_2_14
9. *Kumar U., Wettersten N., Garimella P.S.* Cardiorenal syndrome: Pathophysiology. *Cardiol Clin*. 2019;37(3):251–65. DOI: 10.1016/j.ccl.2019.04.001
10. *Баймагамбетова А., Муканова У.А., Рысбеков М.М., Аннаоразов Ы.А.* Разработка методики лечения у больных с разлитыми гнойными перитонитами и абсцессами брюшной полости. *Вестник КазНМУ*. 2020;2:326–9. [*Baymagambetova A., Mukanova U.A., Rysbekov M.M., Annaorazov Y.A.* Development of treatment methods in patients with diffuse purulent peritonitis and abdominal abscesses. *Vestnik KazNMU*. 2020;2:326–9. (In Russ.)].
11. *Tonetti T., Cavalli I., Ranieri V.M., Mascia L.* Respiratory consequences of intra-abdominal hypertension. *Minerva Anestesiol*. 2020;86(8):877–83. DOI: 10.23736/S0375-9393.20.14325-6
12. *Husain-Syed F., Gröne H.J., Assmus B., Bauer P., Gall H., Seeger W., et al.* Congestive nephropathy: A neglected entity? Proposal for diagnostic criteria and future perspectives. *ESC Heart Fail*. 2021;8(1):183–203. DOI: 10.1002/ehf2.13118
13. *Sosa G., Gandham N., Landeras V., Calimag A.P., Lerma E.* Abdominal compartment syndrome. *Dis Mon*. 2018;65(1):5–19. DOI: 10.1016/j.disamonth.2018.04.003
14. *Sun J., Sun H., Sun Z., Yang X., Zhou S., Wei J.* Intra-abdominal hypertension and increased acute kidney injury risk: A systematic review and meta-analysis. *J Int Med Res*. 2021;49(5):3000605211016627. DOI: 10.1177/03000605211016627
15. *Agrawal S., Chaudhary T., Sahu S.K.* Correlation of intra-abdominal pressure with the outcome of perforation peritonitis. *International Surgery Journal*. 2017;4(5):1584–7. DOI: 10.18203/2349-2902.isj20171601
16. *Špička P., Chudáček J., Řezáč T., Starý L., Horáček R., Klos D.* Prognostic significance of simple scoring systems in the prediction of diffuse peritonitis morbidity and mortality. *Life (Basel)*. 2022;12(4):487. DOI: 10.3390/life12040487
17. *De Laet I.E., Malbrain M.L.N.G., De Waele J.J.* A clinician's guide to management of intra-abdominal hypertension and abdominal compartment syndrome in critically ill patients. *Crit Care*. 2020;24(1):97. DOI: 10.1186/s13054-020-2782-1
18. *Smit M., van Meurs M., Zijlstra J.G.* Intra-abdominal hypertension and abdominal compartment syndrome in critically ill patients: A narrative review of past, present, and future steps. *Scand J Surg*. 2022;111(1):14574969211030128. DOI: 10.1177/14574969211030128
19. *Мугазов М.М., Матюшко Д.Н., Исмаилов Э.А.* Клинико-лабораторные показатели при экстренной абдоминальной патологии в зависимости от степени интраабдоминальной гипертензии. *Медицинский журнал Астана*. 2019;2(100):285–90. [*Mugazov M.M., Matyushko D.N., Ismailov E.A.* Clinical and laboratory indicators in emergency abdominal pathology depending on the degree of intraabdominal hypertension. *Astana Medical Journal*. 2019;2(100):285–90. (In Russ.)].
20. *Тимербулатов В.М., Тимербулатов Ш.В., Фаязов Р.Р., Тимербулатов М.В., Гайнуллина Э.Н., Смыр Р.А.* Диагностика и выбор метода лечения интраабдоминальной гипертензии и абдоминального компартмент-синдрома. *Вестник Российской академии медицинских наук*. 2019;74(3):210–5. [*Timerbulatov V.M., Timerbulatov Sh.V., Fayazov R.R., Timerbulatov M.V., Gaynullina E.N., Smir R.A.* Diagnosis and choice of a method of treatment of intraabdominal hypertension and abdominal compartment syndrome. *Annals of the Russian Academy of Medical Sciences*. 2019;74(3):210–5. (In Russ.)]. DOI: 10.15690/vramn1080
21. *Kumar P.N., Bains L., Lal P., Mishra A., Yasir Beg M., Maranna H.* Role of intra-abdominal pressure in the outcomes of perforation peritonitis: A prospective observational study. *Turk J Surg*. 2021;37(3):253–9. DOI: 10.47717/turkjsurg.2021.4945
22. *Лещишин Я.М., Баранов А.И., Потехин К.В., Ярощук С.А., Валуйских Ю.В.* Применение интегральных оценочных шкал у пациентов с распространенным гнойным перитонитом. *Медицина в Кузбассе*. 2020;19(2):20–7. [*Leshchishin Ya.M., Baranov A.I., Potekhin K.V., Yaroshchuk S.A., Valuiskikh Y.V.* Application of integral evaluation scales in patients with widespread purulent peritonitis. *Medicine in Kuzbass*. 2020;19(2):20–7. (In Russ.)]. DOI: 10.24411/2687-0053-2020-10013
23. *Jampani S.R., Vattikonda S., Jampani S., Vasireddi V.* Intra-abdominal pressure monitoring and outcome in patients with peritonitis. *Journal of Evidence Based Medicine and Healthcare*. 2016;3(42):2064–7. DOI: 10.18410/jebmh/2016/460
24. *Kidwai R., Baral R.K., Sharma A.* Analysis of intra-abdominal pressure in obstructive and perforative lesions of gastro-intestinal tract. *Journal of Nepalgunj Medical College*. 2018;16(2):31–4. DOI: 10.3126/jngmc.v16i2.24871
25. *Basu A., Pai D.R.* Early elevation of intra-abdominal pressure after laparotomy for secondary peritonitis: A predictor of relaparotomy? *World J Surg*. 2008;32(8):1851–6. DOI: 10.1007/s00268-008-9605-x
26. *Кадиров Ш.Н.* Внутрибрюшное давление при острой тонкокишечной непроходимости осложненным перитонитом. *Инновационные подходы в современной науке: сборник статей по материалам XLI Международной научно-практической конференции «Инновационные подходы в современной науке».* 2019;5(41):7–10. [*Kad-*

irov Sh.N. Intra-abdominal pressure in acute small bowel obstruction complicated by peritonitis. *Innovatsionnye podkhody v sovremennoy nauke: sbornik statey po materialam XLI Mezhdunarodnoy nauchno-prakticheskoy konferentsii "Innovatsionnye podkhody v sovremennoy nauke"*. 2019;5(41):7–10. (In Russ.)].
27. *Коровин А.Я., Базлов С.Б., Андреева М.Б., Нарсия В.В., Трифанов Н.А.* Проявления абдоминального сепсиса у больных с распространенным перитонитом. *Кубанский научный медицинский вестник.* 2017;6:78–83. [*Korovin A.Ya., Bazlov S.B., Andreeva M.B., Narsiya V.V., Trifanov N.A.* Manifiststions of abdominal sepsis in patients with diffuse peritonitis. *Kuban Scientific Medical Bulletin.* 2017;6:78–83. (In Russ.)]. DOI: 10.25207/1608-6228-2017-24-6-78-83
28. *Остаев А.О., Бархударов А.А., Климов А.Е.* Эпидемиологическая характеристика и факторы риска развития перитонита в современном мире. *Современная наука: актуальные проблемы теории и практики. Серия: Естественные и Технические Науки.* 2023;11:168–72. [*Ostaev A.O., Barkhudarov A.A., Klimov A.E.* Epidemiological characteristics and risk factors of the development of peritonitis in the modern world. *Modern science: Actual Problems of Theory and Practice. Series: Natural and Technical Sciences.* 2023;11:168–72. (In Russ.)]. DOI: 10.37882/2223-2966.2023.11.22

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