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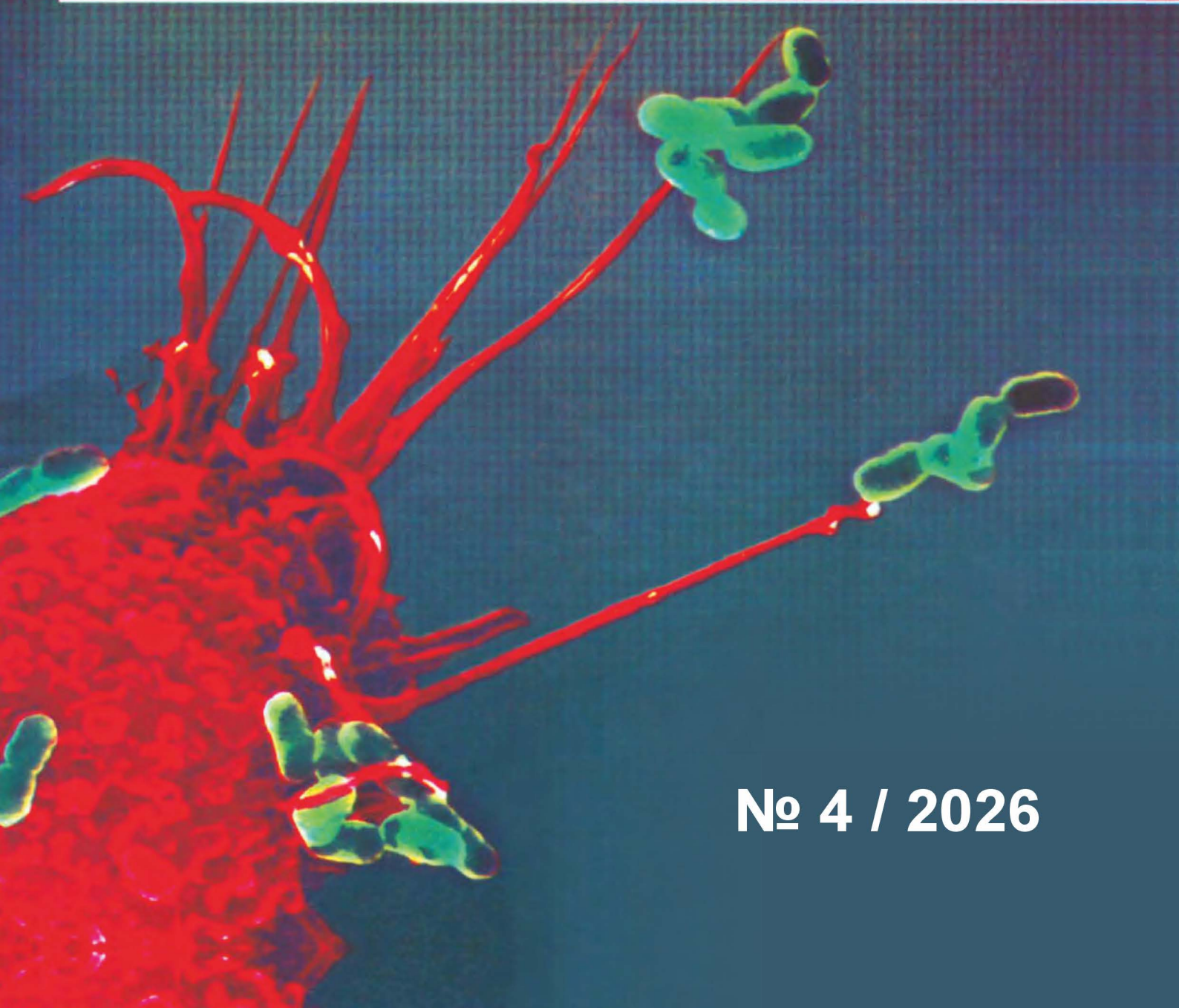
ИНФЕКЦИЯ, ИММУНИТЕТ и ФАРМАКОЛОГИЯ



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IMMUNE-ASSOCIATED BIOCHEMICAL DISTURBANCES IN ACUTE ADHESIVE INTESTINAL OBSTRUCTION

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Acute adhesive intestinal obstruction (AAO) remains an important and unresolved issue in modern abdominal surgery due to relatively high incidence of morbidity and mortality associated with delayed diagnostics and suboptimal therapy of this condition [3, 7, 12]. The etiology of AAO consists not only in the mechanical occlusion of intestine lumen but also in the cascade of systemic and local disruptions of physiological homeostasis including inflammation, coagulation imbalance, as well as ischemia–reperfusion injury that lead to disease progression and complications. Nevertheless, despite intensive clinical studies, the specific pathogenetic bases of AAO, especially regarding the immunobiochemical regulation of this process, remain poorly understood [1, 5, 11].

Modern developments in biomedicine provide an insight into the important role of the immune system and regulatory growth factors in the pathogenesis of adhesive intestinal obstruction. Namely, changes in the concentration of proinflammatory cytokines, chemokines, angiogenic factors, and hemostatic system elements are found which indicate complicated molecular mechanisms that regulate the reaction of the organism to adhesions and ischemia [2, 6, 8]. Still, existing data are fragmentary, and integrative pathogenetic models which take into account the severity of the disease are yet to be developed [4, 9, 10].

Hemostasis plays a central role in the pathophysiology of acute intestinal obstruction in an adhesive variant, when mechanical disruption is combined with ischemic damage to the intestinal wall. This pathogenesis is associated with a synchronous activation of the processes of both coagulation and anticoagulation, which can result in hypercoagulation and, in severe cases, in the development of DIC. Coagulation tests and the measurement of fibrinolytic activity allow obtaining valuable information about the severity of the pathological process and the risks of the occurrence of such complications as intestinal ischemia and necrosis [3, 7].

Even though the understanding of these mechanisms has increased significantly, insufficient data are available concerning the difference in hemostasis between uncomplicated and complicated variants of AAO [5, 11]. Evaluation of these parameters can help detect compli-

cations earlier and optimize surgical and intensive care tactics.

Study objectives: To study the mechanisms of interaction of the hemostasis and fibrinolysis systems in patients with acute adhesive intestinal obstruction.

Materials and methods of study: In total, 46 patients suffering from AAIO were recruited for the study and represented the study sample. According to their clinical presentation, patients were divided into two groups: patients with an uncomplicated course of the disease (n=35) and complicated forms of the disease (n=11) that required hospital surgical treatment within 2019-2024. The control group consisted of 20 age-matched healthy individuals. Patients with chronic diseases of the liver and kidneys, malignant tumors, and mental disorders were excluded from the study. Plasma levels of tissue plasminogen activator (tPA) and plasminogen activator inhibitor-1 (PAI-1) were measured by means of ELISA technique with commercial kits (ELISA Kits, Russian Federation).

Research results and their discussion: The evaluation of the parameters of the hemostasis showed significant difference from control values that suggests the activation of the coagulation system during the process of inflammation and hypoxia in the abdominal cavity.

In case of an uncomplicated AAO, the prothrombin time (PT) was 9.23±0.25 seconds that was 1.14-fold greater than in control group (8.07±0.18 seconds). At the same time, in case of complicated AAO, the PT amounted to 9.31±0.29 seconds and this value was 1.15-fold greater than the control one.

This increase in PT testifies to the disturbance of the mechanism of extrinsic blood coagulation, which is due to the depletion of factors II, V, VII, and X during the process of inflammatory reaction and vascular damage.

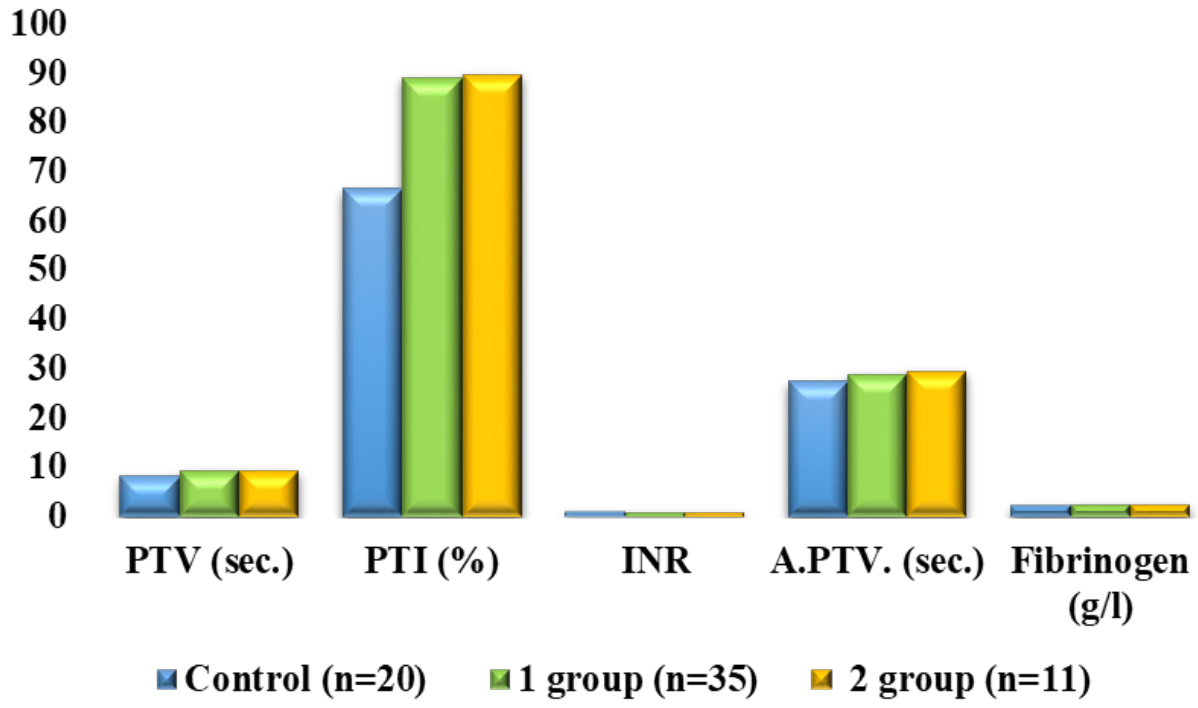

Fig. 1. **Indicators of the coagulation system in the examined subjects**

Prothrombin index (PTI) in the control group was 66.4±2.1%. However, in case of uncomplicated patients, PTI was equal to 88.9±2.4% (1.34-fold increase), whereas in patients with complications, it amounted to 89.7±2.7% (1.35-fold increase compared to control). High PTI is caused by compensation and activation of the coagulation process in order to keep hemostasis in the state of balance under ischemic and hypoxic influence of the intestines.

International normalized ratio (INR) for healthy people was 0.81±0.03. For those without complications, INR was increased up to 0.94±0.05 (1.16-fold increase), whereas in complicated group it was equal to 0.88±0.04 (1.09-fold increase compared to control). Increase in INR indicates relative deficiency of coagulation factors under the constant activity of coagulation cascade and can be considered as an indicator of hypercoagulable imbalance

Activated partial thromboplastin time (APTT) in the control group was 27.3±0.6 seconds. In patients with uncomplicated AAO, it increased to 28.7±0.7 seconds (1.05-fold), and in complicated cases to 29.5±0.9 seconds (1.08-fold above control). The prolonged duration of the APTT is a marker of activation of the intrinsic coagulation cascade due to inflammatory mediators, the secretion of cytokines, and the activation of factor XII in connection with the irritation and damage to the peritoneum.

In the control group, the concentration of fibrinogen amounted to 2.06±0.12 g/L, in the group of patients without complications – 2.29±0.14 g/L (1.11-fold), and in the group of patients with complications – 2.41±0.17 g/L (1.17-fold), (fig.1).

Increased levels of fibrinogen can be considered as evidence of the occurrence of the inflammatory reaction, as the acute-phase protein production occurs under the influence of interleukin-6.

Therefore, all the above-described parameters allow us to say about the development of a subcompensated hypercoagulable state, which is more marked among patients with complications. The changes described above are characteristic of activation of both intrinsic and extrinsic coagulation cascades.

The increase in fibrinogen and PTI levels, along with PT and APTT prolongations, indicate the activation of the hemostatic process in connection with the tissue hypoxia, endothelial dysfunction, increased levels of proinflammatory cytokines, and tissue thromboplastin secretion from the ischemic or necrotic sections of the intestine.

Tissue Plasminogen Activator (tPA) is a serine protease that is produced by vascular endothelial cells and acts as an important agent for initiating fibrinolysis via conversion of plasminogen into plasmin, hence ensuring the degradation of fibrin and dissolution of clots. tPA is involved in the maintenance of homeostasis between coagulation and fibrinolysis under normal conditions so that no excessive clotting occurs. With regard to acute adhesive intestinal obstruction, tPA becomes a marker of endothelium integrity and its ability to perform fibrinolysis, as any kind of ischemia, inflammation, and disturbance of microcirculation affects the activity of endothelium.

In patients with an uncomplicated course, tPA levels decreased to 6.87±0.29 ng/mL, which is 1.43-fold lower than in the control group (9.83±0.34 ng/mL). However, in case of complicated AAO, this reduction was more obvious: the values were down to 4.72±0.21 ng/mL or about

2.08 times lower than the norm (fig.2). Thus, the fibrinolytic system activity was progressively inhibited with increase in the disease severity.

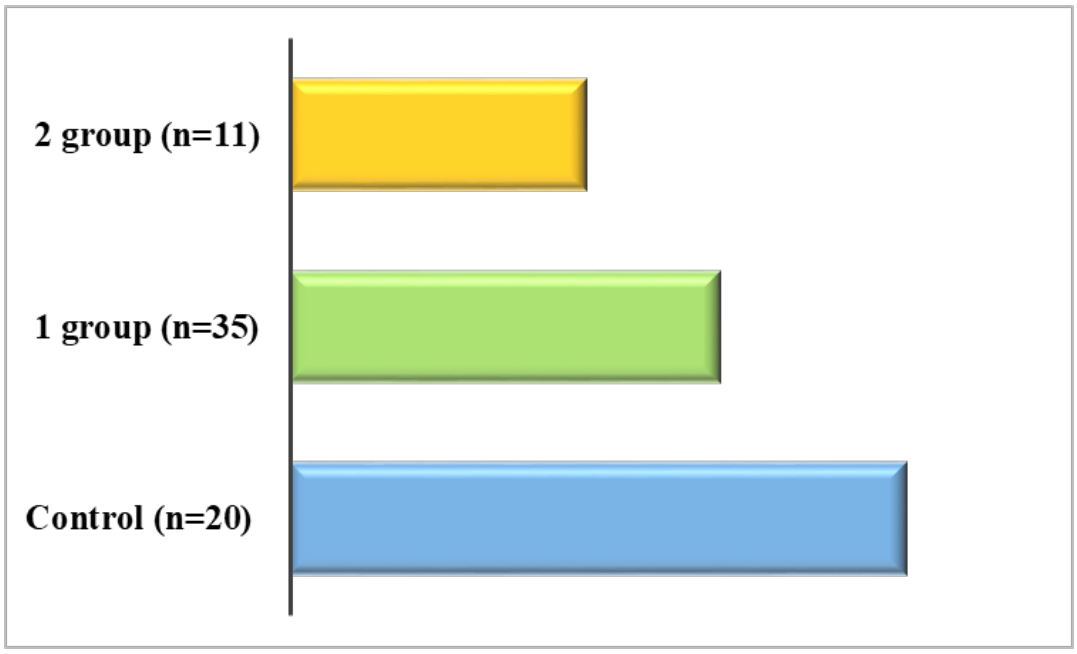

Fig. 2. **Levels of tissue plasminogen activator in subjects (ng/ml)**

The decrease in tissue plasminogen activator (tPA) content reflects decreased fibrinolytic capability of the blood. It is a consequence of multiple factors such as ischemic damage of the vascular wall, causing a decrease in tPA production; activation of the coagulation cascade leading to the use of fibrinolytic factors; imbalance between coagulation and fibrinolysis processes. All of them create favorable conditions for microthrombosis formation within the walls of the intestines and make the disease progression toward complicated condition possible.

Accordingly, in patients with acute adhesive intestinal obstruction-particularly in the presence of complications-a pronounced depression of fibrinolytic activity is observed. Such conditions facilitate thrombogenesis, decrease perfusion of blood in ischemic areas, and intensify hypoxia of tissues, thus ensuring even more deterioration of intestinal viability.

Plasminogen activator inhibitor-1 (PAI-1) is considered a key modulator of the fibrinolytic system and acts as a major inhibitor of tissue plasminogen activator (tPA) and urokinase-type plasminogen activator (uPA). High activity of PAI-1 results in inhibition of fibrinolysis, accumulation of fibrin and development of adhesions, which play an important role in the development of adhesive obstruction of intestine.

Since adhesive obstruction of intestine is characterized by inflammation and disorders of microcirculation, measurement of the PAI-1 level is important for understanding activation of prothrombotic and pro-adhesive processes. That is why PAI-1 can be regarded not only as a marker of fibrinolytic inhibition but also as a prognostic one.

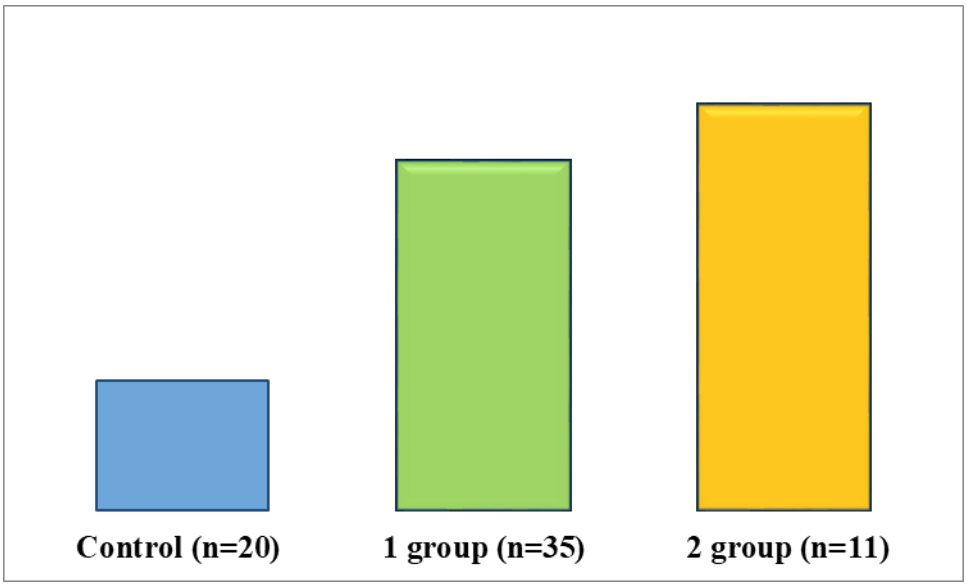

Fig. 3. **Plasminogen activator inhibitor-1 levels in subjects (ng/ml)**

The concentration of PAI-1 in the control group was 32.7±2.1 ng/mL (physiological norm). In patients with uncomplicated acute adhesive intestinal obstruction, PAI-1 increased significantly to 88.1±5.6 ng/mL (a 2.7-fold increase compared to the control group ($p<0.01$)). In the case of complicated course, PAI-1 increased up to 102.3±7.8 ng/mL (an increase of 3.1-fold compared to the control group ($p<0.01$) and 1.2-fold compared to the group with uncomplicated course (fig.3).

The observed increasing tendency corresponds to stepwise fibrinolytic activity inhibition depending on the disease severity. The increase in PAI-1 level in patients with acute adhesive intestinal obstruction is mainly due to systemic inflammation activation, endothelial dysfunction, and macrophage activation with increased production of pro-inflammatory cytokines, including IL-6 and TNF-α that promote PAI-1 synthesis. During intestinal ischemia and venous stasis, coagulation activation with concomitant inhibition of fibrinolysis leads to excessive fibrin deposition in the abdominal cavity.

In those patients who have complicated variants of this condition, such as perforation, peritonitis, or necrosis of the intestine, there is still higher PAI-1 secretion, depending on the intensity of inflammation and extent of microcirculation disturbances.

Thus, PAI-1 may be considered not only as an indicator of the severity of the pathology but also as a target for treatment in order to decrease the likelihood of developing postoperative adhesions and recurrence of obstruction of the intestine.

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REZUME
O‘TKIR CHANDIQLI ICHAK TUTILISHIDA IMMUN BILAN BOG‘LIQ BIOKIMYOVIY BUZILISHLAR

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Kalit so‘zlar: *o‘tkir chandiqli ichak tutilishi, fibrinoliz, tPA, PAI-1, gemostaz, koagulyatsiya, asoratlar, ichak ishemiyasi, biomarkerlar, immunobiokimyo.*

O‘tkir chandiqli ichak tutilishi (O‘ChIT) abdominal jarrohlik amaliyotida murakkab va prognostik jihatdan noqulay patologiyalardan biri bo‘lib qolmoqda, bu esa operatsiyadan keyingi asoratlar va o‘lim ko‘rsatkichlarining yuqoriligi bilan bog‘liq. Mazkur tadqiqotning maqsadi O‘ChIT bilan kasallangan bemorlarda immunobiokimyoviy o‘zgarishlarni, ayniqsa gemostaz tizimi va fibrinoliz faolligi holatini kompleks baholashdan iborat bo‘ldi. Tadqiqotga 46 nafar O‘ChIT bilan kasallangan bemor jalb qilinib, ular kasallikning asoratsiz va asoratlangan kechishiga ko‘ra ikki guruhga ajratildi. Nazorat guruhini esa 20 nafar amalda sog‘lom shaxs tashkil etdi. Protrombin vaqti (PT), xalqaro normallashtirilgan nisbat (INR), faollashtirilgan qisman tromboplastin vaqti (APTT), fibrinogen miqdori, shuningdek fibrinolizning asosiy markerlari bo‘lgan to‘qima plazminogen aktivatori (tPA) va plazminogen aktivatori ingibitori-1 (PAI-1) ko‘rsatkichlarini tahlil qilish natijasida bemorlarda subkompensatsiyalangan giperkoagulyatsiya holati aniqlanib, bu ayniqsa asoratlangan OChIT holatlarida yaqqol namoyon bo‘ldi. Asoratlangan guruhda fibrinogen va PAI-1 darajalarining sezilarli oshishi hamda tPA miqdorining pasayishi kuzatildi, bu esa endotelial disfunksiya, tizimli yallig‘lanish jarayonlarining faollashuvi va fibrinolitik faollikning susayganligini ko‘rsatadi. Ushbu patofiziologik o‘zgarishlar trombotik asoratlar hamda ichak devorining ishemik shikastlanishi xavfining ortishi bilan bog‘liq. Olingan natijalar O‘ChIT da koagulyatsiya va fibrinoliz markerlarini monitoring qilish yuqori prognostik ahamiyatga ega ekanligini hamda ularni kasallikning asoratlangan kechishini erta aniqlash va individual davolash strategiyalarini ishlab chiqishda qo‘llash maqsadga muvofiqligini tasdiqlaydi.

РЕЗЮМЕ
ИММУНОАССОЦИИРОВАННЫЕ БИОХИМИЧЕСКИЕ НАРУШЕНИЯ ПРИ ОСТРОЙ СПАЕЧНОЙ КИШЕЧНОЙ НЕПРОХОДИМОСТИ

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Ключевые слова: *острая спаечная кишечная непроходимость, фибринолиз, tPA, PAI-1, гемостаз, коагуляция, осложнения, ишемия кишечника, биомаркеры, иммунобиохимия.*

Острая спаечная кишечная непроходимость (ОСН) до сих пор считается одним из наиболее сложных и наименее благоприятных с точки зрения прогноза заболеваний в абдоминальной хирургии из-за высокой частоты послеоперационных осложнений и высокой смертности. Целью нашего исследования было детальное изучение изменений иммунобиохимических характеристик у пациентов с ОСН, особенно тех, которые связаны с функциональным состоянием системы гемостаза и фибринолиза. В исследовании приняли участие 46 пациент с ОСН, которые были разделены на две клинические группы в зависимости от наличия осложненной или неосложненной формы заболевания. Комплексное изучение показателей коагуляционного звена гемостаза, включая протромбиновое время (ПВ), международное нормализованное отношение (МНО), активированное частичное тромбопластиновое время (АЧТВ), уровень фибриногена, а также ключевых маркеров фибринолиза – тканевого активатора плазминогена (tPA) и ингибитора активатора плазминогена-1 (PAI-1), выявило состояние субкомпенсированной гиперкоагуляции, наиболее выраженное у пациентов с осложнённым течением ОСКН. У больных данной группы отмечалось достоверное повышение концентрации фибриногена и уровня PAI-1 в сочетании со значительным снижением содержания tPA, что свидетельствовало о развитии эндотелиальной дисфункции, активации системного воспалительного ответа и угнетении фибринолитической активности. Выявленные патофизиологические изменения связаны с повышенным риском тромботических осложнений и ишемического повреждения кишечной стенки. Указанные изменения в патогенезе сопровождаются повышением вероятности тромботических осложнений и ишемии кишечной стенки. Полученные данные доказывают большую прогностическую значимость оценки показателей системы свертывания и фибринолиза при острой кишечной непроходимости, а также демонстрируют возможность использования показателей для ранней диагностики осложненного течения патологии и создания персонализированного подхода к терапии и диагностике. лечебно-диагностических подходов.