НЕОНАТОЛОГІЯ, ХІРУРГІЯ ТА ПЕРИНАТАЛЬНА МЕДИЦИНА
NEONATOLOGY, SURGERY AND PERINATAL MEDICINE
т. XVI, №1(59), 2026
VOL. XVI, №1(59), 2026
ISSN 2226-1230 (PRINT) ISSN 2413-4260 (ONLINE)
KEY TITLE: NEONATOLOGÌÂ, HÌRURGÌÂ TA PERINATAL'NA MEDICINA (ONLINE)
ABBREVIATED KEY TITLE: NEONATOL. HÌR. PERINAT. MED. (ONLINE)

UDC: 616.381-008.64-07:616.155.194-07:612.017
DOI: 10.24061/2413-4260.XVI.1.59.2026.17

IMMUNOBIOCHEMICAL CHARACTERISTICS OF ACUTE ADHESIVE INTESTINAL OBSTRUCTION

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Summary.

Acute adhesive intestinal obstruction (AAIO) represents one of the most challenging conditions in abdominal surgery and is characterised by high complication and mortality rates.

Objective. *To examine the interrelationships among proinflammatory mediators, haemostasis system parameters, and fibrinolysis markers in acute adhesive intestinal obstruction.*

Materials and Methods. *Correlations between proinflammatory cytokines (IFN-γ, TNF-α, IL-6), haemostatic parameters (prothrombin time [PT], international normalised ratio [INR], activated partial thromboplastin time [APTT], fibrinogen), and fibrinolysis markers (tissue plasminogen activator [tPA], plasminogen activator inhibitor-1 [PAI-1]) were analysed in 91 patients with AAIO, subdivided into uncomplicated and complicated forms, and compared with 20 healthy controls. Immunobiochemical markers were determined by enzyme-linked immunosorbent assay (ELISA). Written informed consent was obtained from all participants prior to enrolment. All procedures were approved by the Ethics Committee of the Institute and were conducted in accordance with the principles of the Declaration of Helsinki, as revised at the 75th General Assembly of the World Medical Association in October 2024. Statistical analysis was performed using SPSS version 26.0. Quantitative data are presented as the arithmetic mean (M) ± standard deviation (SD) or standard error of the mean (m), as appropriate. The normality of distribution was assessed prior to analysis. Intergroup comparisons were conducted using Student's t-test for normally distributed variables and the Mann–Whitney U-test for non-parametric data. A p-value of less than 0.05 was considered statistically significant. study was conducted as part of the research plan of the Bukhara State Medical Institute (05.2022 DSc.135), entitled «Development of New Approaches to Early Diagnosis, Treatment, and Prevention of Pathological Conditions Affecting the Health of the Population of the Bukhara Region after COVID-19 (2022-2026)».*

Results. *Two distinct immune-coagulative phenotypes were identified. IFN-γ correlated positively with INR (r = 0.41) and tPA (r = 0.50), and negatively with fibrinogen (r = –0.36) and PAI-1 (r = –0.50), reflecting enhanced fibrinolysis and reduced coagulation potential. Unlike IFN-γ, TNF-α and IL-6 were associated with elevated fibrinogen and PAI-1 levels, prolonged APTT, and decreased INR and tPA, indicative of hypercoagulability, suppressed fibrinolysis, and endothelial dysfunction.*

Conclusion. *These alterations were most pronounced in the complicated subgroup, suggesting an elevated risk of thrombotic events and ischemic intestinal damage. The findings demonstrate the prognostic value of monitoring coagulation and fibrinolysis markers in AAIO and support their use for early detection of complications and personalisation of antithrombotic strategies.*

Keywords: *Adhesive Intestinal Obstruction; Fibrinolysis; Tissue Plasminogen Activator; Plasminogen Activator Inhibitor 1; Haemostasis; Blood Coagulation; Postoperative Complications; Intestinal Ischaemia; Biomarkers; Immunobiochemistry.*

Introduction

Acute adhesive intestinal obstruction (AAIO) remains one of the pressing problems in abdominal surgery and is characterized by high complication and mortality rates, particularly in cases of delayed diagnosis and treatment [1-5]. The development of the pathological process in AAIO is associated not only with mechanical compression of the intestine, but also with severe disturbances in systemic and local homeostasis, including activation of inflammation, coagulation shifts, ischaemia-reperfusion syndrome. Despite the substantial body of clinical research, the pathogenetic mechanisms of AAIO, particularly at the level of immunobiochemical markers, have not been fully elucidated [6, 7].

Current evidence indicate an important role of the immune system and growth factors in the pathogenesis of AAIO. Alterations in proinflammatory cytokine levels, chemokines, angiogenesis factors, and components of the haemostasis system have been identified, reflecting complex molecular interactions that determine the body's response to adhesive and ischaemic aggression [8, 9]. The available data remain fragmented, and generalised models accounting for the severity of the process are still under development [10, 11].

The haemostasis system plays a central role in the pathogenesis of acute intestinal obstruction, particularly in its adhesive form, when ischemic tissue damage is superimposed on mechanical disturbances of intestinal patency. These processes are accompanied by activation of the coagulation and anticoagulation pathways, which may result in hypercoagulability syndrome and an increased risk of disseminated intravascular coagulation (DIC). Coagulogram parameters and fibrinolytic activity serve as indicators of the severity of the pathological process and the development of complications, including ischaemia and necrosis of the intestinal wall [12, 13].

Despite established knowledge of pathophysiological mechanisms involved, data on the differentiated state of the haemostasis system in uncomplicated and complicated forms of AAIO remain limited [14]. A more detailed

examination of these parameters in relation to the clinical course of the disease may contribute to more accurate early detection of complications and to the selection of optimal surgical and intensive care management strategies.

Objective of the study. To examine the interaction among the proinflammatory mediators, haemostasis system and fibrinolysis markers in acute adhesive intestinal obstruction

Materials and methods of the study

Patients with AAIO were enrolled as the study group (n = 91) and subsequently divided into two subgroups: Group 1 comprised patients with uncomplicated AAIO (n = 35), and Group 2 comprised patients with complicated AAIO (n = 11), all of whom received inpatient surgical treatment at the clinic of Tashkent State Medical University between 2019 and 2024. The control group consisted of healthy age-matched individuals (n = 20). The exclusion criteria were hepatic disease, renal disease, malignant neoplasms, and mental disorders. Immunobiochemical markers (tPA, PAI-1, IFN-y, TNF-α, IL-6) were determined by enzyme-linked immunosorbent assay (ELISA; Elisa Kids, RF).

Statistical analysis was performed using SPSS version 26.0. Quantitative data are presented as the arithmetic mean (M) ± standard deviation (SD) or standard error of the mean (m), as appropriate. The normality of distribution was assessed prior to analysis. Intergroup comparisons were conducted using Student's t-test for normally distributed variables and the Mann–Whitney U-test for non-parametric data. A p-value of less than 0.05 was considered statistically significant.

Written informed consent was obtained from all participants prior to enrolment. All procedures were approved by the Ethics Committee of the Institute and were conducted in accordance with the principles of the Declaration of Helsinki, as revised at the 75th General Assembly of the World Medical Association in October 2024.

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Research results and discussion.

Analysis of haemostasis system parameters in patients with acute adhesive intestinal obstruction (AAIO) revealed statistically significant deviations from control values, reflecting the activation of the coagulation cascade in the setting of inflammatory-hypoxic changes in the abdominal cavity.

In patients of Group 1, prothrombin (PT) was 9.23 ± 0.25 s, exceeding the control value (8.07 ± 0.18 s) by a factor of 1.14. In Group 2 (complicated course), PT reached 9.31 ± 0.29 s, representing a 1.15-fold increase relative to the control. Prolongation of the PT indicates inhibition of the extrinsic coagulation pathway, attributable to the consumption of factors II, V, VII and X in the setting of systemic inflammation and endothelial dysfunction.

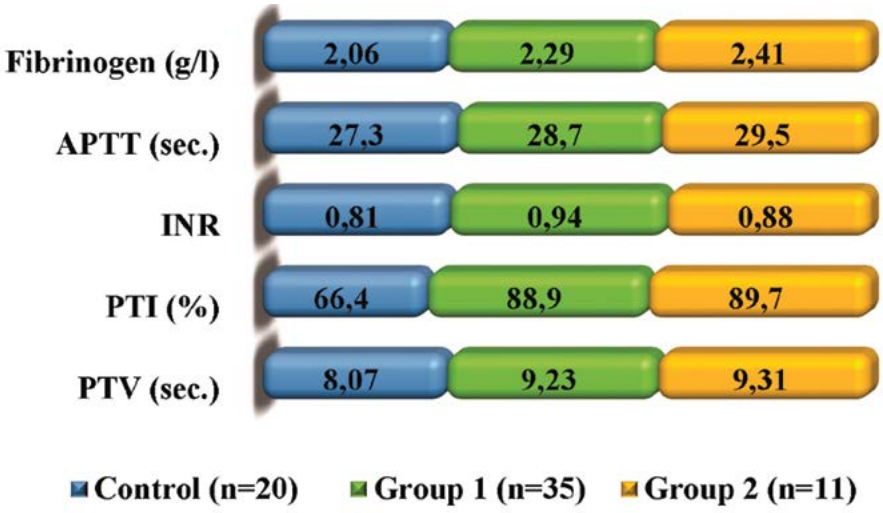


Fig. 1. Indicators of the coagulation system in the examined subjects.

The prothrombin index (PTI) in the control group was 66.4 ± 2.1%, whereas in Group 1 it was 88.9 ± 2.4% (a 1.34-fold increase) and in Group 2 it was 89.7 ± 2.7% (a 1.35-fold increase). The elevation of PTI reflects compensatory activity of the coagulation system directed at maintaining haemostasis under conditions of intestinal ischaemia and tissue distress.

The mean INR value in the control group was 0.81 ± 0.03, with an increase to 0.94 ± 0.05 in Group 1 (a 1.16-fold increase) and to 0.88 ± 0.04 in Group 2 (a 1.09-fold increase relative to the control). Elevation of INR is indicative of a relative deficiency of coagulation factors during activation of the coagulation cascade and serves as an indirect marker of a *hypercoagulability* shift.

APTT in the control group was 27.3 ± 0.6 s, compared with 28.7 ± 0.7 s in subgroup 1 (a 1.05-fold increase) and 29.5 ± 0.9 s in subgroup 2 (a 1.08-fold increase). Prolongation of APTT indicates activation of the intrinsic coagulation pathway, attributable to the release of tissue factors and cytokines and to activation of factor XII in response to inflammation and peritoneal irritation.

The fibrinogen level in the control group was 2.06 ± 0.12 g/l, rising to 2.29 ± 0.14 g/l in subgroup 1 (a 1.11-fold increase) and to 2.41 ± 0.17 g/l in subgroup 2

(a 1.17-fold increase) (Fig. 1). Elevation of fibrinogen, an acute-phase protein, confirms the presence of a systemic inflammatory response, increased hepatic synthesis under the influence of IL-6, and activation of the hepatic acute-phase response.

Tissue plasminogen activator (tPA) is a serine protease produced by endothelial cells that plays a central role in the initiation of fibrinolysis. It converts plasminogen to plasmin, thereby promoting fibrin degradation and thrombus dissolution [15]. Under physiological conditions, tPA maintains the balance between coagulation and fibrinolysis, preventing excessive thrombosis. In AAIO, tPA is regarded as a sensitive marker of endothelial function and fibrinolytic system activity, as intestinal ischaemia, systemic inflammation, and microvascular thrombosis directly affect the vascular endothelium and disrupt the hemostatic balance.

In patients without complications (Group 1), the tPA level decreased to 6.87 ± 0.29 ng/ml, representing a 1.43-fold reduction relative to the control group (9.83 ± 0.34 ng/ml). In patients with complicated course of AAIO (Group 2), tPA decreased markedly, to 4.72 ± 0.21 ng/ml, corresponding to a 2.08-fold reduction relative to the reference range (Fig. 2).

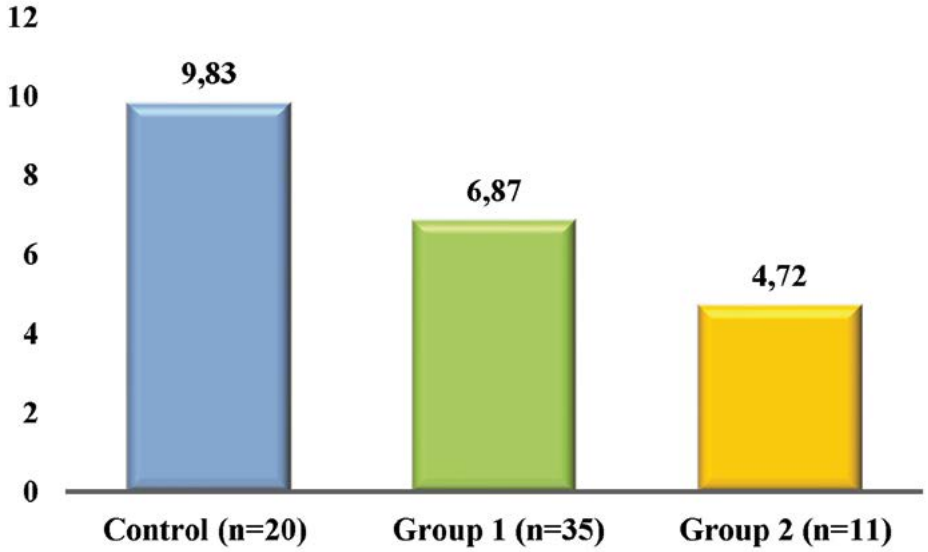


Fig. 2. Levels of tissue plasminogen activator in subjects (ng/ml)

A decrease in tPA concentration reflects functional inhibition of the fibrinolytic system. This process may be attributed to the following mechanisms: damage to the vascular endothelium resulting from ischaemia, leading to reduced tPA production; activation of the hypercoagulation cascade, accompanied by consumption of fibrinolytic system components; and development of an imbalance between coagulation and fibrinolysis, which promotes microthrombosis in the intestinal wall and progression to a complicated disease course.

Plasminogen activator inhibitor-1 (PAI-1) is a key component of the fibrinolysis system and the principal inhibitor of both tissue plasminogen activator (tPA) and urokinase plasminogen activator (uPA) [16]. Elevated PAI-1 levels suppress fibrinolysis, promote fibrin deposition, and facilitate the formation of fibrous adhesions, which is of particular significance in the pathogenesis of adhesive intestinal obstruction. Given that AAIO is accompanied by a pronounced inflammatory response and impaired microcirculation, PAI-1 levels may be used to assess the degree of activation of pro-adhesive and pro-thrombotic mechanisms and serve as a prognostic marker for the risk of complicated disease.

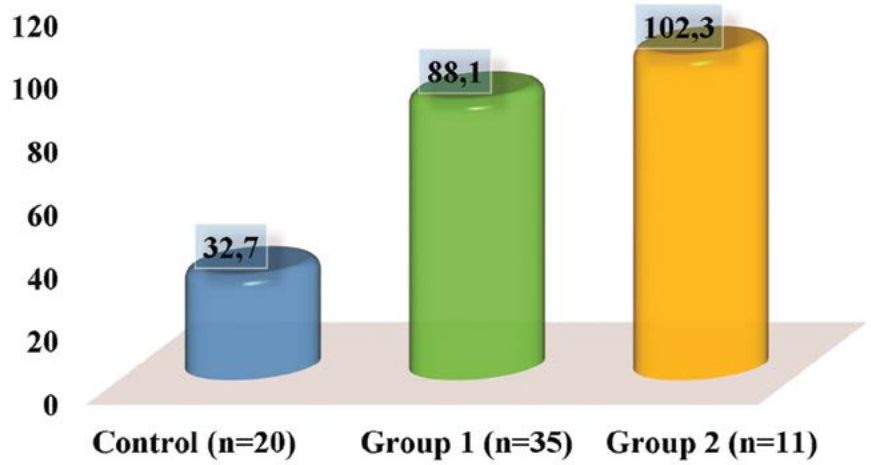
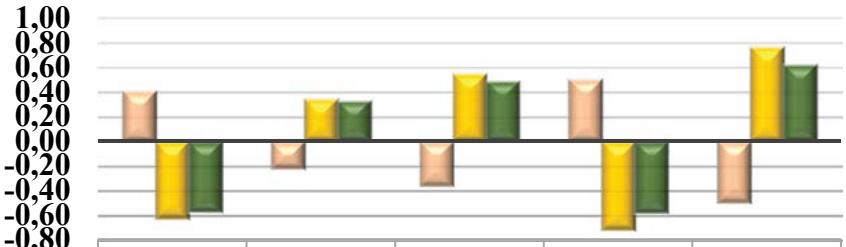


Fig. 3. Plasminogen activator inhibitor-1 levels in subjects (ng/ml)

In the control group, the PAI-1 level was 32.7 ± 2.1 ng/ml, corresponding to physiological parameters. In Group 1, PAI-1 was significantly elevated at 88.1 ± 5.6 ng/ml, exceeding the control value by a factor of 2.7 ($p < 0.01$). In Group 2, PAI-1 reached 102.3 ± 7.8 ng/ml, exceeding the control value by a factor of 3.1 ($p < 0.01$) and the Group 1 values by a factor of 1.2 (Fig. 3). This pattern indicates progressive suppression of fibrinolytic activity with worsening clinical presentation. The elevation of PAI-1 concentration in AAIO is attributable to a systemic inflammatory response, activation endothelial cells and macrophages, and production of proinflammatory cytokines –including IL-6 and TFN-α – which stimulate PAI-1expression. Under conditions of intestinal wall ischaemia and blood stagnation, additional activation of the coagulation system and suppression of fibrinolysis occurs, resulting in excessive fibrin formation in the abdominal cavity. In patients with complicated AAIO, including those with perforation, peritonitis, or intestinal necrosis, markedly greater PAI-1 hyperproduction is observed, which correlates with the severity of inflammation and the severity of microcirculatory disturbance.



	MNO	APTT (sec)	Fibrinogen (g/l)	tPA (ng/ml)	PAI-1 (ng/ml)
IFNy	0,41	-0,22	-0,36	0,50	-0,50
TNFa	-0,63	0,35	0,55	-0,72	0,77
IL-6	-0,58	0,33	0,49	-0,58	0,63

Fig. 4. Correlation relationships between cytokine levels and components of the haemostasis system (P≤0.05).

Correlation analysis demonstrated a moderate positive correlation between IFN-γ and INR (r = 0.41) and tPA (r = 0.50), whereas negative correlation with fibrinogen concentration (r = –0.36) and PAI-1 level (r = –0.50) was.

Correlation analysis showed that IFNγ has a moderate positive correlation with the INR level (r = 0.41) and tPA (r = 0.50), whereas negative correlations were identified with fibrinogen concentration (r = –0.36) and PAI-1 level (r = –0.50). These findings suggest that IFN-γ, as a Th1 response cytokine, may promote endothelial expression of tPA, thereby enhancing fibrinolysis. Concurrently, it exerts a weak inhibitory effect on the production of fibrinogen, an important coagulation substrate, and reduces PAI-1. These associations may be interpreted as a manifestation of compensatory hypofibrinogenaemia and a tendency towards increased fibrinolytic activity in the setting of inflammation.

Elevation of PAI-1, the principal inhibitor of the plasminogen-activating system, suppresses tPA activity, reduces the efficiency of fibrin dissolution, and predisposes to thrombosis. The negative association with INR confirms increased prothrombotic activity.

A similar pattern of associations was identified for TNF-α: negative correlations with INR (r = –0.63) and tPA (r = –0.72), and positive correlations with fibrinogen (r = 0.55), PAI-1 (r = 0.77) and APTT (r = 0.35) (Fig. 4). These results indicate the involvement of TNF-α in activation of the coagulation cascade and reduction of fibrinolytic activity, consistent with its established capacity to induce tissue factor expression and increase hepatic synthesis of acute-phase proteins, including fibrinogen. The positive association with APTT, despite the classical interpretation of this parameter as reflecting anticoagulant activity, may be attributable to non-specific factors such as consumption of coagulation factors in the microvascular bed.

IL-6 demonstrated similar, albeit less pronounced correlations: negative with INR (r = –0.58) and tPA (r = –0.58), and positive with PAI-1 (r = 0.63), fibrinogen (r = 0.49), and APTT (r = 0.33). As a universal mediator of the acute-phase inflammatory response, IL-6 stimulates fibrinogen production and may contribute to the imbalance between coagulation and fibrinolysis. These correlations confirm its role in potentiating the hypercoagulable state in complicated AAIO, particularly in the presence of a pronounced systemic inflammatory response.

The aggregate data indicate the presence of a subcompensated hypercoagulable state, most pronounced in patients with a complicated course of AAIO. The observed parameter dynamics reflect involvement of both extrinsic and intrinsic haemostasis pathways. Elevation of fibrinogen and PTI, together with prolongation of PT and APTT, indicate systemic activation of blood coagulation driven by tissue hypoxia, endothelial dysfunction, elevated proinflammatory cytokine levels, and release of tissue thromboplastin from necrotic intestinal segments [17]. These changes create conditions conducive to thrombosis and necessitate careful monitoring of the haemostasis system throughout the perioperative period. In patients with acute intestinal obstruction, particularly in the presence of complications, marked suppression of fibrinolytic activity is observed, which increases the risk of thrombosis, impairs blood rheology in the ischemic zone, and contributes to

further progression of tissue hypoxia [18]. PAI-1 may be regarded not only as a marker of disease severity but also as a potential therapeutic target in the prevention of postoperative adhesions and recurrent obstruction.

Conclusion.

Analysis of the associations between proinflammatory cytokines and components of the haemostasis system indicates the existence of two functional regulatory axes: (1) IFN-γ-mediated enhancement of fibrinolysis and reduction of fibrinogen, and (2) TNF-α/IL-6-mediated potentiation of coagulation and suppression of fibrinolytic activity. These differences may account for the variability of the clinical presentation in patients with complicated AAIO, particularly the predisposition to thrombotic or hemorrhagic complications, and may potentially serve as targets for personalised anticoagulant therapy.

Prospects for Further Research

Further studies should focus on expanding the sample size and conducting multicentre investigations to validate the identified immune-coagulative phenotypes in AAIO. Particular attention should be directed to dynamic monitoring of cytokine–haemostasis interactions in the perioperative period to determine their predictive value for thrombotic and ischaemic complications. The development of personalised anticoagulant and anti-inflammatory strategies based on individual biomarker profiles represents an area of considerable research potential. Experimental studies aimed at modulating PAI-1 activity represent a further productive direction that may provide new preventive approaches to reducing postoperative adhesion formation and recurrent obstruction.

Author Contributions: B. Khamdamov – conceptualisation of the study, supervision, and final approval of the manuscript; S. Safarov – clinical data collection, patient management, and statistical supervision; N. Fayzullaeva – laboratory immunobiochemical analysis and interpretation of cytokine data; U. Nasritdinov – data processing, statistical analysis, and preparation of figures; S. Davlatov – study design development, critical revision of the manuscript, scientific editing, and coordination.

All authors have reviewed the final version of the manuscript and have consented to its publication.

Conflict of Interest. The authors declare no conflict of interest regarding the publication of this article.

Acknowledgements. The authors express their sincere gratitude to the staff of the surgical departments and laboratory specialists of the clinic of the Tashkent State Medical University for their participation in patient recruitment, biological sample processing, and technical support provided during the study.

References:

1. Hasbahceci M, Basak F, Alimoglu O. Cecal volvulus. Indian Journal of Surgery. 2012;74(6):476-9. DOI: https://doi.org/10.1007/s12262-012-0432-9

2. Kurbanyazov Z, Yuldashov P, Rakhmanov K, Davlatov S, Khamdamov B, Yanchenko S. Enhancement of Surgical Treatment Methods for Postoperative Ventral Hernia: Experience of Laparoscopic Prosthetic Hernioplasty. Surgery Eastern Europe. 2025;14(2):268-77. DOI: https://doi.org/10.34883/PI.2025.14.2.027

3. Rakhmanov K, Hujamov O, Davlatov S, Khamdamov B, Yanchenko S, Abdullayeva N, et al. Innovative aspects of open inguinal hernia surgery: reducing complications and preventing recurrences. Neonatology, Surgery and Perinatal Medicine. 2025;2(56):150-62. DOI: https://doi.org/10.24061/2413-4260.XV.2.56.2025.21

4. Kapshytar OV, Kapshytar OO, Tantsura PY. Surgical treatment of acute adhesive small intestine obstruction. Kharkiv Surgical School. 2022;20(2):28-32. DOI: https://doi.org/10.37699/2308-7005.2.2022.05

5. Andersen P, Jensen KK, Erichsen R, Frøslev T, Krarup PM, Madsen MR, et al. Nationwide population-based cohort study to assess risk of surgery for adhesive small bowel obstruction following open or laparoscopic rectal cancer resection. BJS Open. 2017;1(2):30-8. DOI: http://doi.org/10.1002/bjs5.5 PMID: 29951603; PMCID: PMC5989974.

6. Gojayev A, Erkent M, Aydin HO, Karakaya E, Yildirim S, Moray G. Is laparoscopic surgery safe and feasible in acute adhesive ileus? Medicine (Baltimore). 2023;102(34): e34894. DOI: http://doi.org/10.1097/MD.0000000000034894 PMID: 37653802; PMCID: PMC10470780.

7. Borle DP, Agrawal N, Arora A, Kumar S, Chattopadhyay TK. Ileostomy site approach for adhesiolysis and laparoscopic cholecystectomy in a hostile abdomen: A novel technique. J Minim Access Surg. 2018;14(3):261-3. DOI: http://doi.org/10.4103/jmas.JMAS_73_17 PMID: 28782748; PMCID: PMC6001291.

8. Krytskyi I, Popovych D, Dzhyvak V, Boiko V, Hevko U, Bai A. Substantiation of the Importance of Rehabilitation for Children with Intestinal Obstruction in the Postoperative Period. Health of man. 2025;2:86-96. DOI: https://doi.org/10.30841/2786-7323.2.2025.337357

9. Dusu K, Dindyal S, Gadhvi V. Small bowel obstruction via herniation through an iatrogenic defect of the falciform ligament following laparoscopic cholecystectomy. Ann R Coll Surg Engl. 2015;97(6): e93-5. DOI: http://doi.org/10.1308/rcsann.2015.0022 PMID: 26274760; PMCID: PMC5126248.

10. Rami Reddy SR, Cappell MS. A Systematic Review of the Clinical Presentation, Diagnosis, and Treatment of Small Bowel Obstruction. Curr Gastroenterol Rep. 2017;19(6):28. DOI: http://doi.org/10.1007/s11894-017-0566-9 PMID: 28439845.

11. Attard JA, MacLean AR. Adhesive small bowel obstruction: epidemiology, biology and prevention. Can J Surg. 2007;50(4):291-300. PMID: 17897517; PMCID: PMC2386166.

12. Lee WK, Park YH, Choi S, Lee WS. Is liquid-based hyaluronic acid equivalent to sodium hyaluronate-based bioresorbable membrane to reduce small bowel obstruction in patients undergoing colorectal surgery. Asian J Surg. 2019;42(2):443-9. DOI: http://doi.org/10.1016/j.asjsur.2018.09.009 PMID: 30527555.

13. Madhavan S, Augustine A. Jejunojejunal intussusception: an unusual case of postoperative intestinal obstruction. Ann R Coll Surg Engl. 2018;100(7): e165-7. DOI: http://doi.org/10.1308/rcsann.2018.0079 PMID: 29909685; PMCID: PMC6214071.

14. Correa-Rovelo JM, Villanueva-López GC, Medina-Santillan R, Carrillo-Esper R, Díaz-Girón-Gidi A. Intestinal obstruction secondary to postoperative adhesion formation in abdominal surgery. Review. Cir Cir. 2015;83(4):345-51. DOI: http://doi.org/10.1016/j.circir.2015.05.024 PMID: 26116038.

15. Ouaïssi M, Gaujoux S, Veyrie N, Denève E, Brigand C, Castel Bet al. Post-operative adhesions after digestive surgery: their incidence and prevention: review of the literature. J Visc Surg. 2012;149(2): e104-14. DOI: http://doi.org/10.1016/j.jviscsurg.2011.11.006 PMID: 22261580.

16. Gu L, Zhu F, Xie T, Feng D, Gong J, Li N. Use of the Water-Soluble Contrast Medium Gastrografin in Treatment of Adhesive Small Bowel Obstruction in Patients with and Without Chronic Radiation Enteropathy: A Single-Center Retrospective Study. Med Sci Monit. 2021;27: e930046. DOI: http://doi.org/10.12659/MSM.930046 PMID: 33771966; PMCID: PMC8011282.

17. Llarena NC, Shah AB, Milad MP. Bowel injury in gynecologic laparoscopy: a systematic review. Obstet Gynecol. 2015;125(6):1407-17. DOI: http://doi.org/10.1097/AOG.0000000000000855 PMID: 26000512.

18. Mishchenko VV, Mishchenko VP, Rudenko IV, Goriachiy VV. Diagnostic and treatment approaches of acute adhesion intestinal obstruction in pregnant women and women in labor in the conditions of full-scale war in Ukraine. World of Medicine and Biology. 2025;21(92):99. DOI: http://doi.org/10.26724/2079-8334-2025-2-92-99-104

19. Samad S, Vatrika M, Basit R, Mangam S, Doughan S. Acute Mediastinitis Following a Laparotomy for Small Bowel Obstruction. Journal of Current Surgery. 2014;4(4):113-6. DOI: http://dx.doi.org/10.14740/jcs252w

20. Zhou P, Yang H, Wang J, Sun M, Yan S. Traditional Chinese medicine for adhesive intestinal obstruction: theory, methods and mechanisms of action. Front Med (Lausanne). 2025;12:1573655. DOI: http://doi.org/10.3389/fmed.2025.1573655 PMID: 40385573; PMCID: PMC12081424.

21. Zuparov KF, Fayzullayeva NY, Tursumetov AA. Justification of the expediency of using light propylene nets for onlay plastic surgery of postoperative ventral hernias. Trends in Clinical and Medical Sciences (TCMS). 2023;3(1):28-34. DOI: https://doi.org/10.30538/psrp-tmcs2023.0043

22. Tabibian N, Swehli E, Boyd A, Umbreen A, Tabibian JH. Abdominal adhesions: A practical review of an often overlooked entity. Ann Med Surg (Lond). 2017;15:9-13. DOI: http://doi.org/10.1016/j.amsu.2017.01.021 PMID: 28203370; PMCID: PMC5295619.

23. Ghimire P, Maharjan S. Adhesive Small Bowel Obstruction: A Review. J Nepal Med Assoc. 2023;61(260):390-6. DOI: http://doi.org/10.31729/jnma.8134 PMID: 37208871; PMCID: PMC10089019.

24. Jaanimäe L, Lepner U, Graumann K, Kryshevskaja A, Saar S, Kirsimägi Ü, Nikkolo C. Management of small bowel obstruction and the use of contrast media: a prospective observational cohort study. International Journal of Surgery Open. 2026; 64(1):1-7. DOI: http://doi.org/10.1097/IO9.0000000000000320

25. Mansurov TT, Daminov FA. Complex Diagnosis and Treatment of Patients with Acute Adhesive Intestinal Obstruction. Central Asian Journal of Medical and Natural Science. 2022;3(2):339-42. DOI: https://doi.org/10.51699/cajmns.v3i2.672

ІМУНОБІОХІМІЧНІ ОСОБЛИВОСТІ ГОСТРОЇ СПАЙКОВОЇ КИШКОВОЇ НЕПРОХІДНОСТІ

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Резюме.

Гостра спайкова кишкова непрохідність (ГСКН) є одним із найскладніших станів у абдомінальній хірургії, що характеризується високою частотою ускладнень та летальності.

Мета. Вивчити взаємодію прозапальних медіаторів, системи гемостазу та маркерів фібринолізу при гострій спайковій кишковій непрохідності.

Матеріали та методи. У дослідженні проаналізовано кореляційні зв'язки між рівнями прозапальних цитокінів (IFNγ, TNFα, IL-6), параметрами гемостазу (PT, INR, APTT, фібриноген) та маркерами фібринолізу (tPA, PAI-1) у 91 пацієнта з ГСКН, яких поділено на підгрупи з ускладненим та неускладненим перебігом, а також у 20 здорових осіб (контроль). Імуно-біохімічні показники визначали за допомогою ELISA. Усі пацієнти підписали інформовану згоду на участь у дослідженні. Протоколи дослідження були схвалені Етичним комітетом Інститутута відповідали принципам Гельсінської декларації, ухваленої у жовтні 2024 року на 75-й Генеральній асамблеї Всесвітньої медичної асоціації. Обробку отриманих даних проводили з використанням програмного забезпечення SPSS версії 26.0. Результати представлено у вигляді середнього арифметичного (M) ± стандартного відхилення (SD) або стандартної похибки середнього (m). Для оцінки вірогідності міжгрупових відмінностей застосовували параметричний критерій Стьюдента та непараметричний U-критерій Манна–Уїтні залежно від характеру розподілу даних. Рівень статистичної значущості встановлювали при $p < 0,05$. Робота виконана в рамках науково-дослідного плану Бухарського державного медичного інституту (05.2022 DSc.135) «Розробка нових підходів до ранньої діагностики, лікування та профілактики патологічних станів, що впливають на здоров'я населення Бухарської області після COVID-19 (2022-2026)».

Результати. Виявлено два різних імуно-коагуляційних фенотипи. IFNγ позитивно корелював з INR ($r = 0,41$) та tPA ($r = 0,50$), і негативно з фібриногеном ($r = –0,36$) і PAI-1 ($r = –0,50$), що свідчить про активацію фібринолізу та зниження коагуляційного потенціалу. Натомість TNFα та IL-6 асоціювалися з підвищенням рівня фібриногену та PAI-1, подовженням APTT, зниженням INR і tPA – що вказує на гіперкоагуляційний стан, пригнічення фібринолізу та ендотеліальну дисфункцію.

Висновок. Такі зміни були найбільш виражені у групі з ускладненим перебігом, що свідчить про високий ризик тромботичних подій та ішемічного ушкодження кишечника. Загалом дослідження підкреслює прогностичну цінність моніторингу маркерів гемостазу та фібринолізу при ГСКН і обґрунтовує їх застосування для раннього виявлення ускладнень та персоналізації антитромботичної терапії.

Ключові слова: спайкова кишкова непрохідність; фібриноліз; tPA; PAI-1; гемостаз; коагуляція; ускладнення; ішемія кишечника; біомаркери; імуно-біохімія.

ISSN 2226-1230 (PRINT) ISSN 2413-4260 (ONLINE)
KEY TITLE: NEONATOLOGÌÂ, HÌRURGÌÂ TA PERINATAL'NA MEDICINA (ONLINE)
ABBREVIATED KEY TITLE: NEONATOL. HÌR. PERINAT. MED. (ONLINE)

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Received by the editorial office: 10 January 2026.
Approved for publication: 23 February 2026.
Published: 27 March 2026.