Nexus Global Research Journal of Medical Sciences
Journal Homepage: https://nexusglobalresearch.com/index.php/ngrjms

Research Article:

Evaluation of Laboratory Changes During Long-Term Anticoagulant Use

Abdullayev Lazizbek [1], Ilxomova Ruxshona [2], Meyliyeva Bexruza[3], Tolibova Dilnoza[4]

[1,2,3,4] *Faculty of Medical Biology, Group 401, Samarkand State Medical University*

ABSTRACT:
As is often the case in medicine, the use of oral anticoagulants began by chance. The history of their discovery is associated with the diagnosis of an unusual hemorrhagic diathesis observed in cattle in North America and Canada in the early twentieth century. In the 1920s, studies emerged attributing this disease to prothrombin deficiency in the blood [1], which developed when cattle consumed spoiled hay [2]. An accurate diagnosis became possible only after A. Quick developed a method for determining prothrombin time in 1935 [3]. The results of this test demonstrated that fatal bleeding in animals was caused by a deficiency of prothrombin complex factors. The toxic substance isolated from spoiled hay was characterized as 4-hydroxycoumarin and named "dicumarol."

Keyword: Anticoagulants, coagulogram, background coagulation, thrombosis, coagulation factors.

Citation: Lazizbek A. *et al.* (2026). Evaluation of Laboratory Changes During Long-Term Anticoagulant Use. Nexus *Global Research Journal of Medical Sciences, 2*(6), 184–190.

INTRODUCTION

Bishydroxycoumarin was obtained as a medicinal substance in 1940 by the American biochemist K. Link, who named it "dicumarol" [6]. Almost simultaneously, studies conducted by N. Dam demonstrated that chickens fed a diet lacking the ether-soluble fraction also developed hemorrhagic manifestations associated with prothrombin deficiency [7].

The substance missing from the diet was named vitamin K (coagulation vitamin). At the same time, vitamin K was shown to be an effective antidote for prothrombin deficiency caused by bishydroxycoumarin [8].

Dicumarol soon became the precursor of numerous derivatives, all of which exhibited anticoagulant effects through antagonistic interactions with vitamin K. This group of antithrombotic drugs became known as vitamin K antagonists (VKAs), or indirect anticoagulants.

Table 1. Dosages and elimination half-lives of oral anticoagulants

Drug	Elimination half-life (hours)	Mean maintenance dose (mg/day)	Maintenance dose range (mg/day)
Warfarin	42	6	1–20
Phenprocoumon	160	3	0.75–6
Dicumarol	24–100	75	25–150
Ethyl biscoumacetate	2.5	450	150–900
Phenindione	5	100	25–200
Phenylin	5	75	50–150

Anticoagulants, like many other medications, pass through three stages during their introduction into clinical practice:

1. The stage of general adoption and reassessment of therapeutic efficacy.

***Corresponding Author:** Abdullayev Lazizbek
Received: 30 May 2026 | **Accepted:** 20 Jun 2026 | **Published:** 26 Jun 2026
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2. The stage of identifying limitations and disadvantages.
3. The stage of determining the therapeutic significance of treatment strategies and methods, as well as resolving numerous issues on the basis of clinical experience and specialized research.

However, despite more than 50 years of extensive clinical experience with vitamin K antagonists, several issues remain unresolved. The indications for their use continue to be debated.

Currently, the effectiveness of VKAs in the treatment of venous thrombosis and pulmonary embolism is no longer questioned. In the latter condition, long-term treatment with vitamin K antagonists is primarily indicated to prevent recurrent episodes of thromboembolism. These medications are successfully used to prevent thromboembolic complications in atrial fibrillation of various origins [19, 20].

The necessity of administering VKAs to patients following cardiac surgery has been emphasized by both domestic and international researchers. Their effectiveness in preventing thromboembolic complications following the implantation of artificial heart valves has also been demonstrated [21].

Oral anticoagulants have also become an integral component of measures aimed at preventing postoperative thrombosis. In this form of thromboprophylaxis, low-dose heparin or low-molecular-weight heparin is given priority in certain clinical situations, including plastic surgery and other operations accompanied by extensive destruction of thigh tissues and substantial tissue separation. Numerous clinical studies conducted in this patient population have demonstrated the superior prophylactic effect of vitamin K antagonists compared with heparin [22]. Although the majority of researchers have reached a favorable conclusion regarding VKA administration in all the clinical situations listed above, physicians both domestically and internationally continue to discuss the appropriateness of prescribing this group of medications for cardiovascular diseases.

Oral anticoagulants were first used for the treatment of myocardial infarction [13]. Since then, numerous clinical studies have been conducted to determine the effectiveness of oral anticoagulants in this patient population. Their findings have both supported their use and failed to demonstrate the effectiveness of VKAs. Only a comprehensive retrospective analysis of numerous clinical observations made it possible to identify several methodological errors in their implementation, as a result of which the beneficial effects of VKAs were often obscured. These errors concerned the principles of patient selection, the appropriate formation of control groups, the number of patients and duration of follow-up, as well as the criteria used to assess the effectiveness of oral anticoagulant treatment and the maintained degree of hypocoagulation. Thus, Chalmers et al. (1977) analyzed 32 clinical studies conducted in various countries involving patients receiving VKAs during the hospital phase of myocardial infarction and concluded that these medications reduced mortality by 20%.

The researchers concluded their work with the following statement:

"It is difficult to imagine that concerns regarding hemorrhagic complications could overshadow a small but statistically significant reduction in mortality, as well as a clinically meaningful and statistically significant reduction in thromboembolic complications associated with the use of vitamin K antagonists in patients with acute myocardial infarction." Subsequent long-term VKA treatment in patients with myocardial infarction also confirmed their effectiveness. According to several authors, after five years of oral anticoagulant therapy in these patients, overall mortality decreased twofold and the frequency of recurrent myocardial infarction decreased threefold compared with the control group.

Table 2. Recommended therapeutic INR values during oral anticoagulant therapy

Indications	BSH (1990)	NHLBI (1989)
Postoperative deep vein thrombosis (general surgery)	2.0–2.5	2.0–3.0
Postoperative deep vein thrombosis in surgical practice	2.0–3.0	2.0–3.0
Prevention of venous thromboembolism in myocardial infarction	2.0–3.0	2.0–3.0
Treatment of venous thrombosis	2.0–3.0	2.0–3.0
Treatment of pulmonary embolism	2.0–3.0	2.0–3.0
Prevention of transient ischemic attacks	2.0–3.0	—
Prevention in patients with tissue heart valves	2.0–3.0	2.0–3.0
Prevention in atrial fibrillation	2.0–3.0	2.0–3.0

Prevention in valvular heart disease	2.0–3.0	2.0–3.0
Prevention in arterial diseases	3.0–4.5	2.0–3.0
Prevention in patients with mechanical prosthetic heart valves	3.0–4.5	3.0–4.5
Prevention of recurrent systemic embolism	3.0–4.5	3.0–4.5

Note: NHLBI — National Heart, Lung, and Blood Institute; BSH — British Society for Haematology.

The clinical significance of reduced antithrombin III levels and recent developments in their treatment have been recognized as another indication for VKA administration. An increase in antithrombin III levels has been observed during VKA treatment. This phenomenon has been described in patients with both hereditary and acquired deficiencies [25]. These observations provided a basis for recommending the prophylactic and therapeutic use of VKAs in patients with acute nephrotic syndrome, in whom antithrombin III levels are also reduced.Undoubtedly, as our understanding of the mechanisms of thrombus formation and their role in the development and progression of various diseases continues to deepen, the indications for VKA use will expand.

MECHANISM OF ACTION OF ORAL ANTICOAGULANTS

When describing the mechanism of action of oral anticoagulants, it should be emphasized that, unlike other medications that inhibit fibrin formation (heparin and defibrinating agents), vitamin K antagonists interfere with the formation of coagulation factors III, IX, and X in hepatocytes.

These medications share several characteristics: they do not affect blood outside the body, they are effective when administered orally, their action is mediated through proteins of the prothrombin complex, and they have a common antagonist, vitamin K.

Their antithrombotic effect generally becomes apparent after an average of 3–5 days. Although factor VII, which has the shortest half-life, begins to disappear from plasma within several hours under the influence of VKAs, the complete antithrombotic effect develops only when the concentrations of all four vitamin K-dependent coagulation factors decrease [28]. Considering the half-lives of the remaining three factors (3–4 days for prothrombin and 1 and 2 days for factors IX and X, respectively), the antithrombotic effect can be expected several days after the initiation of treatment.

These observations regarding the initial dose and type of VKA made it possible to abandon the use of loading doses, which, as demonstrated, provided no additional benefit and only caused a sharp decrease in factor VII levels during the first 48 hours, resulting in hemorrhagic complications [29].

According to their chemical structure, indirect anticoagulants can be divided into four subgroups: monocoumarin, dicoumarin, cyclocoumarin, and indandione derivatives. From a clinical perspective, these medications differ in their gastrointestinal absorption, elimination half-life, and frequency of adverse effects. Table 1 presents the characteristics of the most widely used vitamin K antagonists worldwide. Experience with warfarin use in Russia has been limited, although it is frequently prescribed abroad because of its generally favorable tolerability and optimal duration of action, allowing once-daily administration.

The disadvantages of many coumarin derivatives include difficulties in monitoring their anticoagulant effects because of their excessively long (phenprocoumon) or excessively short (ethyl biscoumacetate) half-lives and incomplete gastrointestinal absorption (dicumarol).

Indandione derivatives, represented domestically by phenylin, which is prescribed to the majority of patients, have anticoagulant effects similar to those of coumarins. However, according to J. Perkins [30], they should not be used frequently because of the adverse effects they cause, including various allergic skin manifestations and toxic effects on the liver. Furthermore, phenindione metabolites may cause the urine to acquire a pink or orange coloration, potentially misleading patients and physicians into suspecting hematuria. The latter can be excluded if the coloration disappears after adding several drops of diluted acetic acid to the urine [31]. The difficulty of VKA therapy is associated with the need to determine an optimal drug dose that prevents thrombus formation without causing bleeding.

At the same time, the optimal dose is strictly individualized and depends on the patient's sex, age, diet, concomitant diseases, medications taken simultaneously, and other factors. The causes of these variations appear almost unlimited, and the optimal VKA dose may change during treatment, even in the same patient. Because optimal hypocoagulation must be maintained throughout

the entire treatment period, the importance of laboratory monitoring is beyond question. Throughout the clinical use of VKAs, the principal monitoring method has been, and remains, the determination of prothrombin time (PT) according to A. Quick (1937) or its modifications, including the P and P test and the Thrombotest method.

This method is based on the activation of blood plasma by a tissue extract known as thromboplastin. This reaction involves factors of the extrinsic coagulation cascade, whose concentrations are primarily reduced under the influence of VKAs. Currently, approximately 30 different thromboplastins are used in this test to activate blood or plasma coagulation. They are obtained from various sources (human brain, rabbit or bovine brain, and others) and differ in their sensitivity to coagulation defects induced by VKAs. They are also inhibited to varying degrees by PIVKA (proteins induced by vitamin K absence). This accounts for significant differences in PT results obtained by different laboratories and makes it difficult to establish a single optimal level of hypocoagulation.

Additional difficulties have arisen because of the existence of various methods for expressing PT results, particularly:

1. Prothrombin ratio: the ratio of the patient's PT to the donor's PT.

2. Percentage of prothrombin activity: calculated from a dilution curve using saline or adsorbed plasma.

3. Prothrombin index: the reciprocal of the prothrombin ratio multiplied by 100%.

Intensive efforts to standardize the method of PT determination have yielded positive results. On the recommendation of the World Health Organization (WHO), the most widely used thromboplastins worldwide were calibrated [32], and a unified standard scale was established to express the intensity of hypocoagulation. The standardized indicator of PT results was designated the International Normalized Ratio (INR). Reference plasmas prepared artificially [32, 33] and obtained from the blood of patients receiving VKAs [32, 34, 35] began to be used to assess the degree of hypocoagulation. Calibration constants known as the International Sensitivity Index (ISI), determined during the standardization of thromboplastins, made it possible to compare PT results regardless of the thromboplastin used.

A similar standardization of the most commonly used thromboplastins was performed in 1983 [36]. The ISI values of these thromboplastins were found to be very close to the international standard, British Comparative Thromboplastin (BCT), and were slightly greater than one [30]. The standardization of methods for monitoring VKA therapy enabled researchers worldwide to confirm once again that an appropriately selected dose is one of the principal factors influencing treatment effectiveness.

At present, the dependence of the antithrombotic effect of oral anticoagulants on the degree of hypocoagulation has been clearly demonstrated. An analysis comparing PT values with the incidence of myocardial infarction showed that the recurrence rate did not exceed 50% only when prothrombin activity decreased to 15–25%. However, when prothrombin activity exceeded 28%, the recurrence rate reached 100% [32].

Studies aimed at determining the optimal levels of monitoring tests for preventing pulmonary embolism in patients with venous thrombosis receiving VKAs established that the frequency of thromboembolism was 2.5 times lower when prothrombin activity was within the range of 10–29% compared with values of 30–49% [19]. According to L. Poller, during VKA treatment, the frequency of venous thrombosis decreases from 29.7% in the control group to 2.8% when the INR is maintained at 2.0–2.5, whereas it increases to 8.5% when low doses of warfarin (1 mg) are prescribed [20].

During long-term follow-up of patients with artificial heart valves, the frequency of thromboembolic complications was three times lower among patients whose PT values remained within the therapeutic range for most of the observation period (at least 75%) compared with patients who received insufficient treatment [21]. Numerous studies conducted in this field have made it possible to establish optimal levels of hypocoagulation that ensure maximum efficacy and safety during VKA therapy in patients with various diseases. Table 2 presents the INR values recommended by the British Society for Haematology (1990) in the left column and the National Heart, Lung, and Blood Institute (1989) in the right column, together with the prothrombin index used in Russia.

ORAL ANTICOAGULANT THERAPY

Despite the difficulties associated with this type of antithrombotic therapy, the following practical recommendations may be helpful for physicians who decide to initiate VKA treatment. In the absence of contraindications (an increased tendency to bleeding due

to various causes, recent operations involving the central nervous system and eyes, diastolic blood pressure exceeding 110 mmHg, and ulcerative diseases of the gastrointestinal tract), VKAs are prescribed at a dose slightly higher than the average daily dose.

During the first week of treatment, PT is determined daily; during the second week, twice weekly; and subsequently, once laboratory parameters have stabilized, approximately once every four weeks. If PT results in a patient with a stable level of hypocoagulation differ slightly from the optimal range, the medication dose should not be changed immediately.

The next laboratory examination should be scheduled somewhat earlier than the usual monitoring interval, and the dose should be adjusted only if repeated deviations from the desired range are observed.

If the patient's monitoring parameters are unstable, efforts should be made to identify the underlying causes. Most frequently, these are associated with dietary changes, initiation of new medications or discontinuation of long-term medications, excessive alcohol consumption, exacerbation of the underlying disease, and other factors.

In our opinion, during long-term VKA treatment, patients should undergo urine examinations to determine the number of erythrocytes in addition to PT monitoring. The appearance of microhematuria (more than 10–15 erythrocytes per microscopic field of view) is an early sign of anticoagulant overdose. Hemorrhagic complications most frequently occur in the gastrointestinal tract, urinary tract, skin, and nasal mucosa. Most fatal bleeding episodes involve the gastrointestinal tract or intracranial structures [1].

Treatment of hemorrhagic complications depends on their severity. In cases of hypocoagulation detected only through laboratory investigations, treatment should be temporarily discontinued for several days, taking into account the type of anticoagulant and its elimination half-life. Subsequently, therapy should be resumed at a reduced dose. Life-threatening bleeding requires the immediate transfusion of prothrombin complex factor concentrate, fresh frozen plasma, or whole blood (500 mL). In more favorable circumstances, vitamin K1 may be administered intravenously at a dose of 25–50 mg, which restores complete hemostasis within 6–24 hours but makes the patient resistant to anticoagulant therapy for the following two weeks. A dose of 5–15 mg reduces the manifestations of hypocoagulation while allowing VKA treatment to be continued when necessary. Vitamin K1 preparations are also available for intramuscular and oral administration. It should be emphasized that the antidote to indirect anticoagulants is vitamin K1 (Kanavit, Konakion), not vitamin K3 (Vikasol). The latter is rapidly eliminated from the body within 40 minutes and does not have sufficient time to displace the anticoagulant from its complex with the apoenzyme. Resistance to VKAs is extremely rare and, in most cases, is apparent rather than genuine, resulting from incorrect administration or drug interactions.

Sometimes, the consumption of foods rich in vitamin K may contribute to this effect, including green leafy vegetables, particularly root vegetables and cauliflower. However, all problems associated with VKA therapy can be resolved if the physician and patient function as a single, unified team consisting of a responsible physician and a compliant patient. Only through genuinely close cooperation between these parties can treatment be made as effective and safe as possible.

REFERENCES:

1. Roderick LM. The pathology of sweet clover disease in cattle. Journal of the American Veterinary Medical Association. 1929;74:314.
2. Schofield FW. Damaged sweet clover. The cause of a new disease in cattle simulating hemorrhagie septicemia and blackleg. Journal of the American Veterinary Medical Association, 1924;64:553.
3. Quick AJ, Leu M. Quantitative determination of prothrombin. Journal of Biological Chemistry, 1937;119:81.
4. Campbell HA, Smith WK, Roberts WL, Link KP. Studies on the hemorrhagic sweet clover disease. The bioassay of the hemorrhagic concentrates by following the prothrombin level in the plasma of rabbit blood. - J Biol Chem, 1941;138:1.

5. Stachmann MA. Huebner CF, Link KP. Studies on the hemorrhagic sweet clover disease. Identification and synthesis of the hemorrhagic agent. - J Biol Chem. 1941;138:513.
6. F.X.Mamatkulova., X.I.Axmedov. Temir tanqisligi kamqonligining kelib chiqish sabablari va davolashga zamonaviy yondoshuv. “SCIENCE AND EDUCATION” VOLUME 4, ISSUE1.2023/195-203
7. Dadajonov, U., Abdiyev, K., Mamatkulova, F., & Dadajonov, U. (2021). Innovatsionniye metodi lecheniya immunnoy trombotsitopenicheskoy purpuri u lits molodogo vozrasta. Obshestvo i innovatsii, 2(4/S), 52-56.
8. Mamatkulova F. X. Mamatova N. T. Ruziboeva.O. N. Prevention of Anemia In Patients With Tuberculosis. The American Journal of Medical Sciences and Pharmaceutical Research, 2(11), 62–65.
9. L. S. Makhmonov., F. Kh. Mamatkulova., M. B. Berdiyarova, K.E. Shomurodov.THE MAIN CAUSES OF ANEMIA IN IRON AND VITAMIN B 12 DEFICIENCY ASSOCIATED WITH HELICOBACTER PYLORI
10. Makhmonov L. S., Mamatkulova F. Kh., Kholturaeva D. F., Muyiddinov Z. Z. IMPORTANCE OF DETECTION OF HEPSIDINE AND INTERLEUKINS IN "Science and Education" Scientific Journal / Impact Factor 3,848 (SJIF) February 2023 / Volume 4 Issue 2.
11. Mamatkulova F. X. Mamatova N. T. Ruziboeva.O. N. Prevention Of Anemia In Patients With Tuberculosis. The American Journal of Medical Sciences and Pharmaceutical Research, 2(11), 62–65.
12. L. S. Makhmonov., F. Kh. Mamatkulova., M. B. Berdiyarova., K.E. Shomurodov.THE MAIN CAUSES OF ANEMIA IN IRON AND VITAMIN B 12 DEFICIENCY ASSOCIATED WITH HELICOBACTER PYLORI
13. Makhmonov L. S., Mamatkulova F. Kh., Kholturaeva D. F., Muyiddinov Z. Z. IMPORTANCE OF DETECTION OF HEPSIDINE AND INTERLEUKINS IN IRON DEFICIENCY ANEMIA. Asian Journal of Multidimensional Research ISSN: 2278-4853 Vol. 11, Issue 4, April 2022
14. Dadajanov U. D., Mamatkulova Feruza Xaydarovna, R. Oyjamol N. Features Of Thrombophilia In Covid-19 European Journal of Molecular & Clinical Medicine2020/12/26. 07/03
15. Mamatkulova Feruza Khaydarovna, Akhmedov Husan Isrofilovich, Abdiev Kattabek Makhmatovich. Essential Thrombocythemia - Principal Analysis in Children and Adolescents. JOURNAL OF INTELLECTUAL PROPERTY AND HUMAN RIGHTS Volume: 2 Issue: 10 | Oct – 2023 ISSN: 2720-6882. 23-29.
16. Maxmonov L.S., Mamatqulova F.X., Holiqulov B.Y. Gemorragik diatezlar bilan kasallangan ayollarda tuxumdon apopleksiyasi asoratini davolash tamoyillari //Science and Education. – 2022. – Т. 3. – №. 12. – С. 237-244.17. Barkagan Z.S. // Gematologiya bo’yicha qo’llanma / Ed. Vorobieva A.I. - M., 1985. - T. 2. - S. 337–338.
17. Abdiyev K.M., Dadajanov U.D., Mamatkulova F.X. Nekotoriye aspekti vedeniya bolnix s trombotsitopenicheskoy purpuroy oslojnennoy s apopleksiyey yaichnika. Problemi ekologii, zdorovya, farmatsii i parazitologii. Nauchniye trudi. Moskva. 2013 g. Str. 372-373.
18. Makhmonov L.S., Sh. Koraboev S.K., Gapparova N..Sh, Mamatkulova F. Kh. Early diagnosis and treatment of funicular myelosis in v12 deficiency anemia. Asian Journal of Multidimensional Research Year : 2022, Volume : 11, Issue : 5.First page : (369) Last page : (373)Online ISSN : 2278-4853.
19. Mamatkulova F.X., Alimov O.E., Namozov M.N.O‘. Abdominal jarroxlik operatsiyalardan keyingi davrda regional anesteziyaning samaradorligi va rivojlangan kamqonlikni davolash //Science and Education. – 2023. – Т. 4. – №. 2. – С. 445-452.
20. Маматкулова Ф. Х., Ахмедов Х. И. Темир танқислиги камқонлигининг келиб чиқиш сабаблари ва даволашга замонавий ёндошув //Science and Education. – 2023. – Т. 4. – №. 1. – С. 195-203.
21. MF Khaydarovna, AH Isrofilovich, AK Makhmatovich Essential Thrombocythemia-Principal Analysis in Children and Adolescents. Journal of Intellectual Property and Human Rights 2 (10), 23-29
22. Mamatkulova F.Kh. Shomurodov K.E.,Temirov N. N. Significance. Of Helicobacter Pylori In Iron Deficiency. International Journal for Research in Applied.Science & Engineering

Technology (IJRASET)ISSN: 2321-9653; Volume.9 Issue XII Dec.2021.https://doi.org/10.22. 214/ijraset.2021.39443. 1103-1106

23. Maxmonov L.S.,Mamatqulova F.X.,Holiqulov B.Y.Trombotsitopatiya bilan kasallangan ayollarda tuxumdon apopleksiyasi asoratini davolash tamoyillari Biologiya va tibbiyot muammolari 2022, №1.UDK: 615.3:617.01.134 ISSN 2181-5674 61-67s.
24. Махмонов, Л., Маматкулова, Ф., Абдиев, К., & Амерова, Д. (2021). The importance of using clinical audit in teaching the subject of hematology. Общество и инновации, 2(6), 215-221.
25. L.S. Makhmonov, FK Mamatkulova, MB Berdiyarova, KE Shomurodov. The main causes of anemia in iron and vitamin b 12 deficiency associated with helicobacter pylori.Nveo-natural volatiles & essential oils Journal| NVEO, 10167- 10174I ВЕКА. Ст.41
26. KM Abdiev, FK Mamatkulova, KM Shomirzaev.STRUCTURE OF COMORBIDITY IN IDIOPATHIC THROMBOCYTOPENIC PURPLE ACADEMICIA: An International Multidisciplinary Research Journal 12 (12), 52-56
27. Abdiyev K. M., Mamatkulova F. X., Shomirzayev X. M. Immun trombotsitopenik purpurani davolashning innovatsion va noanananaviy usullari //Science and Education. – 2023. – Т. 4. – №. 1. – S. 228-234.
28. Abdiev Kattabek Makhmatovich, Mamatkulova Feruza Khaydarovna. Structure of comorbidity in idiopathic thrombocytopenic purple SKM ACADEMICIA: An International Multidisciplinary Research Journal 22 (12), 56-60
29. U.D DADAJONOV, KM ABDIEV, FX MAMATKULOVA. Innovative methods of treatment of immune thrombocytopenic purpura in young people Society and innovations, 52-56 Society and innovations, 52-56
30. Mamatkulova F. X., Usmonqulov J. Sh. O‘. Vitamin V12 kamqonligi va uni davolash //Science and Education. – 2023. – Т. 4. – №. 2. – S. 252-259.
31. Maxmonov, L., Mamatkulova, F., Abdiyev, K., & Amerova, D. (2021). The importance of using clinical audit in teaching the subject of hematology. Obshestvo i innovatsii, 2(6), 215-221.
32. Маматкулова Ф. Х., Алимов О. Э., Намозов М. Н. Ў. Абдоминал жаррохлик операциялардан кейинги даврда регионал анестезиянинг самарадорлиги ва ривожланган камқонликни даволаш //Science and Education. – 2023. – Т. 4. – №. 2. – С. 445-452.
33. Maxmonov, L. S., Mamatqulova, F. X., & Meliqulov, B. S. (2023). Trombotsitopatiya bilan kasallangan ayollarda tuxumdon apopleksiyasi kechishi va asoratini davolash tamoyillariga zamonaviy yondashuv. Science and Education, 4(2), 384-391.