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ПРЕДИКТОРЫ 28-ДНЕВНОЙ ЛЕТАЛЬНОСТИ ПРИ ЦИРРОЗЕ ПЕЧЕНИ

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Clinical and Laboratory Characteristics and Mortality in Patients with Liver Cirrhosis

Резюме

Цель: Определение независимых клинико-лабораторных предикторов 28-дневной летальности у пациентов с циррозом печени (ЦП). **Материалы и методы:** В проспективное когортное исследование включены 137 пациентов с циррозом печени (декомпенсированный цирроз без Acute-on-Chronic Liver Failure (ACLF), n=72; с ACLF, n=65). Для выявления независимых предикторов 28-дневной летальности использовался однофакторный и многофакторный регрессионный анализ Кокса. Были построены и сравнены три модели: комплексная (все значимые переменные), базовая клиническая (CLIF-C OFs, SpO_2/FiO_2, пневмония, стадии ACLF) и расширенная лабораторная (базовая модель + лактат, аммиак, С-реактивный белок (СРБ)). **Результаты:** В однофакторном анализе значимыми предикторами летальности были степень тяжести по шкале CLIF-C OFs, стадии ACLF 2 и 3, наличие пневмонии и инфекции мочевыводящих путей (ИМП), повышенные уровни лактата, аммиака, СРБ и трансаминаз, а также снижение SpO_2/FiO_2. Во всех многофакторных моделях независимыми предикторами неблагоприятного исхода оставались: CLIF-C OFs, стадии ACLF 2 и ACLF 3, наличие пневмонии, ИМП, повышенный уровень лактата, аммиака, СРБ, снижение SpO_2/FiO_2. **Заключение:** Краткосрочный прогноз при ЦП определяется тяжестью органной недостаточности, инфекционными осложнениями и маркерами метаболического стресса и воспаления. Для стратификации риска необходим комплексный подход с использованием шкал CLIF-C OFs и мониторингом лактата, аммиака и СРБ, что позволит оптимизировать ведение пациентов.

Ключевые слова: *цирроз печени, острая печеночная недостаточность на фоне хронической (ACLF), декомпенсация цирроза печени*

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Abstract

Background: To identify independent clinical and laboratory predictors of 28-day mortality in patients with liver cirrhosis. **Materials and Methods:** A prospective cohort study included 137 patients with liver cirrhosis (decompensated cirrhosis without ACLF, n=72; with ACLF, n=65). Univariate and multivariate Cox regression analysis was used to identify independent predictors of 28-day mortality. Three models were built and compared: a comprehensive model (all significant variables), a basic clinical model (CLIF-C OFs, SpO_2/FiO_2, pneumonia, ACLF stages), and an extended laboratory model (basic model + lactate, ammonia, C-reactive protein). **Results:** In the univariate analysis, significant predictors of mortality were the severity according to the CLIF-C OFs score, ACLF grades 2 and 3, the presence of pneumonia and urinary tract infection (UTI), elevated levels of lactate, ammonia, C-reactive protein (CRP) and transaminases, as well as decreased SpO_2/FiO_2. In all multivariate models, the following remained independent predictors of an unfavorable outcome: CLIF-C OFs score, ACLF grades 2 and 3, presence of pneumonia, UTI, elevated levels of lactate, ammonia, CRP, and decreased SpO_2/FiO_2. **Conclusion:** The short-term prognosis in liver cirrhosis is determined by the severity of organ failure, infectious complications, and markers of metabolic stress and inflammation. A comprehensive approach using the CLIF-C OFs score and monitoring lactate, ammonia, and CRP is necessary for risk stratification, which will allow for optimized patient management.

Key words: *liver cirrhosis, acute-on-chronic liver failure (ACLF), decompensation of liver cirrhosis.*

Conflict of interests

Co-author of the article Nikitin I.G. is a member of the editorial board of the journal «The Russian Archives of Internal Medicine». The article has passed the peer-review procedure adopted by the journal. Nikitin I.G. did not participate in the decision to publish this article. The authors did not declare any other conflicts of interest

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Conformity with the principles of ethics

The study protocol was approved by the Local Ethics Committee of N.I. Pirogov Russian national research medical university (Approval No. 235, December 18, 2023). Informed consent was obtained from all patients who participated in the study.

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ALT — alanine aminotransferase, AST — aspartate aminotransferase, DLC — decompensated liver cirrhosis, UTI — urinary tract infection, CRP — C-reactive protein, MBP — mean blood pressure, LC — liver cirrhosis, ACLF — Acute-on-Chronic Liver Failure, CLIF-C OFs — CLIF Consortium Organ Failure score, HBV — Hepatitis B Virus, PAMPs — Pathogen-Associated Molecular Patterns

Introduction

Acute-on-chronic liver failure (ACLF) is one of the most severe complications of liver cirrhosis (LC) characterized by the multi-organ failure and high short-term mortality. Despite predictive models, the structure and significance of clinical and laboratory death predictors in patients with decompensated liver cirrhosis (DLC) without ACLF remain rather indefinite.

The results of the first part of our study demonstrated a significant contribution of infectious and metabolic factors into DLC and ACLF mortality. Due to this, an issue of complex risk stratification and validation of multifactorial predictive models remains important.

LC is one of the leading causes of death globally, while ACLF determines the poor prognosis in this patient category.

According to the "Global, Regional, and National Cirrhosis Burden 2017" International Study, Russia holds a fourth place globally considering the LC mortality increment, in which alcoholic liver injuries play the key role [1].

Risk stratification and prediction of outcomes in such patients are an important, but complex objective for clinical physicians.

Considering the increasing LC mortality, the detection of the corresponding associated factors is of utmost significance.

Study objective: determining independent clinical & laboratory 28-day mortality predictors in patients with LC.

Study Materials and Methods

The study was arranged in the State Budget Healthcare Institution "V.M. Buyanov City Clinical Hospital" (G.I. Storozhakov Department of Hospital Therapy of the Institute of Clinical Medicine). The analysis included 137 patients with LC (76 males and 61 females; mean age 50 [42–58] years) hospitalized within the period from October 2023 till April 2025.

Inclusion criteria: males and females aged 18 to 75 years with LC that signed the informed consent for the study participation and anonymous publication of results.

Non-inclusion criteria: severe decompensated somatic extrahepatic diseases; psychic diseases; a malignancy detected in the patient before or during the hospitalization; patients not signing the informed consent.

The diagnosis was established based on the clinical and laboratory-instrumental data. According to the CLIF-C OF (organ dysfunction) score, patients were divided into two groups — the main group (n=72, mean age 52±11 years, 37 (51.4 %) males)) with ACLF and the reference group (n=65, mean age 49±11 years, 38 (58.5 %) males)) with the DLC without ACLF.

To assess the independent contribution of clinical, laboratory, and infectious factors into the 28-day mortality prognosis, a Cox proportional-hazard regression model was used. The analytical approach included two steps: first, a univariate analysis was arranged for all potentially significant variables (demographic, clinical parameters, laboratory markers, infections present); variables with the significance level of $p < 0.1$ in the univariate analysis together with clinically significant factors determined by experts were selected for the multivariate analysis.

Three models were prepared for the multivariate regression analysis: a complex model (all variables with $p < 0.1$); a basic clinical model (CLIF-C OFs score, SpO_2/FiO_2, presence of pneumonia, ACLF stage); an advanced laboratory model (markers of inflammation and metabolic disorders: lactate, ammonia, C-reactive protein (CRP) added to the clinical model). The model was optimized step-by-step using the Wald's backward method. The entrance and exclusion criteria were $p < 0.1$ and $p > 0.15$, respectively. The hazard ratio (HR) was calculated for all models with 95 % confidence intervals and p values; values of $p < 0.05$ were considered statistically significant.

Results

Baseline clinical and laboratory characteristics of groups compared (DLC without ACLF and ACLF of variable severity) are specifically described in the first part of the publication.

Table 1 describes the results of uni- and multivariate Cox regression analyses arranged to detect factors associated with the risk of poor outcomes in patients. The analysis included demographic, clinical, and laboratory parameters, as well as infectious complications and severity based on the CLIF-C OFs score.

Considering the univariate Cox analysis, statistically significant factors associated with the increased risk included CLIF-C OFs parameters (HR 1.582; 95 % CI 1.291–1.938; $p < 0.001$) and ACLF stages determined based on the CLIF-C OFs score: the risk was significantly elevated in patients with ACLF 2 (HR 4.567; 95 % CI 1.546–13.493; p=0.006) and ACLF 3 (HR 7.382; 95 % CI 2.325–23.439; p=0.001) vs. decompensated patients without ACLF. The following factors also increased the risks of death: presence of pneumonia (HR 5.049; 95 % CI 1.103–23.098; p=0.037), urinary tract infections (UTIs) (HR 3.004; 95 % CI 1.230–7.339; p=0.016), elevated levels of lactate (HR 1.209; 95 % CI 1.031–1.418; p=0.020), ammonia (HR 1.010; 95 % CI 1.003–1.018; p=0.006), CRP (HR 1.012; 95 % CI 1.003–1.020; p=0.006), and transaminases (ALT and AST).

An inverse correlation was detected for SpO_2/FiO_2 (HR 0.994; 95 % CI 0.990–0.998; p=0.009): each index increase by 10 units decreased the risk of death by 6 %. A trend to the increased risk of death was observed for repeated hospitalizations (HR 1.482 [0.508–1.716]; p=0.088) and ascites on admission (HR 0.986 [0.327–2.972]; p=0.091).

The complex multivariate model included all variables with $p < 0.1$ based on the univariate analysis results. In this model, the independent predictors of poor outcomes still included CLIF-C OFs (HR 1.545; 95 % CI 1.260–1.895; $p < 0.001$), ACLF 2 and 3, SpO_2/FiO_2 (HR 0.995; 95 % CI 0.991–0.999; p=0.022), lactate (HR 1.185; 95 % CI 1.015–1.384; p=0.032), ammonia (HR 1.009; 95 % CI 1.002–1.016; p=0.015), CRP (HR 1.010; 95 % CI 1.001–1.019; p=0.028), as well as the presence of pneumonia (HR 4.880; 95 % CI 1.080–22.050; p=0.040), UTIs (HR 2.950; 95 % CI 1.210–7.190; p=0.018).

Regarding the basic multivariate model, all variable parameters (CLIF-C OFs, SpO_2/FiO_2, pneumonia, ACLF stages) retained their statistical significance ($p < 0.05$). With that, the most significant impact on the prognosis was observed in ACLF 3 (HR 7.600; 95 % CI 2.450–23.580; $p < 0.001$) and in patients with pneumonia (HR 5.200; 95 % CI 1.150–23.500; p=0.032), which defines a more than five-fold increased risk of death. Increased CLIF-C OFs score and decreased SpO_2/FiO_2 were also associated with the increased risk of poor outcomes.

The advanced model additionally included laboratory parameters (lactate, ammonia, CRP). All the aforementioned factors retained their statistical significance in the latter model. The inclusion of additional laboratory markers helped to increase the prognostic value of the laboratory tests for lactate (HR 1.172; 95 % CI 1.005–1.367; p=0.042), ammonia (HR 1.008; 95 % CI 1.001–1.015; p=0.022), and CRP (HR 1.009; 95 % CI 1.000–1.018; p=0.050).

The introduction of laboratory markers (lactate, ammonia, CRP) into the advanced (final) model helped to enhance the accuracy of the death risk prognosis compared to a clinical-only assessment. This confirms the required complex approach to the stratification of patients with DLC with or without ACLF.

Thus, based on the multivariate analysis, the main independent predictors of poor outcomes in patients included the severity parameters (CLIF-C OFs), presence of pneumonia, and laboratory markers of systemic inflammation and metabolic disorders.

Table 1. *Association of clinical and laboratory factors with patient survival: results of univariate and multivariate Cox analyses*

Factors	ОР (95% ДИ) однофакторный анализ/ HR (95% CI) Univariate analysis	p value	ОР (95% ДИ) Комплексная модель/ HR (95% CI) Complex model	p value	ОР (95% ДИ) Базовая клиническая модель/ HR (95% CI) Basic clinical model	p value	ОР (95% ДИ) Расширенная лабораторная модель/ HR (95% CI) Extended laboratory model	p value
Age	1,019 [0,984; 1,056]	0,285	Не применимо	NA	Не применимо	NA	Не применимо	NA
Gender (male)	1,036 [0,469; 2,287]	0,930	Не применимо	NA	Не применимо	NA	Не применимо	NA
Readmissions	1,482 [0,508; 1,716]	0,088	1,415 [0,762; 1,762]	0,067	Не применимо	NA	Не применимо	NA
Ascites	0,986 [0,327; 2,972]	0,091	1,549 [0,277; 8,671]	0,062	Не применимо	NA	Не применимо	NA
SpO_2/FiO_2	0,994 [0,990; 0,998]	0,009	0,995 [0,991; 0,999]	0,022	0,992 [0,988; 0,996]	<0,001	0,993 [0,989; 0,997]	0,003
MAP	0,977 [0,941; 1,016]	0,244	Не применимо	NA	Не применимо	NA	Не применимо	NA
CLIF-C OFs	1,582 [1,291; 1,938]	<0,001	1,545 [1,260; 1,895]	<0,001	1,612 [1,320; 1,970]	<0,001	1,580 [1,295; 1,928]	<0,001
Lactate	1,209 [1,031; 1,418]	0,020	1,185 [1,015; 1,384]	0,032	Не применимо	NA	1,172 [1,005; 1,367]	0,042
ALT	1,007 [1,002; 1,011]	0,008	1,006 [1,001; 1,010]	0,018	Не применимо	NA	Не применимо	NA
AST	1,003 [1,001; 1,004]	0,006	1,002 [1,000; 1,004]	0,045	Не применимо	NA	Не применимо	NA
CRP	1,012 [1,003; 1,020]	0,006	1,010 [1,001; 1,019]	0,028	Не применимо	NA	1,009 [1,000; 1,018]	0,050
NLR	1,064 [0,996; 1,136]	0,064	1,055 [0,990; 1,125]	0,098	Не применимо	NA	Не применимо	NA
Ammonia	1,010 [1,003; 1,018]	0,006	1,009 [1,002; 1,016]	0,015	Не применимо	NA	1,008 [1,001; 1,015]	0,022
Total Bilirubin	1,002 [1,000; 1,003]	0,067	1,001 [0,999; 1,003]	0,210	Не применимо	NA	Не применимо	NA
alkaline phosphatase	1,001 [1,000; 1,003]	0,052	Не применимо	NA	Не применимо	NA	Не применимо	NA
UTI	3,004 [1,230; 7,339]	0,016	2,950 [1,210; 7,190]	0,018	Не применимо	NA	Не применимо	NA
Pneumonia	5,049 [1,103; 23,098]	0,037	4,880 [1,080; 22,050]	0,040	5,200 [1,150; 23,500]	0,032	5,100 [1,130; 23,020]	0,035
ACLF 2	4,567 [1,546; 13,493]	0,006	4,320 [1,480; 12,610]	0,008	4,850 [1,680; 14,000]	0,004	4,700 [1,620; 13,650]	0,005
ACLF 3	7,382 [2,325; 23,439]	0,001	7,150 [2,290; 22,340]	0,001	7,600 [2,450; 23,580]	<0,001	7,420 [2,380; 23,150]	<0,001

Note: HR — hazard ratio; CI — confidence interval. Data are presented as HR (95% CI). NA indicates that the variable was not included in the corresponding multivariate model. ACLF 2 and ACLF 3 variables were included as dummy variables using "decompensated cirrhosis without ACLF" as the reference category. The comprehensive model included all variables with p <0.1 from the univariate analysis. The basic clinical model was based on key clinical indicators. The extended laboratory model included variables from the basic clinical model with the addition of laboratory markers. SpO2 — pulse oximetric saturation, FiO2 — fraction of inspired oxygen; MAP — mean arterial pressure; CLIF-C OFs — CLIF Consortium Organ Failure score; ALT — alanine aminotransferase; AST — aspartate aminotransferase; CRP — C-reactive protein; NLR — neutrophil-to-lymphocyte ratio; UTI — urinary tract infection

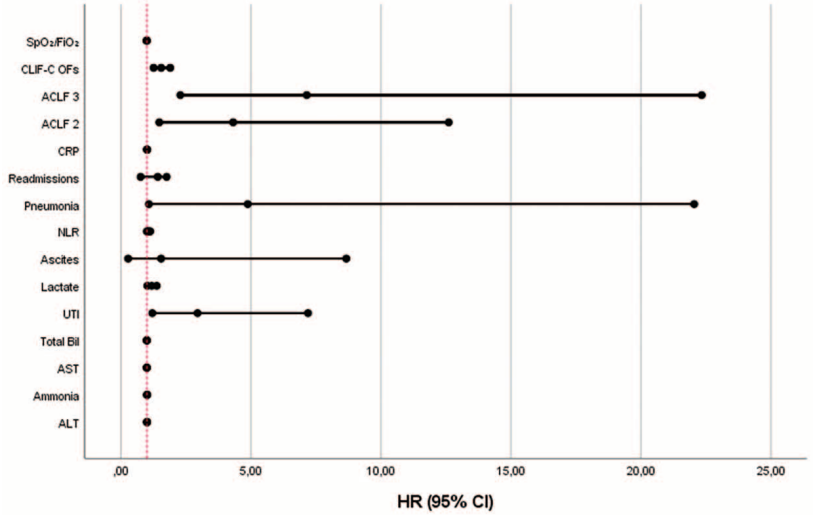

Figure 1. *Forest plot: independent predictors of 28-day mortality in patients with decompensated liver cirrhosis and ACLF according to multivariate Cox regression analysis. Hazard ratios (HR) and 95 % confidence intervals (CI) are displayed; significant factors are highlighted*

Note: SpO2 — pulse oximetric saturation, FiO2 — fraction of inspired oxygen; CLIF-C OFs — CLIF Consortium Organ Failure score; ACLF — Acute-on-Chronic Liver Failure; CRP — C-reactive protein; NLR — neutrophil-to-lymphocyte ratio; UTI — urinary tract infection; Total Bil. — total bilirubin; AST — aspartate aminotransferase; ALT — alanine aminotransferase.

The SpO_2/FiO_2 demonstrated protective effects. As the respiratory index increased, the mortality level decreased (and vice versa). The data obtained confirmed the required complex assessment of the patient's condition accounting for both clinical and laboratory parameters for the risk stratification and optimization of the management tactics.

See Figure 1 for the visual representation of the main independent risk factors for death.

Discussion of Results

Our analysis of factors affecting the 28-day mortality demonstrated that the lactate, CRP, ammonia levels, the CLIF-C OFs score, presence of pneumonia or UTIs demonstrated the highest values.

Thus, decreased SpO_2/FiO_2 index (p=0.001) and mean blood pressure (MBP) levels ($p < 0.001$), as well as elevated lactate levels (p=0.040) in deceased persons reflect the severity of circulatory and metabolic disorders typical for terminal ACLF stages.

Lactate is a product of anaerobic glycolysis, a marker of tissue hypoxia and metabolic alterations emerging in response to hormonal effects (e.g., epinephrine release). Lactate is excreted predominantly via three pathways — gluconeogenesis, tricarboxylic acid cycle in the liver, and renal excretion. The liver is responsible for about 70 % of the total lactate clearance in the body. The liver dysfunction leads to elevated plasma lactate levels, which is due to the impaired mitochondrial oxidation and decreased hepatocyte ability to utilize lactate. Besides, hyperlactatemia may develop due to the impaired microcirculation and tissue hypoxia leading to the enhanced anaerobic glycolysis. LC manifests with the impaired function of visceral microvessels, which is accompanied by hypoperfusion and, thus, hypoxia in the microcirculation of peripheral tissues. Thus, elevated blood lactate levels serves as a biochemical marker of tissue hypoperfusion and metabolic stress.

Multiple experimental and clinical studies in animals and humans have confirmed that the lactate level efficiently reflects the patient's condition severity and may be used to predict their outcomes [2, 3].

A prospective Chinese trial evaluating patients with the Hepatitis B Virus (HBV) and ACLF demonstrated that elevated serum lactate levels were an independent prognostic marker of a 28-day, 3-month, and 6-month mortality. The investigators proposed to include lactate into MELD and MELD-Na models (prognostic models used to assess the liver cirrhosis severity and short-term mortality), which could enhance their prognostic significance when assessing patient survival [4].

Besides its role in the pathogenesis of hepatic encephalopathy, hyperammoniemia is a potential marker in the assessment of severity and outcomes of chronic liver diseases.

In our study, ammonia levels were statistically significantly higher in the ACLF group; the maximum values obtained in the ACLF 3 group confirm the required further analysis of the association between hyperammoniemia and the organ dysfunction severity.

Besides, ammonia levels in the blood of deceased persons were somewhat higher than in the group of survived ones, although the difference was not statistically significant (184 vs. 142 μmol/L; p=0.068). With that, the inclusion of ammonia into predictive models has demonstrated its association with the increased risk of death, which was also shown by Wang X., et al. [5].

The Russian Liver Society experts consider hyperammoniemia as one of the main pathogenetic mechanisms in LC; however, measuring ammonia levels is recommended only for the differential diagnosis of encephalopathy [6].

Increased ammonia levels are a definite prognostic marker of organ dysfunction in ACLF in patients with alcoholic hepatitis and LC. The studies have demonstrated that ammonia levels may serve as an indicator of the condition severity and can predict clinical outcomes.

In the study of Tranah T. H., et al. [7], ammonia was proposed as an independent prognostic criterion for hospitalization due to the emergence of hepatic complications and mortality in LC patients, demonstrating a higher prognostic accuracy compared to the traditional scales analyzing the hepatic dysfunction severity.

Thus, the CANONIC study showed that the CLIF-SOFA organ dysfunction score, white blood cell count, a history of LC decompensation, and ascites on admission were independent factors associated with death.

In our study, repeated patient hospitalizations presumed a 48 % increase in the mortality risk; however, accounting for a wide CI [0.508–1.716] and p=0.088, this association cannot be considered clinically significant. Additionally, no significant associations were detected for the ascites on admittion and death ($p > 0.05$), while wide CIs (especially in the multivariate model) represent indefinite assessments. These trends are worth analyzing in studies with larger sample powers.

Infectious complications play an important role in the pathogenesis of ACLF, leading to poor prognosis. The multivariate analysis in our cohort demonstrated that pneumonia and UTIs were independent predictors of 28-day mortality in patients with DLC and ACLF. Pneumonia increased the risk of death more than 5-fold (HR 5.049–5.200; $p < 0.05$ in all models), while UTIs increased it almost 3 times (HR 2.950–3.004; $p < 0.02$), which confirms their critical role in the development of organ failure, sepsis, and multi-organ dysfunction in patients with LC.

Mechanisms of infectious complications in LC are associated with the significant immune imbalance caused by the hepatic failure itself and systemic metabolic disorders. Bacterial infections act as a trigger for the systemic inflammatory reaction, activating PAMPs (pathogen-associated molecular patterns) and proinflammatory cytokines, which in turn worsens the organ dysfunction and becomes the main event in the pathogenesis of ACLF.

The results obtained define the need for active prevention and quick diagnosis of infections in a high-risk patient group, especially with early ACLF signs. Adequate antibiotic therapy is also critically important to improve outcomes in this population. All together, our data confirm the latest publications, where infectious complications were considered one of the most significant poor prognostic factors in patients with DLC and ACLF [8, 9].

Conclusion

Our data confirm the decisive role of systemic inflammation, infectious complications, and metabolic disorders in the short-term prognosis for patients with DLC and ACLF. The key independent 28-day mortality predictors include the ACLF stages, severity based on the CLIF-C OFs score, elevated lactate, ammonia, and CRP levels, presence of pneumonia and urinary tract infections.

The implementation of the CLIF-C OFs score for routine death risk stratification in patients with DLC and ACLF will help to detect patients with the most probable poor outcomes, timely referring them to the specialized departments and liver transplant centers.

Вклад авторов:

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Тараки Б.М.: сбор и анализ данных, статистическая обработка материала, написание текста.

Адамова И.Г.: сбор и анализ данных, статистическая обработка материала.

Федоров И.Г.: получение данных, обзор литературных источников

Тотолян Г.Г.: оформление статьи и сопроводительных материалов

Петренко Н.В.: разработка концепции и дизайна исследования

Никитин И.Г.: утверждение окончательного текста статьи

Contribution of Authors

All the authors made a significant contribution to the preparation of the work, read and approved the final version of the article before publication

Taraki B.M.: data collection and analysis, statistical processing of data, text writing

Adamova I.G.: data collection and analysis, statistical processing of data

Fedorov I.G: data collection, literature review

Totolyan G.G.: design of the article and accompanying materials

Petrenko N.V.: concept and design of the study

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