https://doi.org/10.22416/1382-4376-2024-34-6-100-119
UDC 616.36-002-07/-08

Diagnosis and Treatment of Patients with Autoimmune Hepatitis (Experts' Agreement)

Yuliya G. Sandler[1]*, Elena V. Vinnitskaya[1], Karina L. Raikhelson[2], Konstantin V. Ivashkin[3], Sergey N. Batskikh[1], Elena N. Aleksandrova[1], Dzhamal T. Abdurakhmanov[3], Diana I. Abdulganieva[4], Igor G. Bakulin[5], Alexey O. Bueverov[3], Sergey L. Vorobyev[6], Olga A. Gerasimova[2,7], Anastasia I. Dolgushina[8], Mariia S. Zhuravleva[5], Liudmila Yu. Ilchenko[9], Vadim E. Karev[10], Natalia V. Korochanskaya[11], Irina L. Kliaritskaia[12], Nikolay S. Karnaukhov[1], Sergey V. Lapin[13], Maria A. Livzan[14], Marina V. Maevskaya[3], Natalia V. Marchenko[7], Tatiyana P. Nekrasova[3], Igor G. Nikitin[9], Alexander A. Novikov[1], Rafik G. Saifutdinov[15], Ekaterina V. Skazyvaeva[5], Vladimir E. Syutkin[16,17], Mariya K. Prashnova[7], Tatyana Yu. Khaymenova[1], Sergey G. Khomeriki[1]

[1] *Loginov Moscow Clinical Scientific Center, Moscow, Russian Federation*
[2] *Saint Petersburg State University, Saint Petersburg, Russian Federation*
[3] *I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russian Federation*
[4] *Kazan State Medical University, Kazan, Russian Federation*
[5] *North-Western State Medical University named after I.I. Mechnikov, Saint Petersburg, Russian Federation*
[6] *OOO National Center for Clinical Morphological Diagnostics, Saint Petersburg, Russian Federation*
[7] *Russian Scientific Center for Radiology and Surgical Technologies named after Academician A.M. Granov, Saint Petersburg, Russian Federation*
[8] *South Ural State Medical University, Chelyabinsk, Russian Federation*
[9] *N.I. Pirogov Russian National Research Medical University, Moscow, Russian Federation*
[10] *Pediatric Research and Clinical Center for Infectious Diseases under the Federal Medical Biological Agency, Saint Petersburg, Russian Federation*
[11] *Kuban State Medical University, Krasnodar, Russian Federation*
[12] *V.I. Vernadsky Crimean Federal University, Simferopol, Russian Federation*
[13] *Academician I.P. Pavlov First St. Petersburg State Medical University, Saint Petersburg, Russian Federation*
[14] *Omsk State Medical University, Omsk, Russian Federation*
[15] *Kazan State Medical Academy — Branch Campus of the Russian Medical Academy of Continuous Professional Education, Kazan, Russian Federation*
[16] *Medical and Biological University of Innovation and Continuous Education, State Research Center — Byrnasyan Federal Medical Biophysical Center of Federal Biological Agency, Moscow, Russian Federation*
[17] *N.V. Sklifosovsky Research Institute for Emergency Care of the Moscow City Health Department, Moscow, Russian Federation*

Background. In the last decade, the understanding of the pathogenesis of autoimmune hepatitis (AIH) has significantly deepened, based on the results of new clinical studies some diagnostic issues have been revised and immunosuppressive therapy regimens have been optimized.
Materials and methods. The latest Russian clinical guidelines for the diagnosis and treatment of AIH were presented in 2013; and in 2017, the first Russian agreement on the diagnosis and treatment of AIH was held. Updating approaches to the management of patients with AIH necessitated next systematization for use in clinical practice. In February 2024, the final session was held to discuss the provisions of the second agreement on the diagnosis and treatment of AIH.
Results. This publication presents the main discussion points of the agreement regarding methods and algorithms for detecting autoantibodies, the role of liver biopsy, revised morphological criteria for AIH, optimized immunosuppressive therapy regimens, updated criteria for assessing the response to therapy.
Conclusions. The agreement was the result of the work of a group of experts on the diagnosis and treatment of AIH and represents the basis for the creation of updated federal clinical guidelines.
Keywords: autoimmune hepatitis, agreement, autoantibodies, immunosuppressive therapy, federal clinical guidelines
Conflict of interest: the authors declare that there is no conflict of interest.

For citation: Sandler Yu.G., Vinnitskaya E.V., Raikhelson K.L., Ivashkin K.V., Batskikh S.N., Aleksandrova E.N., Abdurakhmanov D.T., Abdulganieva D.I., Bakulin I.G., Bueverov A.O., Vorobyev S.L., Gerasimova O.A., Dolgushina A.I., Zhuravleva M.S., Ilchenko L.Yu., Karev V.E., Korochanskaya N.V., Kliaritskaia I.L., Karnaukhov N.S., Lapin S.V., Livzan M.A., Maevskaya M.V., Marchenko N.V.,

Nekrasova T.P., Nikitin I.G., Novikov A.A., Saifutdinov R.G., Skazyvaeva E.V., Syutkin V.E., Prashnova M.K., Khaymenova T.Yu., Khomeriki S.G. Diagnosis and Treatment of Patients with Autoimmune Hepatitis (Experts' Agreement). Russian Journal of Gastroenterology, Hepatology, Coloproctology. 2024;34(6):100–119. https://doi. org/10.22416/1382-4376-2024-34-6-100-119

Диагностика и лечение пациентов с аутоиммунным гепатитом (соглашение специалистов)

Ю.Г. Сандлер[1]*, Е.В. Винницкая[1], К.Л. Райхельсон[2], К.В. Ивашкин[3], С.Н. Бацких[1], Е.Н. Александрова[1], Д.Т. Абдурахманов[3], Д.И. Абдулганиева[4], И.Г. Бакулин[5], А.О. Буеверов[3], С.Л. Воробьев[6], О.А. Герасимова[2,7], А.И. Долгушина[8], М.С. Журавлева[5], Л.Ю. Ильченко[9], В.Е. Карев[10], Н.В. Корочанская[11], И.Л. Кляритская[12], Н.С. Карнаухов[1], С.В. Лапин[13], М.А. Ливзан[14], М.В. Маевская[3], Н.В. Марченко[7], Т.П. Некрасова[3], И.Г. Никитин[9], А.А. Новиков[1], Р.Г. Сайфутдинов[15], Е.В. Сказываева[5], В.Е. Сюткин[16,17], М.К. Прашнова[7], Т.Ю. Хайменова[1], С.Г. Хомерики[1]

[1] ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы, Москва, Российская Федерация

[2] ФГБОУ ВО «Санкт-Петербургский государственный университет», Санкт-Петербург, Российская Федерация

[3] ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет), Москва, Российская Федерация

[4] ФГБОУ ВО «Казанский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Казань, Российская Федерация

[5] ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация

[6] ООО «Национальный центр клинической морфологической диагностики», Санкт-Петербург, Российская Федерация

[7] ФГБУ «Российский научный центр радиологии и хирургических технологий им. академика А.М. Гранова» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация

[8] ФГБОУ ВО «Южно-Уральский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Челябинск, Российская Федерация

[9] ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации, Москва, Российская Федерации

[10] ФГБУ «Детский научно-клинический центр инфекционных болезней Федерального медико-биологического агентства», Санкт-Петербург, Российская Федерация

[11] ФГБОУ ВО «Кубанский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Краснодар, Российская Федерация

[12] ФГАОУ ВО «Крымский федеральный университет им. В.И. Вернадского», Симферополь, Российская Федерация

[13] ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. академика И.П. Павлова» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация

[14] ФГБОУ ВО «Омский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Омск, Российская Федерация

[15] Казанская государственная медицинская академия — филиал ФГБОУ ДПО «Российская медицинская академия непрерывного профессионального образования» Министерства здравоохранения Российской Федерации, Казань, Российская Федерация

[16] Медико-биологический университет инноваций и непрерывного образования, ФГБУ «Государственный научный центр Российской Федерации — Федеральный медицинский биофизический центр им. А.И. Бурназяна», Москва, Российская Федерация

[17] ГБУЗ г. Москвы «НИИ скорой помощи им. Н.В. Склифосовского» Департамента здравоохранения г. Москвы, Москва, Российская Федерация

Введение. В последнее десятилетие существенно углубилось понимание патогенеза аутоиммунного гепатита (АИГ), пересмотрены некоторые вопросы диагностики, на основании результатов новых клинических исследований оптимизированы схемы иммуносупрессивной терапии.

Материалы и методы. Последние Российские клинические рекомендации по диагностике и лечению АИГ были представлены в 2013 г. В 2017 г. было создано первое Российское соглашение по вопросам диагностики и лечения АИГ. Обновление подходов к ведению пациентов с АИГ обусловило необходимость очередной систематизации для применения в клинической практике. В феврале 2024 г. состоялась заключительная сессия по обсуждению положений второго соглашения по диагностике и лечению АИГ.

Результаты. В настоящей публикации представлены основные положения соглашения, касающиеся методов и алгоритмов выявления аутоантител, роли биопсии печени, пересмотренных морфологических критериев АИГ, оптимизированных схем иммуносупрессивной терапии, а также обновленных критериев оценки ответа на терапию.

Выводы. Соглашение стало итогом работы группы российских экспертов по диагностике и лечению АИГ и представляет собой основу для создания обновленных федеральных клинических рекомендаций.

Ключевые слова: аутоиммунный гепатит, соглашение, аутоантитела, иммуносупрессивная терапия, федеральные клинические рекомендации
Конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

Для цитирования: Сандлер Ю.Г., Винницкая Е.В., Райхельсон К.Л., Ивашкин К.В., Бацких С.Н., Александрова Е.Н., Абдурахманов Д.Т., Абдулганиева Д.И., Бакулин И.Г., Буеверов А.О., Воробьев С.Л., Герасимова О.А., Долгушина А.И., Журавлева М.С., Ильченко Л.Ю., Карев В.Е., Корочанская Н.В., Кляритская И.Л., Карнаухов Н.С., Лапин С.В., Ливзан М.А., Маевская М.В., Марченко Н.В., Некрасова Т.П., Никитин И.Г., Новиков А.А., Сайфутдинов Р.Г., Сказываева Е.В., Сюткин В.Е., Прашнова М.К., Хайменова Т.Ю., Хомерики С.Г. Диагностика и лечение пациентов с аутоиммунным гепатитом (соглашение специалистов). Российский журнал гастроэнтерологии, гепатологии, колопроктологии. 2024;34(6):100–119. https://doi.org/10.22416/1382-4376-2024-34-6-100-119

Introduction

Autoimmune hepatitis (AIH) is a chronic immune-mediated liver disease of unknown cause, most commonly affecting individuals with a genetic predisposition. It has a wide range of clinical presentations and is marked by elevated levels of alanine aminotransferase and aspartate aminotransferase. Key features include increased immunoglobulin G (IgG) levels (and/or gamma-globulins), the presence of specific autoantibodies, and histological evidence of active portal and/or lobular hepatitis. AIH typically responds well to immunosuppressive therapy but can progress if left untreated.

The clinical presentation of AIH is highly variable, ranging from asymptomatic cases (characterized by mild elevations in liver enzymes and nonspecific symptoms lasting for years) to acute forms leading to liver failure. Approximately one-third of patients are diagnosed at the stage of cirrhosis. Early diagnosis and consistent, effective immunosuppressive therapy are essential for a favorable prognosis, which is achievable with proper treatment.

Autoimmune hepatitis is relatively rare in the general population and is often overlooked due to its diverse clinical manifestations and, at times, complex diagnostic process. It should be considered in any case of unexplained acute or chronic liver disease.

In the Russian Federation, reliable data on the prevalence of AIH are currently unavailable. The most recent Russian clinical guidelines for the diagnosis and treatment of AIH were published in 2013 [1].

However, recent advancements include updated diagnostic criteria for AIH, the identification of patient subgroups with distinct disease phenotypes, and new findings from randomized clinical trials. These developments offer the potential to improve diagnostic accuracy, enhance treatment outcomes, and significantly improve the quality of life for patients with AIH.

Although AIH is considered a rare condition, with a prevalence of fewer than 50 cases per 100,000 people, its incidence and prevalence have risen significantly in the past decade — by 3.1-fold and 2.8-fold, respectively, compared to rates before the 2000s [2, 3].

Epidemiological studies also highlight an increase in AIH cases across all age groups, with a particularly notable rise among individuals over 65 years old and in certain ethnic populations [4, 5].

Materials and methods

The development of the agreement's provisions involved 32 Russian experts who were invited to participate based on their clinical experience, peer-reviewed publications on the subject, and contributions to previous Russian guidelines and recommendations.

The consensus document was developed using the Delphi method. The working group identified key issues in the diagnosis and treatment of autoimmune hepatitis that required discussion to formulate the provisions. These provisions were grounded in the best available evidence and recommendations, following the GRADE system (Grading of Recommendations Assessment, Development, and Evaluation) [6], which assesses the grade of recommendations and level of evidences. In the absence of sufficient evidence, experts' opinions were used.

Only provisions that achieved unanimous approval after three rounds of online voting were included in the final agreement. During the first round of online discussions, participants expressed their agreement or disagreement, and the provisions were revised accordingly. Each revised provision was then subject to further voting.

The voting system consisted of six levels: 1) complete agreement; 2) agreement with minor objections; 3) agreement with major concerns; 4) disagreement with major or minor concerns; 5) complete disagreement.

A provision was deemed approved if at least 80 % of participants expressed agreement (combining votes for complete agreement and agreement with minor objections).

Questions, provisions, and recommendations

Within the classification of autoinflammatory and autoimmune diseases in the continuum of immunological disorders [7], autoimmune hepatitis displays all the key features of a classic autoimmune disease. It can be defined as:

An immune-mediated inflammatory liver disease of unknown origin, predominantly affecting genetically predisposed individuals, with a broad spectrum of clinical presentations. It is characterized by elevated aspartate and alanine aminotransferase levels, typically increased immunoglobulin G levels (and/or gamma-globulins), the presence of autoantibodies, and histological evidence of active portal or lobular hepatitis. AIH generally responds well to immunosuppressive therapy but progresses in the absence of treatment.

In voting, 77.8 % of participants expressed "complete agreement" (A+), while 22.2 % agreed with "minor objections" (A).

The absence of definitive pathognomonic markers for AIH, along with its clinical variability across age, sex, disease onset, progression, outcomes, serological profiles, histological patterns, response to immunosuppressive therapy, and the frequency of associated immune-mediated diseases, creates significant challenges in diagnosis. A notable proportion of patients (approximately one-third) are diagnosed at the stage of cirrhosis, complicating timely treatment and prognosis.

The heterogeneous clinical, laboratory, and morphological features of AIH often make diagnosis difficult, even for experts. To address this, the International Autoimmune Hepatitis Group (IAIHG) established diagnostic criteria in the late 20th and early 21st centuries. These include the revised and expanded criteria (IAIHG, 1999) and the simplified criteria (IAIHG, 2008), both of which remain widely used in clinical practice [8, 9].

AIH's diagnostic features lack specificity, emphasizing the need to exclude other liver diseases as part of the diagnostic process. However, AIH is not solely a diagnosis of exclusion [10, 11] and can coexist with other liver conditions, further complicating diagnosis.

Codified diagnostic scoring systems from 1999 are now recommended for use in clinical research or particularly complex clinical cases. In routine practice, effective diagnosis requires not only an understanding of clinical and morphological criteria but also clinical expertise and judgment.

A clear definition of AIH diagnosis was proposed and formulated as follows:

The diagnostic criteria for AIH should include elevated transaminase levels, increased immunoglobulin G or gamma-globulins, the presence of serum autoantibodies, and histological evidence of active portal or lobular hepatitis, with other causes of hepatitis (e.g., viral hepatitis) excluded.

This formulation was approved by 78.6 % of participants with "complete agreement" (A+), 17.8 % with "agreement with minor objections" (A), and 3.6 % with "agreement with major concerns" (A–).

Grade of recommendations — A;
level of evidence — 2.

Several aspects of AIH diagnosis and treatment generated significant discussion, including: 1) the optimal use of autoantibody diagnostic methods and their sequencing in AIH patients; 2) the role of liver biopsy in AIH diagnosis, including the relevance of revised morphological criteria and current systems for assessing histological activity and liver fibrosis; 3) optimal criteria for evaluating treatment response; 4) medications for first-, second-, and third-line regimens of immunosuppressive therapy, as well as rescue therapies; 5) the appropriate duration of immunosuppressive therapy in AIH patients with cirrhosis.

Optimal use of autoantibody testing methods and sequence of application

The presence of autoantibodies is a key criterion in the diagnosis of AIH, but it also presents significant challenges in interpreting results. The accurate and reliable diagnosis of AIH relies on the correct application of detection methods and testing algorithms. After extensive expert discussions and revisions, the following recommendations were approved as the most effective approach for identifying autoantibodies in patients suspected of having AIH.

In the initial stage of AIH diagnosis, it is recommended to perform serological testing for antinuclear antibodies (ANA) using indirect immunofluorescence on HEp-2 cells (human laryngeal carcinoma epithelial cells) or a "triple substrate" (a tissue complex

of cryosections from rat liver, kidney, and stomach). Testing should also include smooth muscle antibodies (SMA) and liver/kidney microsomal antibodies type 1 (anti-LKM-1) using indirect immunofluorescence on the "triple substrate".

This recommendation achieved "complete agreement" (A+) from 78.6 % of participants and "agreement with minor objections" (A) from 21.4 %.

Grade of recommendations — B;
level of evidence — 2.

In cases where initial testing is negative, but AIH is still suspected, it is recommended to proceed with serological testing for antibodies to soluble liver antigen (anti-SLA) and liver cytosol antigen type 1 (anti-LC-1) using solid-phase immunoassays (e.g., immunoblot or ELISA). Additionally, testing for atypical perinuclear anti-neutrophil cytoplasmic antibodies (pANCA) using indirect immunofluorescence is recommended if available. Other antigens, such as ASPGR and Ro-52, are considered minor markers due to their low diagnostic sensitivity and specificity, which complicates their interpretation.

This recommendation achieved "complete agreement" (A+) from 81.5 % of participants and "agreement with minor objections" (A) from 18.5 %.

Grade of recommendations — B;
level of evidence — 2.

Antinuclear antibodies (ANA) are the most frequently detected autoantibodies in AIH, present in ~80 % of cases, followed by SMA in 60–65 % of cases. Anti-actin antibodies, a subset of SMA, are found in 86–100 % of SMA-positive patients. ANA and SMA often co-occur, with both detected in ~43 % of cases. In adults with AIH, anti-LKM-1 antibodies may be detected, often in the absence of ANA and SMA. Anti-LKM-1 is found in ~3 % of adults and 13–38 % of children, while it is rarely seen in hepatitis C (0–13 %) [12, 13]. Anti-LKM-3 antibodies are extremely rare.

Antibodies to soluble liver antigen (anti-SLA) are the most specific diagnostic marker for AIH, with a specificity of 99 %. They are detected in 7–22 % of patients and are the sole marker in 14–20 % of cases. Anti-SLA is associated with disease severity and an increased risk of relapse after therapy withdrawal [14, 15].

Antibodies to liver cytosol antigen type 1 (anti-LC-1) are present in 24–32 % of pediatric patients and frequently co-occur with anti-LKM-1 (up to 75 %). These antibodies are less common in adults but are strongly associated with severe AIH when present [12, 16, 17].

Atypical perinuclear antineutrophil cytoplasmic antibodies (pANCA) are detected in 20–96 % of AIH cases, 26–94 % of primary sclerosing cholangitis cases, and 33–83 % of patients with inflammatory bowel disease [12, 18, 19]. Atypical pANCA serves as an auxiliary diagnostic marker for AIH when other autoantibodies (ANA, SMA, anti-LKM-1, LC-1, and SLA) are absent [20, 21].

Antibodies against the asialoglycoprotein receptor (ASGPR) are sometimes detected in patients with negative results for other autoantibodies [20, 22], but their low specificity makes routine testing unnecessary [23].

None of the autoantibodies, except anti-SLA, are highly specific for AIH, and many can also be found in other liver conditions. ANA, for instance, is common in rheumatologic autoimmune diseases, while low-titer ANA and SMA can be detected in non-alcoholic fatty liver disease (NAFLD). Therefore, the presence of autoantibodies cannot definitively confirm AIH and should not be the sole diagnostic criterion [12].

If no autoantibodies are detected, the condition is classified as *seronegative AIH*. This occurs in ~7 % of acute severe cases and in 1–34 % of chronic cases [23–25].

Autoantibody detection in AIH is based on various methods, including indirect immunofluorescence, enzyme-linked immunosorbent assays (ELISA), immunoblot, chemiluminescent immunoassays, and multiplex immunoassays. The recommended testing algorithm consists of two stages, with indirect immunofluorescence serving as the primary method for autoantibody detection [12, 20].

In the first stage, screening tests should be conducted to detect ANA, SMA, AMA (to exclude primary biliary cholangitis), and anti-LKM-1 using indirect immunofluorescence with a "triple substrate". Diagnostic titers for adults are defined as ≥ 1:40 [12]. For ANA testing, indirect immunofluorescence with HEp-2 cells is preferred, with diagnostic titers defined as ≥ 1:160. ANA fluorescence patterns on HEp-2 cells should be described according to the International Consensus on ANA Patterns (ICAP) [26, 27].

The indirect immunofluorescence method on HEp-2 cells is considered the gold standard for ANA detection, offering superior sensitivity compared to tissue substrates. It also allows differentiation of nuclear and cytoplasmic fluorescence patterns, including those characteristic of specific antibodies like sp100, gp210, centromere antibodies, and AMA-M2.

Screening for ANA using solid-phase immunoassays (ELISA or immunoblot) is not recommended

due to their limited ability to detect a broad range of autoantigens, which reduces diagnostic sensitivity and increases the likelihood of false-negative results [26, 28].

In the second stage, confirmatory and additional tests should be performed to identify specific liver-related antibodies, including:

1. Anti-LKM-1, anti-LKM-3, anti-LC-1, anti-F-actin, as well as AMA-M2, sp100, gp210, and centromere antibodies (to exclude primary biliary cholangitis) using ELISA, immunoblot, chemiluminescent immunoassays, or multiplex immunoassays. Since no nuclear antigen specific to AIH has been identified, confirmatory testing for antigen-specific ANA (e.g., antibodies to DNA, nucleosomes, histones, Ro-52, ribonucleoproteins) is not recommended, except in cases involving AMA-M2, sp100, gp210, or centromere antibodies.
2. Atypical pANCA using indirect immunofluorescence on formalin-fixed human neutrophils. ELISA or immunoblot may also be used for confirmation if available.
3. ASGPR antibodies using ELISA, if available.
4. A group of anti-actin antibodies that are a heterogeneous group of autoantibodies that target primarily F-actin. Antibodies against F-actin stain microfilaments on HEp-2 cells. Autoantibodies against filamentous (F) actin (anti-F-actin) represent a type of SMA and are present in 86–100 % of SMA-positive patients with AIH. Autoantibodies to alpha-actinin (anti-α-actinin) is an investigational marker and is present in 42 % of patients with AIH and 66 % of patients with anti-actinin [20, 29, 30].

The identification and proper interpretation of autoantibody tests are essential for the accurate diagnosis of AIH. However, comprehensive testing for all autoantibodies may not be available in all laboratories. Enhancing the training and expertise of laboratory personnel and clinicians is crucial for improving the accuracy of autoantibody detection and diagnosis.

The role of liver biopsy in AIH diagnosis, relevance of revised morphological criteria, and current systems for assessing histological activity and liver fibrosis in AIH

In clinical practice, despite clear and widely agreed-upon international expert recommendations regarding the necessity of morphological verification for AIH diagnosis, liver biopsy is not always performed. The following position was presented for discussion among Russian experts:

A definitive diagnosis of confirmed AIH can only be established with morphological verification. Morphological verification is an essential diagnostic criterion for AIH and must be performed prior to initiating immunosuppressive therapy unless absolute contraindications exist.

This position received "complete agreement" (A+) of 67.9 % of participants, "agreement with minor objections" (A) of 25 %, and "agreement with major concerns" (A–) by 7.1 %.

Grade of recommendations — A;
level of evidence — 1.

Although formal agreement was achieved, there were diverse comments and additional conditions reflecting real-world clinical practice.

The most intense discussion revolved around whether AIH could be diagnosed without morphological findings. Specifically, the following formulation was proposed: *"Liver biopsy is performed except in cases of acute severe AIH with a high risk of acute liver failure, where the diagnosis of AIH aligns with scoring criteria from the International Autoimmune Hepatitis Group"*. This position appears reasonable and may justify forgoing liver biopsy due to absolute contraindications. In cases of acute severe hepatitis and/or liver failure with a high suspicion of AIH, it is recommended to initiate systemic glucocorticoid (GC) therapy without performing biopsy. After stabilization of the patient's condition, morphological verification should be carried out as early as possible.

There are no pathognomonic morphological criteria for AIH, and the disease can coexist with other immune-mediated or non-immune liver diseases. Biochemical activity does not always correlate with histological findings, particularly in patients with cirrhosis. Additionally, managing AIH often involves prolonged, sometimes lifelong, therapy with potential side effects, all of which highlight the need for morphological verification of AIH [31–33]. Current international and national AIH guidelines consistently emphasize the necessity of liver biopsy for morphological verification of confirmed AIH [2, 21, 23, 33].

The need for repeat biopsy carries less stringent recommendations and is typically determined by the clinical context and a personalized approach to the patient. Until better non-invasive biomarkers become available to assess histological disease activity, morphological verification remains the most reliable method for assessing disease activity, particularly in cases of cirrhosis [31, 34].

Repeat liver biopsy is advisable when determining whether to discontinue or adjust immunosuppressive therapy or in cases where the nature of the liver pathology is

uncertain. The need for liver biopsy is determined on a case-by-case basis by the treating physician or a multidisciplinary team.

This statement received "complete agreement" (A+) from 92.6 % of participants and "agreement with minor objections" (A) from 7.4 %.

Grade of recommendations — B;
level of evidence — 2.

Application of morphological systems for assessing histological activity and liver fibrosis in AIH

Non-invasive methods for diagnosing and staging fibrosis often rely on the METAVIR scoring system, which is also recommended for morphological staging of fibrosis in AIH. When determining whether to initiate or discontinue immunosuppressive therapy, it is recommended to evaluate necroinflammatory activity using the histological activity indices proposed by R.G. Knodell or K.G. Ishak [2, 21, 23]. These systems are therefore preferable for describing histological activity [35–38].

For assessing histological activity and liver fibrosis severity in AIH, it is appropriate to use current international systems (METAVIR, Knodell, Ishak).

This formulation received "complete agreement" (A+) from 77.8 % of participants, "agreement with minor objections" (A) from 14.8 %, "agreement with significant concerns" (A–) from 3.7 %, and "disagreement with major or minor concerns" (D–) from 3.7 %.

Grade of recommendation — B;
level of evidence — 2.

The absence of strictly pathognomonic histological criteria is a key feature of morphological verification in AIH. Previously, certain features (emperipolesis, rosette formation, or prominent plasma cell presence) were thought to be more specific for AIH. According to the simplified diagnostic criteria of the IAIHG (2008), these features, along with interface hepatitis, were considered typical. However, sufficient evidence now indicates that these features reflect the severity of liver damage or tissue regeneration rather than etiology [8, 9, 33]. Additionally, earlier morphological criteria for "definitive" AIH primarily focused on chronic AIH, where inflammatory changes are mainly confined to the portal/interface areas.

In 2022, an international group of pathologists reviewed and adopted updated histological criteria for AIH, categorizing findings into probable, possible, and unlikely morphological features, which include both portal and lobular involvement. These criteria better capture the morphological spectrum of both acute and chronic AIH [33]. Studies have shown that replacing certain components of the 2008 histological scoring system with the modified IAHPG 2022 criteria improved diagnostic sensitivity [39].

There are no strictly pathognomonic morphological criteria for AIH. The most likely histological features of AIH are as follows: 1) moderate or high activity portal hepatitis with lymphocytic and plasma cell infiltration, predominantly periportal, with evidence of interface hepatitis, unrelated to portal or interface bile ducts; 2) moderate or high activity lobular hepatitis with lymphocytic and plasma cell infiltration, unrelated to lobular bile ducts; or 3) predominantly lobular hepatitis with centrilobular perivenous necrosis (or without), along with one of the following: portal lymphoplasmacytic hepatitis, interface hepatitis, or portal fibrosis.

This formulation received "complete agreement" (A+) from 92.6 % of participants, "agreement with minor objections" (A) from 3.7 %, and "agreement with significant concerns" (A–) from 3.7 %.

Grade of recommendation — B;
level of evidence — 2.

The view that AIH is no longer a "diagnosis of exclusion" and can coexist with other liver diseases was unanimously supported by all experts.

The presence of steatosis, bile duct injury, hepatocellular and intracanalicular cholestasis with minimal activity, fibrosing obliterative cholangitis, or nodular regenerative hyperplasia in the liver biopsy does not exclude an AIH diagnosis. In such cases, morphological and clinical-laboratory re-evaluation of the patient is recommended to identify overlapping liver pathologies.

This statement received "complete agreement" (A+) from 96.3 % of participants and "agreement with minor objections" (A) from 3.7 %.

Grade of recommendation — B;
level of evidence — 2.

Optimal criteria for therapy response

Numerous publications on the results of clinical studies involving AIH patients have highlighted and evaluated various intermediate and final "endpoints" of treatment response. These criteria are used to guide decisions regarding therapy adjustments, duration, and discontinuation

of immunosuppressive therapy, all of which can influence disease outcomes and prognosis [40–43].

Among international experts, there is no consensus on the exact criteria for evaluating therapy response in AIH. A similar lack of agreement is seen among Russian experts. There was significant debate regarding the final endpoints for evaluating the response to first- and second-line immunosuppressive therapy in AIH patients, particularly concerning which criteria should be considered optimal and significant in influencing disease outcomes and prognosis based on the available evidence.

In a 2022 publication in *Hepatology*, based on a consensus by the International Autoimmune Hepatitis Group (IAIHG) and data from a systematic review, recommended criteria for evaluating immunosuppressive therapy response were presented. These criteria are suggested for use in both routine clinical practice and clinical research [44].

It is important to note that the optimal therapy response criteria are those that significantly impact disease outcomes.

The optimal outcomes of therapy response in AIH patients should include: complete biochemical response, insufficient response, non-response, and remission. Complete biochemical response is defined as normalization of serum transaminases and IgG levels below the upper limit of normal, achieved no later than six months after starting therapy. Insufficient response refers to the absence of normalization of serum transaminases and IgG below upper limit of normal within six months of therapy initiation. Non-response is defined as less than a 50 % reduction in serum transaminase levels within four weeks of starting therapy. Remission is considered when the histological activity index (HAI) from liver biopsy is less than 4 (HAI < 4).

This formulation received "complete agreement" (A+) from 73.1 % of participants, "agreement with minor objections" (A) from 15.4 %, "agreement with significant concerns" (A–) from 7.7 %, and "disagreement with major or minor concerns" (D–) from 3.8 %.

Grade of recommendation — B;
level of evidence — 2.

This range of expert opinions reflects a general trend and can be explained by the fact that the criteria and timelines for evaluating immunosuppressive therapy response are subject to various interpretations. These include complete and incomplete (insufficient) biochemical response, complete biochemical remission, complete and incomplete histological remission, complete response (encompassing biochemical and histological response), and immunological remission (criteria: sustained normalization of gamma-globulins and/or IgG). However, not all these indicators significantly affect AIH outcomes or can be considered final endpoints.

Complete biochemical response is an independent prognostic factor. Patients in pre-cirrhotic stages who achieve and sustain complete biochemical response experience significantly reduced progression to cirrhosis, which is a critical marker of long-term immunosuppressive therapy success.

However, there are objective counterarguments to some proposed response criteria. First, a substantial cohort of patients (approximately 10–15 % on average, and 25–39 % in cases of acute severe AIH) have baseline IgG levels within the reference range before initiating immunosuppressive therapy. Another group includes cirrhotic patients who, despite long-term immunosuppressive therapy, continue to exhibit elevated gamma-globulin levels or "normal" IgG levels while lacking necroinflammatory activity on histological examination [23, 31, 45–48].

Second, recent results from a multicenter retrospective cohort study of AIH patients (n = 1135) indicate that persistently elevated aminotransferase levels are more strongly associated with poorer long-term outcomes than elevated IgG levels [49].

This topic remains highly debated and likely requires a dedicated article. To validate accepted therapy response endpoints, further prospective comparative studies involving diverse patient databases are needed. To ensure a unified global understanding among researchers, it is crucial to adopt standardized criteria. Over time, as the evidence base expands, these "final therapy response outcomes" may undergo further refinement.

A *Hepatology Report* publication in 2024 presented results from a multicenter cohort study that confirmed the status of complete biochemical response and its correlation with survival criteria. A complete biochemical response was achieved in 128/200 (64.0 %) individuals. Patients who did not achieve complete biochemical response were more likely to have cirrhosis (22.2 % vs. 10.9 %; p = 0.036). Those who achieved sustained biochemical response demonstrated better AIH-related survival (hazard ratio [HR] — 0.118; 95 % CI: 0.052–0.267; $p < 0.0001$) and overall survival (HR — 0.253; 95 % CI: 0.111–0.572; p = 0.0003) [50].

In our opinion, it is important not to overcomplicate or introduce excessive terminological distinctions but to propose a straightforward, evidence-based approach that simplifies the

evaluation of final therapy response outcomes for clinicians.

Questions on optimizing immunosuppressive therapy regimens

In the absence of treatment, AIH leads to the progression of liver fibrosis to cirrhosis and, eventually, to end-stage liver disease. The cumulative 10-year survival of untreated AIH patients is lower than that of those receiving immunosuppressive therapy (67 % vs. 98 %; $p = 0.001$) [51]. Adequate immunosuppressive therapy achieves sustained biochemical response, complete disease remission, and regression of liver fibrosis, even at the stage of compensated advanced chronic liver disease.

Early diagnosis and timely, consistent therapy are critical for favorable AIH outcomes and prognosis. Immunosuppressive therapy initially involves remission induction followed by maintenance therapy, which is often long-term. Glucocorticoid therapy is effective for all forms of AIH during the induction phase, while azathioprine or a combination of low-dose glucocorticoids and azathioprine is preferred for maintaining remission [2, 21, 23, 52].

First-line therapy for treating AIH patients (without decompensated cirrhosis or acute severe hepatitis) consists of a combination of glucocorticoids (prednisone at 0.5–1 mg/kg/day) and azathioprine (1–2 mg/kg/day). Azathioprine should optimally be initiated approximately two weeks after glucocorticoid induction, following the resolution of initial hyperbilirubinemia, if present.

This statement received "complete agreement" (A+) from 92.6 % of participants and "agreement with minor objections" (A) from 7.4 %.

Grade of recommendation — B;
level of evidence — 1.

For more than 40 years, first-line treatment for AIH has included glucocorticoids (prednisone) as monotherapy or in combination with azathioprine. Glucocorticoids suppress cytokine production and inhibit T cell activation, while azathioprine blocks the maturation of T cell precursors, with its effects typically manifesting within two months [53, 54].

The timing of azathioprine initiation during induction therapy, whether early or delayed, does not significantly affect remission rates or adverse drug effects. Data suggests that either strategy can be employed [55]. However, delayed azathioprine initiation (typically after two weeks) can be advantageous, as it confirms the AIH diagnosis through a positive response to glucocorticoids and avoids diagnostic dilemmas related to distinguishing azathioprine-induced hepatotoxicity from primary non-response to therapy [23].

Remission is achieved in 80 % of patients within 2–3 years of immunosuppressive therapy, with 10- and 20-year survival rates exceeding 80 % [53, 54, 56, 57]. Relapses are common: 59 % of patients relapse one year after therapy discontinuation, increasing to 73 and 81 % at two and three years, respectively [32]. After multiple relapses, long-term combination therapy is preferred. Azathioprine monotherapy is optimal for maintaining remission, provided no contraindications exist.

Sustained remission can be achieved in 47 % of patients within 10 years, even after relapse, and long-term maintenance regimens do not necessarily have to be indefinite [53].

Second- and third-line therapy is used when standard first-line drugs are contraindicated, unavailable, or intolerable, or when first-line therapy yields insufficient or no response. Treatment is tailored individually based on the patient's response to therapy.

This received "complete agreement" (A+) from 100 % of participants.

Grade of recommendation — B;
level of evidence — 2.

The average rate of azathioprine discontinuation during AIH treatment is ~15 % within the first year and up to 25 % within two years, with complications requiring discontinuation occurring in ~10 % of patients [54, 55, 57]. Glucocorticoids have an adverse event rate of ~80 % within the first two years, with ~15 % requiring discontinuation [21, 23, 52, 56].

Second-line therapy is used for treatment failure, incomplete response, and drug intolerance [21, 23]. Treatment failure occurs in 7–9 % of adults and is associated with an increased risk of cirrhosis and liver failure, with mortality rates reaching 30 %. Incomplete response is seen in ~15 % of adults and children. Patients unable to normalize transaminases within 36 months have higher rates of cirrhosis and a greater need for liver transplantation [58].

Second-line alternative therapies include: budesonide (9 mg/day, as an alternative to systemic glucocorticoids for glucocorticoid intolerance) combined with azathioprine (1–2 mg/kg/day); Glucocorticoid monotherapy (prednisone 0.5–1 mg/kg/day); mycophenolate mofetil or 6-mercaptopurine combined with glucocorticoids (as alternatives

to azathioprine). Mycophenolate mofetil, at 1–2 g/day in combination with glucocorticoids (prednisone 0.5–1 mg/kg/day), is considered preferable.

This statement received "complete agreement" (A+) from 75 % of participants and "agreement with minor objections" (A) from 25 %.

Grade of recommendation — B;
level of evidence — 2.

Expert discussions focused on the role of mycophenolate mofetil and budesonide in immunosuppressive therapy regimens, specifically whether they should be used as first-line or alternative second-line therapies. Current evidence supports their use primarily in second-line therapy. Clinical trials and meta-analyses suggest mycophenolate mofetil may be more effective than azathioprine for inducing biochemical remission [59–62]. These findings could lead to a reassessment of international guidelines for untreated AIH patients.

Budesonide, a topical glucocorticoid with high glucocorticoid receptor affinity, undergoes 90 % hepatic clearance during first-pass metabolism, producing inactive metabolites (16α-hydroxyprednisolone and 6β-hydroxybudesonide) [63, 64]. A 2010 clinical trial found budesonide more effective than prednisone for inducing AIH remission, with fewer cosmetic and systemic side effects. However, this study was criticized for rapid prednisone dose reduction in the control group, potentially skewing outcomes [43].

Budesonide studies, including dosing below 9 mg, administration frequency, tapering protocols, and long-term efficacy and safety, remain limited. Further large-scale randomized controlled trials are needed [65–68]. Despite criticisms, some international associations recommend budesonide as first-line therapy for non-cirrhotic patients [21], particularly for those at risk of systemic glucocorticoid side effects. Others take a more cautious approach, suggesting budesonide as an alternative pending further research.

Third-line therapy is indicated for non-response to first- and second-line treatments and involves calcineurin inhibitors (tacrolimus, cyclosporine), with individual dose adjustments based on blood levels and gradual glucocorticoid tapering or discontinuation. Tacrolimus is preferred. The initial dose is 1 mg twice daily (in combination with the patient's previous glucocorticoid dose), with an estimated dose of 0.1 mg/kg twice daily, targeting trough levels of 6–8 ng/mL. Third-line therapy should be conducted in specialized centers experienced with this patient category.

This received "complete agreement" (A+) from 75 % of participants and "agreement with minor objections" (A) from 25 %.

Grade of recommendation — B;
level of evidence — 3.

Studies of tacrolimus in cases of treatment failure, incomplete response, or azathioprine intolerance in AIH have demonstrated moderate to high efficacy. Tacrolimus was combined with prednisone, azathioprine, or mycophenolate mofetil, with trough levels ranging from 1 to 10 ng/mL. Adverse effects requiring dose reduction or discontinuation (e.g., tremor, headaches, renal complications, alopecia) occurred in ~25 % of cases [69–71].

In two single-center studies, normalization of transaminases was achieved in 91 and 92 % of cases, while alanine aminotransferase or IgG normalization was observed in 79 % [72]. A systematic review (11 studies, 58 patients) found that cyclosporine achieved remission in 59 % of cases within six months, partial response in 22 %, and treatment failure in 12 % [73].

Cyclosporine may be preferred over tacrolimus in AIH patients with comorbid diabetes, as diabetes can occur as a tacrolimus side effect [74]. In second- or third-line settings, cyclosporine achieved remission rates of 52 and 26 % in 73 and 22 % of patients, respectively [73].

The use of mammalian target of rapamycin (mTOR) inhibitors (e.g., everolimus, sirolimus) in AIH remains limited. These drugs may be considered for patients with a history of malignancy due to their antiproliferative effects. Everolimus has shown some efficacy in patients with AIH refractory to first- and second-line therapies, with 43 % achieving normal alanine aminotransferase levels and 57 % reducing alanine aminotransferase below 55 U/L after five months [75, 76].

Rescue therapy for non-response to first-, second-, and third-line treatments may include rituximab, infliximab, or belimumab. Treatment should be conducted in specialized centers experienced in managing this patient category and administering biologic therapies.

This received "complete agreement" (A+) from 75 % of participants and "agreement with minor objections" (A) from 25 %.

Grade of recommendation — C;
level of evidence — 4.

Treatment options for AIH remain limited for patients who fail first-, second-, and third-line therapies. For patients who are unresponsive to T-cell-targeted treatments, B-cell-targeted therapies are a logical next step [77]. The literature on these approaches is based on retrospective cohort studies, case series, and case reports, but the evidence base is growing [78].

Infliximab may be considered as emergency therapy in refractory AIH, though it carries a risk of infectious complications. In one study of 11 refractory AIH patients treated with infliximab for six months, biochemical remission was achieved, but seven patients developed infections [79].

Two international multicenter retrospective cohort studies evaluated rituximab in refractory AIH (22 and 35 patients, respectively). Biochemical remission was achieved and sustained in 70 and 89 % of cases over two years without significant adverse effects [80, 81].

Belimumab is also considered a promising treatment for refractory AIH. Case series involving two and three patients demonstrated sustained biochemical remission [82, 83].

The treatment of AIH in conjunction with other immune-mediated diseases presents a significant clinical challenge. Management of these patients requires a multidisciplinary team approach, involving a gastroenterologist alongside specialists tailored to the comorbid condition (e.g., rheumatologist, pulmonologist, neurologist, hematologist, endocrinologist, nephrologist, dermatologist, ophthalmologist, allergist, among others). In such cases, immunosuppressive therapy should prioritize medications and therapeutic regimens that are effective for both AIH and the associated comorbid condition.

Duration of immunosuppressive therapy in patients with cirrhosis

It is well-established that the goal of immunosuppressive therapy in AIH patients is to achieve complete remission and prevent the progression of liver damage. Treatment is therefore aimed at achieving complete biochemical response, histological resolution of inflammation, and ideally, regression of liver fibrosis [2, 21, 23].

For AIH patients without cirrhosis, there is a consensus on the duration of therapy and the possibility of its discontinuation. Immunosuppressive therapy is recommended for at least three years or for no less than two years after the complete normalization of aminotransferase and IgG levels, i.e., achieving a complete biochemical response, if a repeat liver biopsy is not performed. Therapy should not be discontinued in patients without sustained complete biochemical response (for at least two years). If complete biochemical response is maintained for more than two years, liver biopsy to determine the histological activity index (HAI) is advisable, though not strictly mandatory, before discontinuing immunosuppressive therapy. Ideally, therapy can be discontinued when full remission (HAI < 4) is achieved. If histological activity persists (HAI ≥ 4), therapy should not be discontinued [21, 23, 84].

Thus, under current guidelines, the necessary condition for discontinuing immunosuppressive therapy in non-cirrhotic patients is the maintenance of complete biochemical response for at least two years. A repeat liver biopsy is desirable but not strictly required to decide on therapy cessation. Discontinuing treatment and achieving prolonged AIH remission without therapy is possible in a minority of patients.

Regarding the duration of therapy in AIH patients with cirrhosis, immunosuppressive therapy is recommended for those with compensated cirrhosis. The risks and benefits of immunosuppressive therapy in patients with decompensated cirrhosis are less clear. Similarly, it is difficult to determine the necessity and benefit of immunosuppressive therapy in patients with inactive cirrhosis [21, 23, 52].

Observational data on AIH patients with compensated cirrhosis shows that most have normal aminotransferase and IgG levels. However, normal values do not always correlate with the absence of histological activity [31, 34, 49, 60, 84]. Complete tissue recovery reduces the risk of relapse, but in cirrhosis achieving full recovery is almost impossible. Approximately 45 % of patients with normal alanine aminotransferase and IgG levels may still have histological activity (HAI ≥ 4) [34].

There is no consensus on the appropriate duration of therapy for cirrhotic patients. Some studies suggest prescribing glucocorticoids for 18–24 months and azathioprine for 3–5 years before considering discontinuation [84]. Therapy cessation should only be attempted if aminotransferases and IgG remain normal throughout this period. The remission rate in patients with severe fibrosis is higher than in those with cirrhosis (78 % vs. 54 %; $p = 0.007$) [85].

Based on data analysis, the most significant criteria for considering therapy discontinuation in AIH patients with cirrhosis should include:

- regression of liver fibrosis (at least moderate fibrosis);
- regression of portal hypertension and thrombocytopenia (if present);
- regression of inflammatory activity (HAI < 4).

Given the lack of positive correlation between biochemical and histological activity, liver biopsy

should be performed in all cirrhotic patients planning to discontinue immunosuppressive therapy [23].

A comparative study of patients with continuous immunosuppressive therapy durations of more than four years versus 2–4 years showed that the likelihood of sustained biochemical response was 67 and 17 %, respectively [86]. Achieving complete biochemical response and remission in AIH patients with cirrhosis is extremely rare.

Some studies indicate that most AIH patients with compensated cirrhosis and prolonged therapy achieve full and sustained biochemical response, rarely develop decompensation, and in some cases demonstrate cirrhosis stabilization and/or regression of portal hypertension [50, 86].

Thus, the duration of immunosuppressive therapy in cirrhotic patients should be determined by several factors, as reflected in the consensus document:

Immunosuppressive therapy in cirrhotic patients due to AIH may only be discontinued with proven evidence of sustained complete remission and stabilization or recompensation of cirrhosis. Most cirrhotic patients require long-term immunosuppressive therapy at minimally effective maintenance doses.

This statement received "complete agreement" (A+) from 74.1 % of participants, "agreement with minor objections" (A) from 11.1 %, "agreement with significant concerns" (A–) from 7.4 %, and "disagreement with significant objections" (D–) from 3.7 %.

Grade of recommendation — C;
level of evidence — 3.

Conclusion

Based on the latest scientific data and their own clinical experience, specialists have formulated key positions regarding the management of autoimmune hepatitis patients in the framework of this consensus. These provisions, aimed at improving the quality of care for this patient category, are designed to assist clinicians in their practice and may serve as the foundation for updated national clinical guidelines.

References / Литература

1. *Ивашкин В.Т., Буеверов А.О., Абдулганиева Д.И.* Клинические рекомендации по диагностике и лечению аутоиммунного гепатита. М., 2013. [*Ivashkin V.T., Bueverov A.O., Abdulganieva D.I.* Clinical guidelines for the diagnosis and treatment of autoimmune hepatitis. Moscow, 2013. (In Russ.)].
2. *Lamba M., Ngu J.H., Stedman C.A.M.* Trends in incidence of autoimmune liver diseases and increasing incidence of autoimmune hepatitis. *Clin Gastroenterol Hepatol*. 2021;19(3):573–9.e1. DOI: 10.1016/j.cgh.2020.05.061
3. *Hahn J.W., Yang H.R., Moon J.S., Chang J.Y., Lee K., Kim G.A., et al.* Global incidence and prevalence of autoimmune hepatitis, 1970-2022: A systematic review and meta-analysis. *Eclinicalmedicine*. 2023;65:102280. DOI: 10.1016/j.eclinm.2023.102280
4. *van den Brand F.F., van der Veen K.S., de Boer Y.S., van Gerven N.M., Lissenberg-Witte B.I., Beuers U., et al.; Dutch Autoimmune Hepatitis Study Group.* Increased mortality among patients with vs without cirrhosis and autoimmune hepatitis. *Clin Gastroenterol Hepatol*. 2019;17(5):940–7.e2. DOI: 10.1016/j.cgh.2018.09.046
5. *Bittermann T., Lewis J.D., Levy C., Goldberg D.S.* Sociodemographic and geographic differences in the US epidemiology of autoimmune hepatitis with and without cirrhosis. *Hepatology*. 2023;77(2):367–78. DOI: 10.1002/hep.32653
6. *Guyatt G.H., Oxman A.D., Vist G.E., Kunz R., Falck-Ytter Y., Alonso-Coello P., et al.; GRADE Working Group.* GRADE: An emerging consensus on rating quality 36 of evidence and strength of recommendations. *BMJ*. 2008;336(7650):924–6. DOI: 10.1136/bmj.39489.470347.AD
7. *McGonagle D., McDermott M.F.* A proposed classification of the immunological diseases. *PLoS Med*. 2006;3(8):e297. DOI: 10.1371/journal.pmed.0030297
8. *Alvarez F., Berg P.A., Bianchi F.B., Bianchi L., Burroughs A.K., Cancado E.L., et al.* International autoimmune hepatitis group report: Review of criteria for diagnosis of autoimmune hepatitis. *J Hepatol*. 1999;31(5):929–38. DOI: 10.1016/s0168-8278(99)80297-9
9. *Hennes E.M., Zeniya M., Czaja A.J., Parés A., Dalekos G.N., Krawitt E.L., et al.; International Autoimmune Hepatitis Group.* Simplified criteria for the diagnosis of autoimmune hepatitis. *Hepatology*. 2008;48(1):169–76. DOI: 10.1002/hep.22322
10. *Wang Q., Yang F., Miao Q., Krawitt E.L., Gershwin M.E., Ma X.* The clinical phenotypes of autoimmune hepatitis: A comprehensive review. *J Autoimmun*. 2016;66:98–107. DOI: 10.1016/j.jaut.2015.10.006
11. *Czaja A.J., Bayraktar Y.* Non-classical phenotypes of autoimmune hepatitis and advances in diagnosis and treatment. *World J Gastroenterol*. 2009;15(19):2314–28. DOI: 10.3748/wjg.15.2314
12. *Terziroli Beretta-Piccoli B., Mieli-Vergani G., Vergani D.* Autoimmune hepatitis: Serum autoantibodies in clinical practice. *Clin Rev Allergy Immunol*. 2022;63(2):124–37. DOI: 10.1007/s12016-021-08888-9
13. *Czaja A.J.* Autoantibodies as prognostic markers in autoimmune liver disease. *Dig Dis Sci*. 2010;55(8):2144–61. DOI: 10.1007/s10620-010-1268-4
14. *Baeres M., Herkel J., Czaja A.J., Wies I., Kanzler S., Cancado E.L., et al.* Establishment of standardised SLA/LP immunoassays: Specificity for autoimmune hepatitis, worldwide occurrence, and clinical characteristics. *Gut*. 2002;51(2):259–64. DOI: 10.1136/gut.51.2.259
15. *Efe C., Ozaslan E., Wahlin S., Purnak T., Muratori L., Quarneti C., et al.* Antibodies to soluble liver antigen in patients with various liver diseases: A multicentre study. *Liver Int*. 2013;33(2):190–6. DOI: 10.1111/liv.12022
16. *Vergani D., Alvarez F., Bianchi F.B., Cançado E.L., Mackay I.R., Manns M.P., et al.; International Autoimmune Hepatitis Group.* Liver autoimmune serology: A consensus statement from the committee for autoimmune serology of the International Autoimmune Hepatitis Group. *J Hepatol*. 2004;41(4):677–83. DOI: 10.1016/j.jhep.2004.08.002
17. *Sebode M., Weiler-Normann C., Liwinski T., Schramm C.* Autoantibodies in autoimmune liver disease-clinical and diagnostic relevance. *Front Immunol*. 2018;9:609. DOI: 10.3389/fimmu.2018.00609

18. *Terjung B., Söhne J., Lechtenberg B., Gottwein J., Muennich M., Herzog V., et al.* p-ANCAs in autoimmune liver disorders recognise human beta-tubulin isotype 5 and cross-react with microbial protein FtsZ. *Gut*. 2010;59(6):808–16. DOI: 10.1136/gut.2008.157818
19. *Yimam K.K., Bowlus C.L.* Diagnosis and classification of primary sclerosing cholangitis. *Autoimmun Rev*. 2014;13(4–5):445–50. DOI: 10.1016/j.autrev.2014.01.040
20. *Terziroli Beretta-Piccoli B., Mieli-Vergani G., Vergani D.* Serology in autoimmune hepatitis: A clinical-practice approach. *Eur J Intern Med*. 2018;48:35–43. DOI: 10.1016/j.ejim.2017.10.006
21. *Mack C.L., Adams D., Assis D.N., Kerkar N., Manns M.P., Mayo M.J., et al.* Diagnosis and management of autoimmune hepatitis in adults and children: 2019 practice guidance and guidelines from the American Association for the Study of Liver Diseases. *Hepatology*. 2020;72(2):671–722. DOI: 10.1002/hep.31065
22. *Zachou K., Muratori P., Koukoulis G.K., Granito A., Gatselis N., Fabbri A., et al.* Review article: Autoimmune hepatitis – current management and challenges. *Aliment Pharmacol Ther*. 2013;38:887–913.
23. *European Association for the Study of the Liver.* EASL clinical practice guidelines: Autoimmune hepatitis. *J Hepatol*. 2015;63(4):971–1004. DOI: 10.1016/j.jhep.2015.06.030
24. *Czaja A.J.* Autoantibody-negative autoimmune hepatitis. *Dig Dis Sci*. 2012;57(3):610–24. DOI: 10.1007/s10620-011-2017-z
25. *Буеверов А.О.* Серонегативный аутоиммунный гепатит. *Российский журнал гастроэнтерологии, гепатологии, колопроктологии*. 2017;27(2):27–33. [*Bueverov A.O.* Seronegative autoimmune hepatitis. *Russian Journal of Gastroenterology, Hepatology, Coloproctology*. 2017;27(2):27–33. (In Russ.)].
26. *Galaski J., Weiler-Normann C., Schakat M., Zachou K., Muratori P., Lampalzer S., et al.* Update of the simplified criteria for autoimmune hepatitis: Evaluation of the methodology for immunoserological testing. *J Hepatol*. 2021;74(2):312–20. DOI: 10.1016/j.jhep.2020.07.032
27. *Chan E.K.L., von Mühlen C.A., Fritzler M.J., Damoiseaux J., Infantino M., Klotz W., et al.; ICAP Committee.* The International Consensus on ANA Patterns (ICAP) in 2021 – The 6th Workshop and current perspectives. *J Appl Lab Med*. 2022;7(1):322–30. DOI: 10.1093/jalm/jfab140
28. *Muñoz-Sánchez G., Pérez-Isidro A., Ortiz de Landazuri I., López-Gómez A., Bravo-Gallego L.Y., Garcia-Ormaechea M., et al.* on behalf of the Geai-Sei Workshop participants. Working algorithms and detection methods of autoantibodies in autoimmune liver disease: A nationwide study. *Diagnostics (Basel)*. 2022;12(3):697. DOI: 10.3390/diagnostics12030697
29. *Bogdanos D.P., Invernizzi P., Mackay I.R., Vergani D.* Autoimmune liver serology: Current diagnostic and clinical challenges. *World J. Gastroenterol*. 2008;14(21):3374–87. DOI: 10.3748/wjg.14.3374
30. *Александрова Е.Н., Дорофеев А.С., Новиков А.А., Сандлер Ю.Г.* Аутоантитела при аутоиммунных заболеваниях печени (обзор литературы). *Клиническая лабораторная диагностика*. 2023;68(8):464–74. [*Aleksandrova E.N., Dorofeev A.S., Novikov A.A., Sandler Yu.G.* Autoantibodies in autoimmune liver diseases (literature review). *Russian Clinical Laboratory Diagnostics*. 2023;68(8):464–74. (In Russ.)]. DOI: 10.51620/0869-2084-2023-68-8-464-474
31. *Laschtowitz A., Zachou K., Lygoura V., Pape S., Derben F., Jaeckel E., et al.* Histological activity despite normal ALT and IgG serum levels in patients with autoimmune hepatitis and cirrhosis. *JHEP Rep*. 2021;3(4):100321. DOI: 10.1016/j.jhepr.2021.100321
32. *van Gerven N.M., Verwer B.J., Witte B.I., van Hoek B., Coenraad M.J., van Erpecum K.J., et al.; Dutch Autoimmune Hepatitis Working Group.* Relapse is almost universal after withdrawal of immunosuppressive medication in patients with autoimmune hepatitis in remission. *J Hepatol*. 2013;58(1):141–7. DOI: 10.1016/j.jhep.2012.09.009
33. *Lohse A.W., Sebode M., Bhathal P.S., Clouston A.D., Dienes H.P., Jain D., et al.* Consensus recommendations for histological criteria of autoimmune hepatitis from the International AIH Pathology Group: Results of a workshop on AIH histology hosted by the European Reference Network on Hepatological Diseases and the European Society of Pathology: Results of a workshop on AIH histology hosted by the European Reference Network on Hepatological Diseases and the European Society of Pathology. *Liver Int*. 2022;42(5):1058–69. DOI: 10.1111/liv.15217
34. *Dhaliwal H.K., Hoeroldt B.S., Dube A.K., McFarlane E., Underwood J.C., Karajeh M.A., et al.* Long-term prognostic significance of persisting histological activity despite biochemical remission in autoimmune hepatitis. *Am J Gastroenterol*. 2015;110(7):993–9. DOI: 10.1038/ajg.2015.139
35. *Knodell R.G., Ishak K.G., Black W.C., Chen T.S., Craig R., Kaplowitz N., et al.* Formulation and application of a numerical scoring system for assessing histological activity in asymptomatic chronic active hepatitis. *Hepatology*. 1981;1(5):431–5. DOI: 10.1002/hep.1840010511
36. *Hartl J., Denzer U., Ehlken H., Zenouzi R., Peiseler M., Sebode M., et al.* Transient elastography in autoimmune hepatitis: Timing determines the impact of inflammation and fibrosis. *J Hepatol*. 2016;65(4):769–75. DOI: 10.1016/j.jhep.2016.05.023
37. *Lai J.C., Liang L.Y., Wong G.L.* Noninvasive tests for liver fibrosis in 2024: Are there different scales for different diseases? *Gastroenterol Rep (Oxf)*. 2024;12:goae096. DOI: 10.1093/gastro/goae096
38. *Ishak K., Baptista A., Bianchi L., Callea F., De Groote J., Gudat F., et al.* Histological grading and staging of chronic hepatitis. *J Hepatol*. 1995;22(6):696–9. DOI: 10.1016/0168-8278(95)80226-6
39. *Ahn S., Jeong S.H., Cho E.J., Lee K., Kim G., Kim H.* Comparison of four histological scoring systems for autoimmune hepatitis to improve diagnostic sensitivity. *Clin Mol Hepatol*. 2024;30(1):37–48. DOI: 10.3350/cmh.2023.0325
40. *van Buuren H.R., Lammers W.J., Harms M.H., Hansen B.E.* Surrogate endpoints for optimal therapeutic response to UDCA in primary biliary cholangitis. *Dig Dis*. 2015;33 Suppl 2:118–24. DOI: 10.1159/000440759
41. *Ponsioen C.Y., Lindor K.D., Mehta R., Dimick-Santos L.* Design and endpoints for clinical trials in primary sclerosing cholangitis. *Hepatology*. 2018;68(3):1174–88. DOI: 10.1002/hep.29882
42. *Cook G.C., Mulligan R., Sherlock S.* Controlled prospective trial of corticosteroid therapy in active chronic hepatitis. *Q J Med*. 1971;40(158):159–85. DOI: 10.1093/oxfordjournals.qjmed.a067264
43. *Manns M.P., Woynarowski M., Kreisel W., Lurie Y., Rust C., Zuckerman E., et al.* Budesonide induces remission more effectively than prednisone in a controlled trial of patients with autoimmune hepatitis. *Gastroenterology*. 2010;139(4):1198–206. DOI: 10.1053/j.gastro.2010.06.046
44. *Pape S., Snijders R.J.A.L.M., Gevers T.J.G., Chazouilleres O., Dalekos G.N., Hirschfield G.M., et al.; International Autoimmune Hepatitis Group (IAIHG) collaborators.* Systematic review of response criteria and endpoints in autoimmune hepatitis by the International Autoimmune Hepatitis Group. *J Hepatol*. 2022;76(4):841–9. DOI: 10.1016/j.jhep.2021.12.041
45. *Czaja A.J.* Diagnosis and management of autoimmune hepatitis: Current status and future directions. *Gut Liver*. 2016;10(2):177–203. DOI: 10.5009/gnl15352
46. *Urzúa Á., Pizarro C., Gajardo A., Poniachik R., Pavez C., Cattaneo M., et al.* Autoimmune hepatitis with acute presentation: Clinical, biochemical, and histological features of 126 patients. *Can J Gastroenterol Hepatol*. 2022;2022:6470847. DOI: 10.1155/2022/6470847

47. *Yasui S., Fujiwara K., Yonemitsu Y., Oda S., Nakano M., Yokosuka O.* Clinicopathological features of severe and fulminant forms of autoimmune hepatitis. *J Gastroenterol*. 2011;46(3):378–90. DOI: 10.1007/s00535-010-0316-3
48. *Biewenga M., Verhelst X., Baven-Pronk M., Putter H., van den Berg A., Colle I., et al.; Dutch Autoimmune Hepatitis Study Group.* Aminotransferases during treatment predict long-term survival in patients with autoimmune hepatitis type 1: A landmark analysis. *Clin Gastroenterol Hepatol*. 2022;20(8):1776–83.e4. DOI: 10.1016/j.cgh.2021.05.024
49. *Plagiannakos C.G., Hirschfield G.M., Lytvyak E., Ro-berts S.B., Ismail M., Gulamhusein A.F., et al.; Canadian Network for Autoimmune Liver Disease (CaNAL).* Treatment response and clinical event-free survival in autoimmune hepatitis: A Canadian multicentre cohort study. *J Hepatol*. 2024;81(2):227–37. DOI: 10.1016/j.jhep.2024.03.021
50. *Grossar L., Raevens S., Van Steenkiste C., Colle I., De Vloo C., Orlent H., et al.* External validation of the IAIHG autoimmune hepatitis response criteria in a multicentric real-world cohort. *JHEP Reports*. 2024;6(9):101149. DOI: 10.1016/j.jhepr.2024.101149
51. *Czaja A.J.* Features and consequences of untreated type 1 autoimmune hepatitis. *Liver Int*. 2009;29(6):816–23. DOI: 10.1111/j.1478-3231.2008.01904.x
52. *Wang G., Tanaka A., Zhao H., Jia J., Ma X., Harada K., et al.* The Asian Pacific Association for the Study of the Liver clinical practice guidance: The diagnosis and management of patients with autoimmune hepatitis. *Hepatol Int*. 2021;15(2):223–57. DOI: 10.1007/s12072-021-10170-1
53. *Czaja A.J.* Treatment of autoimmune hepatitis. *Semin Liver Dis*. 2002;22(4):365–78. DOI: 10.1055/s-2002-35706
54. *Lamers M.M., van Oijen M.G., Pronk M., Drenth J.P.* Treatment options for autoimmune hepatitis: A systematic review of randomized controlled trials. *J Hepatol*. 2010;53(1):191–8. DOI: 10.1016/j.jhep.2010.01.037
55. *Pape S., Gevers T.J.G., Vrolijk J.M., van Hoek B., Bouma G., van Nieuwkerk C.M.J., et al.* High discontinuation rate of azathioprine in autoimmune hepatitis, independent of time of treatment initiation. *Liver Int*. 2020;40(9):2164–71. DOI: 10.1111/liv.14513
56. *Zhang C., Wu S.S., Dong X.Q., Wu Z., Zhao H., Wang G.Q.* The efficacy and safety of different doses of glucocorticoid for autoimmune hepatitis: A systematic review and meta-analysis. *Medicine (Baltimore)*. 2019;98(52):e18313. DOI: 10.1097/MD.0000000000018313
57. *Stellon A.J., Hegarty J.E., Portmann B., Williams R.* Randomised controlled trial of azathioprine withdrawal in autoimmune chronic active hepatitis. *Lancet*. 1985;1(8430):668–70. DOI: 10.1016/s0140-6736(85)91329-7
58. *Czaja A.J.* Rapidity of treatment response and outcome in type 1 autoimmune hepatitis. *J Hepatol*. 2009;51(1):161–7. DOI: 10.1016/j.jhep.2009.02.026
59. *Santiago P., Schwartz I., Tamariz L., Levy C.* Systematic review with meta-analysis: Mycophenolate mofetil as a second-line therapy for autoimmune hepatitis. *Aliment Pharmacol Ther*. 2019;49(7):830–9. DOI: 10.1111/apt.15157
60. *Dalekos G.N., Arvaniti P., Gatselis N.K., Gabeta S., Samakidou A., Giannoulis G., et al.* Long-term results of mycophenolate mofetil vs. azathioprine use in individuals with autoimmune hepatitis. *JHEP Rep*. 2022;4(12):100601. DOI: 10.1016/j.jhepr.2022.100601
61. *Dalekos G.N., Arvaniti P., Gatselis N.K., Samakidou A., Gabeta S., Rigopoulou E., et al.* First results from a propensity matching trial of mycophenolate mofetil vs. azathioprine in treatment-naive AIH patients. *Front Immunol*. 2022;12:798602. DOI: 10.3389/fimmu.2021.798602
62. *Snijders R.J.A.L.M., Stoelinga A.E.C., Gevers T.J.G., Pape S., Biewenga M., Tushuizen M.E., et al.; Dutch Autoimmune Hepatitis Working Group.* An open-label randomised-controlled trial of azathioprine vs. mycophenolate mofetil for the induction of remission in treatment-naive autoimmune hepatitis. *J Hepatol*. 2024;80(4):576–85. DOI: 10.1016/j.jhep.2023.11.032
63. *Terziroli Beretta-Piccoli B., Mieli-Vergani G., Vergani D.* Autoimmune hepatitis: Standard treatment and systematic review of alternative treatments. *World J Gastroenterol*. 2017;23(33):6030–48. DOI: 10.3748/wjg.v23.i33.6030
64. *Czaja A.J., Lindor K.D.* Failure of budesonide in a pilot study of treatment-dependent autoimmune hepatitis. *Gastroenterology*. 2000;119(5):1312–6. DOI: 10.1053/gast.2000.0010000001
65. *Woynarowski M., Nemeth A., Baruch Y., Koletzko S., Melter M., Rodeck B., et al.; European Autoimmune Hepatitis-Budesonide Study Group.* Budesonide versus prednisone with azathioprine for the treatment of autoimmune hepatitis in children and adolescents. *J Pediatr*. 2013;163(5):1347–53.e1. DOI: 10.1016/j.jpeds.2013.05.042
66. *Peiseler M., Liebscher T., Sebode M., Zenouzi R., Hartl J., Ehlken H., et al.* Efficacy and limitations of budesonide as a second-line treatment for patients with autoimmune hepatitis. *Clin Gastroenterol Hepatol*. 2018;16(2):260–7.e1. DOI: 10.1016/j.cgh.2016.12.040
67. *Wiegand J., Schüler A., Kanzler S., Lohse A., Beuers U., Kreisel W., et al.* Budesonide in previously untreated autoimmune hepatitis. *Liver Int*. 2005;25(5):927–34. DOI: 10.1111/j.1478-3231.2005.01122.x
68. *Lu F.B., Hu E.D., Xu L.M., Hu Y.B., Chen L., Wu J.L., et al.* Comparative efficacy and tolerability of treatments for adult autoimmune hepatitis: A systematic review and network meta-analysis. *Exp Ther Med*. 2018;15(6):4838–50. DOI: 10.3892/etm.2018.6063
69. *Hanouneh M., Ritchie M.M., Ascha M., Ascha M.S., Chedid A., Sanguankeo A., et al.* A review of the utility of tacrolimus in the management of adults with autoimmune hepatitis. *Scand J Gastroenterol*. 2019;54(1):76–80. DOI: 10.1080/00365521.2018.1551498
70. *Stoelinga A.E.C., Tushuizen M.E., van den Hout W.B., Girondo M.D.M.R., de Vries E.S., Levens A.D., et al.; Dutch Autoimmune Hepatitis Group.* Tacrolimus versus mycophenolate for autoimmune hepatitis patients with incomplete response on first-line therapy (TAILOR study): A study protocol for a phase III, open-label, multicentre, randomised controlled trial. *Trials*. 2024;25(1):61. DOI: 10.1186/s13063-023-07832-w
71. *De Lemos-Bonotto M., Valle-Tovo C., Costabeber A.M., Mattos A.A., Azeredo-da-Silva A.L.F.* A systematic review and meta-analysis of second-line immunosuppressants for autoimmune hepatitis treatment. *Eur J Gastroenterol Hepatol*. 2018;30(2):212–6. DOI: 10.1097/MEG.0000000000001019
72. *Tannous M.M., Cheng J., Muniyappa K., Farooq I., Bharara A., Kappus M., et al.* Use of tacrolimus in the treatment of autoimmune hepatitis: A single centre experience. *Aliment Pharmacol Ther*. 2011;34(3):405–7. DOI: 10.1111/j.1365-2036.2011.04749.x
73. *Baven-Pronk M.A., Hew J.M. Jr., Biewenga M., Tushuizen M.E., van den Berg A.P., Bouma G., et al.; Dutch Autoimmune Hepatitis Study Group.* Calcineurin inhibitors in the treatment of adult autoimmune hepatitis: A systematic review. *J Clin Transl Hepatol*. 2022;10(6):1155–66. DOI: 10.14218/JCTH.2021.00535
74. *Efe C., Taii H.A., Ytting H., Aehling N., Bhanji R.A., Hagstrom H., et al.* Tacrolimus and mycophenolate mofetil as second-line therapies for pediatric patients with autoimmune hepatitis. *Dig Dis Sci*. 2018;63(5):1348–54. DOI: 10.1007/s10620-018-5011-x
75. *Ytting H., Larsen F.S.* Everolimus treatment for patients with autoimmune hepatitis and poor response to standard therapy and drug alternatives in use. *Scand J Gastroenterol*. 2015;50(8):1025–31. DOI: 10.3109/00365521.2014.998271
76. *Porcelli L., Quatrale A.E., Mantuano P., Silvestris N., Rolland J.F., Biancolillo L., et al.* Synergistic antiproliferative and antiangiogenic effects of EGFR and mTOR inhibitors. *Curr Pharm Des*. 2013;19(5):918–26.

77. *Weinberg E.M.* Current and emerging treatments for autoimmune hepatitis. *Gastroenterol Hepatol (N Y)*. 2024;20(3):176–8.
78. *Adao I., Klepper A., Tana M.* AIH therapy: Beyond first-line. *Curr Hepatology Rep*. 2024;23:341–8. DOI: 10.1007/s11901-024-00657-4
79. *Weiler-Normann C., Schramm C., Quaas A., Wiegard C., Glaubke C., Pannicke N., et al.* Infliximab as a rescue treatment in difficult-to-treat autoimmune hepatitis. *J Hepatol*. 2013;58(3):529–34. DOI: 10.1016/j.jhep.2012.11.010
80. *Than N.N., Hodson J., Schmidt-Martin D., Taubert R., Wawman R.E., Botter M., et al.* Efficacy of rituximab in difficult-to-manage autoimmune hepatitis: Results from the International Autoimmune Hepatitis Group. *JHEP Rep*. 2019;1(6):437–45. DOI: 10.1016/j.jhepr.2019.10.005
81. *Riveiro-Barciela M., Barreira-Díaz A., Esteban P., Rota R., Álvarez-Navascúes C., Pérez-Medrano I., et al.* Rituximab is a safe and effective alternative treatment for patients with autoimmune hepatitis: Results from the ColHai registry. *Liver Int*. 2024;44(9):2303–14. DOI: 10.1111/liv.15970
82. *Kolev M., Sarbu A.C., Möller B., Maurer B., Kollert F., Semmo N.* Belimumab treatment in autoimmune hepatitis and primary biliary cholangitis — a case series. *J Transl Autoimmun*. 2023;6:100189. DOI: 10.1016/j.jtauto.2023.100189
83. *Arvaniti P., Giannoulis G., Gabeta S., Zachou K., Koukoulis G.K., Dalekos G.N.* Belimumab is a promising third-line treatment option for refractory autoimmune hepatitis. *JHEP Rep*. 2020;2(4):100123. DOI: 10.1016/j.jhepr.2020.100123
84. *Czaja A.J., Carpenter H.A.* Histological features associated with relapse after corticosteroid withdrawal in type 1 autoimmune hepatitis. *Liver Int*. 2003;23(2):116–23. DOI: 10.1034/j.1600-0676.2003.00810.x
85. *Çavuş B., Akyuz F., Iliaz R., Atasoy A., Akyuz U., Demir K., et al.* Is there any predictor for relapse after treatment withdrawal in autoimmune hepatitis patients in the real life? *Int J Immunopathol Pharmacol*. 2022;36:3946320221077860. DOI: 10.1177/03946320221077860
86. *Hatoum S., Rockey D.C.* Long-term outcomes of patients with autoimmune hepatitis induced cirrhosis after immunosuppressive treatment. *Eur J Gastroenterol Hepatol*. 2024;36(6):742–9. DOI: 10.1097/MEG.0000000000002714.

Information about the authors

Yuliya G. Sandler* — Cand. Sci. Med., Senior Researcher of the Department of Hepatology, Loginov Moscow Clinical Scientific Center.
Contact information: sandlerjulia2012@gmail.com;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0003-4291-812X

Elena V. Vinnitskaya — Dr. Sci. (Med.), Head of the Department of Hepatology, Loginov Moscow Clinical Scientific Center.
Contact information: evinn@mail.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0002-0344-8375

Karina L. Raikhelson — Dr. Sci. (Med.), Professor of the Scientific and Educational Center of Gastroenterology and Hepatology, Saint Petersburg State University.
Contact information: kraikhelson@mail.ru;
199226, Saint Petersburg, Korablestroiteley str., 20–1.
ORCID: https://orcid.org/0000-0002-8821-6142

Konstantin V. Ivashkin — Dr. Sci. Med., Professor of the Department of Propaedeutics of Internal Medicine, Gastroenterology and Hepatology of the N.V. Sklifosovsky Institute of Clinical Medicine, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: ivashkin_k_v_1@staff.sechenov.ru;
119435, Moscow, Pogodinskaya str., 1, build. 1.
ORCID: https://orcid.org/0000-0002-5699-541X

Сведения об авторах

Сандлер Юлия Григорьевна* — кандидат медицинских наук, старший научный сотрудник отдела гепатологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: sandlerjulia2012@gmail.com;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0003-4291-812X

Винницкая Елена Владимировна — доктор медицинских наук, заведующая отделом гепатологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: evinn@mail.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0002-0344-8375

Райхельсон Карина Леонидовна — доктор медицинских наук, профессор Научно-клинического и образовательного центра гастроэнтерологии и гепатологии, ФГБОУ ВО «Санкт-Петербургский государственный университет».
Контактная информация: kraikhelson@mail.ru;
199226, г. Санкт-Петербург, ул. Кораблестроителей, 20, корп. 1.
ORCID: https://orcid.org/0000-0002-8821-6142

Ивашкин Константин Владимирович — доктор медицинских наук, профессор кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии Института клинической медицины им. Н.В. Склифосовского, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: ivashkin_k_v_1@staff.sechenov.ru;
119435, г. Москва, ул. Погодинская, 1, стр. 1.
ORCID: https://orcid.org/0000-0002-5699-541X

* Corresponding author / Автор, ответственный за переписку

Sergey N. Batskikh — Dr. Sci. (Med.), Leading Researcher of the Department of Hepatology, Loginov Moscow Clinical Scientific Center.
Contact information: zdoc@mail.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0002-5917-203X

Elena N. Aleksandrova — Dr. Sci. (Med.), Head of the Laboratory of Clinical Immunology, Loginov Moscow Clinical Scientific Center.
Contact information: aleksandrovaen2015@yandex.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0003-4074-5907

Dzhamal T. Abdurakhmanov — Dr. Sci. (Med.), Professor of the Department of Internal, Occupational Diseases and Pulmonology, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: abdurakhmanov_d_t@staff.sechenov.ru;
119435, Moscow, Pogodinskaya str., 1, build. 1.
ORCID: https://orcid.org/0000-0002-3160-2771

Diana I. Abdulganieva — Dr. Sci. Med., Professor, Head of the Department of Hospital Therapy, Kazan State Medical University.
Contact information: diana_s@mail.ru;
420012, Kazan, Butlerova str., 49
ORCID: https://orcid.org/0000-0001-7069-2725

Igor G. Bakulin — Dr. Sci. (Med.), Professor, Head of the Department of Propaedeutics of Internal Medicine, Gastroenterology and Dietetics named after S.M. Ryss, North-Western State Medical University named after I.I. Mechnikov.
Contact information: igbakulin@yandex.ru;
191015, Saint Petersburg, Kirochnaya str., 41.
ORCID: https://orcid.org/0000-0002-6151-2021

Alexey O. Bueverov — Dr. Sci. (Med.), Professor at the Department of Medical and Social Expertise, Emergency and Pathogenetic Therapy, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: bueverov_a_o@staff.sechenov.ru;
119435, Moscow, Pogodinskaya str., 1, build. 1.
ORCID: https://orcid.org/0000-0002-5041-3466

Sergey L. Vorobyev — Cand. Sci. (Med.), Director, OOO National Center for Clinical Morphological Diagnostics.
Contact information: ncmd@ncmd.ru;
192071, Saint Petersburg, Slavy Ave., 32, lit. A.
ORCID: https://orcid.org/0000-0002-7817-9069

Бацких Сергей Николаевич — доктор медицинских наук, ведущий научный сотрудник отдела гепатологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: zdoc@mail.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0002-5917-203X

Александрова Елена Николаевна — доктор медицинских наук, заведующая лабораторией клинической иммунологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: aleksandrovaen2015@yandex.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0003-4074-5907

Абдурахманов Джамал Тинович — доктор медицинских наук, профессор кафедры внутренних, профессиональных болезней и пульмонологии, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: abdurakhmanov_d_t@staff.sechenov.ru;
119435, г. Москва, ул. Погодинская, 1, стр. 1.
ORCID: https://orcid.org/0000-0002-3160-2771

Абдулганиева Диана Ильдаровна — доктор медицинских наук, профессор, заведующая кафедрой госпитальной терапии, ФГБОУ ВО «Казанский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
Контактная информация: diana_s@mail.ru;
420012, г. Казань, ул. Бутлерова, 49.
ORCID: https://orcid.org/0000-0001-7069-2725

Бакулин Игорь Геннадьевич — доктор медицинских наук, профессор, заведующий кафедрой пропедевтики внутренних болезней, гастроэнтерологии и диетологии им. С.М. Рысса, ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации.
Контактная информация: igbakulin@yandex.ru;
191015, г. Санкт-Петербург, ул. Кирочная, 41.
ORCID: https://orcid.org/0000-0002-6151-2021

Буеверов Алексей Олегович — доктор медицинских наук, профессор кафедры медико-социальной экспертизы, неотложной и патогенетической терапии, ФГАОУ ВО «Первый Московский государственный университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский университет).
Контактная информация: bueverov_a_o@staff.sechenov.ru;
119435, г. Москва, ул. Погодинская, 1, стр. 1.
ORCID: https://orcid.org/0000-0002-5041-3466

Воробьев Сергей Леонидович — кандидат медицинских наук, директор, ООО «Национальный центр клинической морфологической диагностики».
Контактная информация: ncmd@ncmd.ru;
192071, г. Санкт-Петербург, просп. Славы, 32, лит. А.
ORCID: https://orcid.org/0000-0002-7817-9069

Olga A. Gerasimova — Dr. Sci. Med., Professor of the Scientific and Educational Center of Gastroenterology and Hepatology, Saint Petersburg State University; Head of the Outpatient Center for Transplantology, Hepatology and Nephrology, Russian Scientific Center of Radiology and Surgical Technologies named after Academician A.M. Granov.
Contact information: ren321@mail.ru;
199226, Saint Petersburg, Korablestroiteley str., 20–1.
ORCID: https://orcid.org/0000-0003-2690-5062

Anastasia I. Dolgushina — Dr. Sci. (Med.), Head of the Department of Hospital Therapy, South Ural State Medical University.
Contact information: dolgushinaai@yandex.ru;
454092, Chelyabinsk, Vorovskogo str., 64.
ORCID: https://orcid.org/0000-0003-2569-1699

Mariia S. Zhuravleva — Cand. Sci. (Med.), Associate Professor of the Department of Propaedeutics of Internal Medicine, Gastroenterology and Dietetics named after S.M. Ryss, North-Western State Medical University named after I.I. Mechnikov.
Contact information: mariya.zhuravleva@szgmu.ru;
191015, Saint Petersburg, Kirochnaya str., 41.
ORCID: https://orcid.org/0000-0003-4838-5707

Liudmila Yu. Ilchenko — Dr. Sci. (Med.), Professor, Professor of the Department of Hospital Therapy named after Academician G.I. Storozhakov, N.I. Pirogov Russian National Research Medical University.
Contact information: ilchenko-med@yandex.ru;
117513, Moscow, Ostrovityanova str., 1.
ORCID: https://orcid.org/0000-0001-6029-1864

Vadim E. Karev — Dr. Sci. (Med.), Head of the Department of Tissue and Pathomorphological Research Methods, Pediatric Research and Clinical Center for Infectious Diseases under the Federal Medical Biological Agency.
Contact information: vadimkarev@yandex.ru;
197022, Saint Petersburg, Professora Popova str., 9.
ORCID: https://orcid.org/0000-0002-7972-1286

Natalia V. Korochanskaya — Dr. Sci. (Med.), Professor of the Department of Surgery No. 3 of the Faculty of Advanced Training and Professional Retraining of Specialists, Kuban State Medical University.
Contact information: nvk-gastro@mail.ru;
350063, Krasnodar, Mitrofana Sedina str., 4.
ORCID: https://orcid.org/0000-0002-5538-9418

Герасимова Ольга Анатольевна — доктор медицинских наук, профессор Научно-клинического и образовательного центра гастроэнтерологии и гепатологии, ФГБОУ ВО «Санкт-Петербургский государственный университет»; руководитель амбулаторного центра трансплантологии, гепатологии и нефрологии, ФГБУ «Российский научный центр радиологии и хирургических технологий им. академика А.М. Гранова» Министерства здравоохранения Российской Федерации.
Контактная информация: ren321@mail.ru;
199226, г. Санкт-Петербург, ул. Кораблестроителей, 20, корп. 1.
ORCID: https://orcid.org/0000-0003-2690-5062

Долгушина Анастасия Ильинична — доктор медицинских наук, заведующая кафедрой госпитальной терапии, ФГБОУ ВО «Южно-Уральский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
Контактная информация: dolgushinaai@yandex.ru;
454092, г. Челябинск, ул. Воровского, 64.
ORCID: https://orcid.org/0000-0003-2569-1699

Журавлева Мария Сергеевна — кандидат медицинских наук, доцент кафедры пропедевтики внутренних болезней, гастроэнтерологии и диетологии им. С.М. Рысса, ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации.
Контактная информация: mariya.zhuravleva@szgmu.ru;
191015, г. Санкт-Петербург, ул. Кирочная, 41.
ORCID: https://orcid.org/0000-0003-4838-5707

Ильченко Людмила Юрьевна — доктор медицинских наук, профессор, профессор кафедры госпитальной терапии им. академика Г.И. Сторожакова, ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации.
Контактная информация: ilchenko-med@yandex.ru;
117513, г. Москва, ул. Островитянова, 1.
ORCID: https://orcid.org/0000-0001-6029-1864

Карев Вадим Евгеньевич — доктор медицинских наук, руководитель отдела тканевых и патоморфологических методов исследования, ФГБУ «Детский научно-клинический центр инфекционных болезней Федерального медико-биологического агентства».
Контактная информация: vadimkarev@yandex.ru;
197022, г. Санкт-Петербург, ул. Профессора Попова, 9.
ORCID: https://orcid.org/0000-0002-7972-1286

Корочанская Наталья Всеволодовна — доктор медицинских наук, профессор кафедры хирургии № 3 факультета повышения квалификации и профессиональной переподготовки специалистов, ФГБОУ ВО «Кубанский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
Контактная информация: nvk-gastro@mail.ru;
350063, г. Краснодар, ул. Митрофана Седина, 4.
ORCID: https://orcid.org/0000-0002-5538-9418

Irina L. Kliaritskaia — Dr. Sci. (Med.), Professor, Head of the Department of Therapy, Gastroenterology, Cardiology and General Medical Practice (Family Medicine) of the Fa-culty of Training of Highly Qualified Medical Personnel and Continuing Professional Education of the S.I. Georgievsky Medical Academy, V.I. Vernadsky Crimean Federal University.
Contact information: klira3@ yandex.ru;
295051, Simferopol, Lenina Blvd, 5/7.
ORCID: https://orcid.org/0000-0001-9840-7885

Кляритская Ирина Львовна — доктор медицинских наук, профессор, заведующая кафедрой терапии, гастроэнтерологии, кардиологии и общей врачебной практики (семейной медицины) факультета подготовки медицинских кадров высшей квалификации и дополнительного профессионального образования Медицинской академии им. С.И. Георгиевского, ФГАОУ ВО «Крымский федеральный университет им. В.И. Вернадского».
Контактная информация: klira3@ yandex.ru;
295051, г. Симферополь, б-р Ленина, 5/7.
ORCID: https://orcid.org/0000-0001-9840-7885

Nikolay S. Karnaukhov — Cand. Sci. (Med.), Head of the Department of Pathomorphology, Loginov Moscow Clinical Scientific Center.
Contact information: nick07@bk.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0003-0889-2720

Карнаухов Николай Сергеевич — кандидат медицинских наук, заведующий отделом патоморфологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: nick07@bk.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0003-0889-2720

Sergey V. Lapin — Cand. Sci. (Med.), Head of the Laboratory for Diagnostics of Autoimmune Diseases of the Scientific and Methodological Center for Molecular Medicine of the Ministry of Health of the Russian Federation, Academician I.P. Pavlov First St. Petersburg State Medical University.
Contact information: svlapin@mail.ru;
197101, Saint Petersburg, L'va Tolstovo str., 6-8.
ORCID: https://orcid.org/0000-0002-4998-3699

Лапин Сергей Владимирович — кандидат медицинских наук, заведующий лабораторией диагностики аутоиммунных заболеваний Научно-методического центра по молекулярной медицине Министерства здравоохранения Российской Федерации, ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. академика И.П. Павлова» Министерства здравоохранения Российской Федерации.
Контактная информация: svlapin@mail.ru;
197101, г. Санкт-Петербург, ул. Льва Толстого, 6-8.
ORCID: https://orcid.org/0000-0002-4998-3699

Maria A. Livzan — Dr. Sci. (Med.), Corresponding Member of the Russian Academy of Sciences, Professor, Head of the Department of Faculty Therapy and Gastroenterology, Rector, Omsk State Medical University.
Contact information: mlivzan@yandex.ru;
644099, Omsk, Lenina str., 12.
ORCID: https://orcid.org/0000-0002-6581-7017

Ливзан Мария Анатольевна — доктор медицинских наук, профессор, член-корреспондент РАН, заведующий кафедрой факультетской терапии и гастроэнтерологии, ректор, ФГБОУ ВО «Омский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
Контактная информация: mlivzan@yandex.ru;
644099, Россия, г. Омск, ул. Ленина, 12.
ORCID: https://orcid.org/0000-0002-6581-7017

Marina V. Maevskaya — Dr. Sci. (Med.), Professor, Consultant at the Diagnostic and Treatment Department of the University Clinical Hospital No. 2, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: mvmaevskaya@me.com;
119048, Moscow, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0001-8913-140X

Маевская Марина Викторовна — доктор медицинских наук, профессор, консультант лечебно-диагностического отделения Университетской клинической больницы № 2, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: mvmaevskaya@me.com;
119048, г. Москва, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0001-8913-140X

Natalia V. Marchenko — Cand. Sci. Med., Associate Professor of the Scientific and Educational Center of Gastroenterology and Hepatology, Saint Petersburg State University; Gastroenterologist, Russian Scientific Center of Radiology and Surgical Technologies named after Academician A.M. Granov.
Contact information: dr.marchenko@gmail.com;
199226, Saint Petersburg, Korablestroiteley str., 20.
ORCID: https://orcid.org/0000-0002-6738-6417

Марченко Наталья Валерьевна — кандидат медицинских наук, доцент Научно-клинического и образовательного центра гастроэнтерологии и гепатологии, ФГБОУ ВО «Санкт-Петербургский государственный университет»; врач-гастроэнтеролог, ФГБУ «Российский научный центр радиологии и хирургических технологий им. академика А.М. Гранова» Министерства здравоохранения Российской Федерации.
Контактная информация: dr.marchenko@gmail.com;
199226, г. Санкт-Петербург, ул. Кораблестроителей, 20, корп. 1.
ORCID: https://orcid.org/0000-0002-6738-6417

Tatiyana P. Nekrasova — Cand. Sci. (Med.), Associate Professor at the Institute of Clinical Morphology and Digital Pathology, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: nekrasova_t_p@staff.sechenov.ru;
119435, Moscow, Pogodinskaya str., 1, build. 1.
ORCID: https://orcid.org/0000-0001-6376-9392

Igor G. Nikitin — Dr. Sci. (Med.), Professor, Head of the Department of Hospital Therapy No. 2 of the Faculty of Therapy, N.I. Pirogov Russian National Research Medical University.
Contact information: igor.nikitin.64@mail.ru;
117513, Moscow, Ostrovityanova str., 1.
ORCID: https://orcid.org/0000-0003-1699-0881

Alexander A. Novikov — Dr. Sci. (Med.), Leading Researcher of the Laboratory of Clinical Immunology, Loginov Moscow Clinical Scientific Center.
Contact information: a.novikov@mknc.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0002-2738-2956

Rafik G. Saifutdinov — Dr. Sci. (Med.), Head of the Department of Therapy, Kazan State Medical Academy — Branch Campus of the Russian Medical Academy of Continuous Professional Education.
Contact information: rgsbancorp@mail.ru;
420012, Kazan, Butlerova str., 36.
ORCID: https://orcid.org/0000-0003-2839-100X

Ekaterina V. Skazyvaeva — Cand. Sci. (Med.), Associate Professor of the Department of Propaedeutics of Internal Medicine, Gastroenterology and Dietetics named after S.M. Ryss, North-Western State Medical University named after I.I. Mechnikov.
Contact information: eskazyvaeva@yandex.ru;
191015, St. Petersburg, Kirochnaya str., 41.
ORCID: https://orcid.org/0000-0002-8563-6870

Vladimir E. Syutkin — Dr. Sci. (Med.), Professor of the Department of Surgery with Courses in Oncosurgery, Endoscopy, Surgical Pathology, Clinical Transplantology and Organ Donation, Medical and Biological University of Innovation and Continuous Education, State Research Center — Byrnasyan Federal Medical Biophysical Center of Federal Biological Agency; Leading Researcher of the Liver Transplant Department, N.V. Sklifosovsky Research Institute of Emergency Care of the Moscow City Health Department.
Contact information: vladsyutkin@gmail.com;
129010, Moscow, Bolshaya Sukharevskaya sq., 3.
ORCID: https://orcid.org/0000-0001-8391-5211

Некрасова Татьяна Петровна — кандидат медицинских наук, доцент, Институт клинической морфологии и цифровой патологии, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: nekrasova_t_p@staff.sechenov.ru;
119435, г. Москва, ул. Погодинская, 1, стр. 1.
ORCID: https://orcid.org/0000-0001-6376-9392

Никитин Игорь Геннадьевич — доктор медицинских наук, профессор, заведующий кафедрой госпитальной терапии № 2 лечебного факультета, ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации.
Контактная информация: igor.nikitin.64@mail.ru;
117513, г. Москва, ул. Островитянова, 1.
ORCID: https://orcid.org/0000-0003-1699-0881

Новиков Александр Александрович — доктор биологических наук, ведущий научный сотрудник лаборатории клинической иммунологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: a.novikov@mknc.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0002-2738-2956

Сайфутдинов Рафик Галимзянович — доктор медицинских наук, заведующий кафедрой терапии, Казанская государственная медицинская академия — филиал ФГБОУ ДПО «Российская медицинская академия непрерывного профессионального образования» Министерства здравоохранения Российской Федерации.
Контактная информация: rgsbancorp@mail.ru;
420012, г. Казань, ул. Бутлерова, 36.
ORCID: https://orcid.org/0000-0003-2839-100X

Сказываева Екатерина Васильевна — кандидат медицинских наук, доцент кафедры пропедевтики внутренних болезней, гастроэнтерологии и диетологии им. С.М. Рысса, ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации.
Контактная информация: eskazyvaeva@yandex.ru;
191015, г. Санкт-Петербург, ул. Кирочная, 41.
ORCID: https://orcid.org/0000-0002-8563-6870

Сюткин Владимир Евгеньевич — доктор медицинских наук, профессор кафедры хирургии с курсами онкохирургии, эндоскопии, хирургической патологии, клинической трансплантологии и органного донорства, Медико-биологический университет инноваций и непрерывного образования, ФГБУ «Государственный научный центр Российской Федерации — Федеральный медицинский биофизический центр им. А.И. Бурназяна»; ведущий научный сотрудник отделения трансплантации печени, ГБУЗ г. Москвы «НИИ скорой помощи им. Н.В. Склифосовского» Департамента здравоохранения г. Москвы.
Контакнтая информация: vladsyutkin@gmail.com;
129010, г. Москва, Большая Сухаревская пл., 3.
ORCID: https://orcid.org/0000-0001-8391-5211

Mariya K. Prashnova — Cand. Sci. (Med.), Teaching Assistant of the Scientific and Educational Center of Gastroenterology and Hepatology. Russian Scientific Center for Radiology and Surgical Technologies named after Academician A.M. Granov.
Contact information: prashnova@mail.ru;
199226, Saint Petersburg, Korablestroiteley str., 20.
ORCID: https://orcid.org/0000-0002-5402-8266

Прашнова Мария Константиновна — кандидат медицинских наук, доцент Научно-клинического и образовательного центра гастроэнтерологии и гепатологии. ФГБУ «Российский научный центр радиологии и хирургических технологий им. академика А.М. Гранова» Министерства здравоохранения Российской Федерации.
Контактная информация: prashnova@mail.ru;
199226, г. Санкт-Петербург, ул. Кораблестроителей, 20, корп. 1.
ORCID: https://orcid.org/0000-0002-5402-8266

Tatyana Yu. Khaymenova — Cand. Sci. (Med.), Head of the Department of Liver Diseases, Loginov Moscow Clinical Scientific Center.
Contact information: t.khaimenova@mknc.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0002-4599-4040

Хайменова Татьяна Юрьевна — кандидат медицинских наук, заведующая отделением заболеваний печени, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: t.khaimenova@mknc.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0002-4599-4040

Sergey G. Khomeriki — Dr. Sci. (Med.), Professor, Head of the Pathomorphology Laboratory, Loginov Moscow Clinical Scientific Center.
Contact information: s.homeriki@mknc.ru;
111123, Moscow, Entuziastov highway, 86.
ORCID: https://orcid.org/0000-0003-4308-8009

Хомерики Сергей Германович — доктор медицинских наук, профессор, заведующий лаборатории патоморфологии, ГБУЗ г. Москвы «Московский клинический научно-практический центр им. А.С. Логинова» Департамента здравоохранения г. Москвы.
Контактная информация: s.homeriki@mknc.ru;
111123, г. Москва, ш. Энтузиастов, 86.
ORCID: https://orcid.org/0000-0003-4308-8009

Submitted: 23.09.2024 Accepted: 01.12.2024 Published: 30.12.2024
Поступила: 23.09.2024 Принята: 01.12.2024 Опубликована: 30.12.2024