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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии
საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებშიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman (Кириллица)**, для текста на грузинском языке следует использовать **AcadNusx**. Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи.** Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - <http://www.spinesurgery.ru/files/publish.pdf> и http://www.nlm.nih.gov/bsd/uniform_requirements.html. В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректур авторам не высылаются, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or computer-printed on a single side of standard typing paper, with the left margin of 3 centimeters width, and 1.5 spacing between the lines, typeface - **Times New Roman (Cyrillic)**, print size - 12 (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf

In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

**Articles that Fail to Meet the Aforementioned
Requirements are not Assigned to be Reviewed.**

ავტორთა საყურადღებო!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე, დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx**. შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემავსებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრაფიის ფოტოსურათები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედეგების ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფხიხლებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

Tolegen A. Toleutayev, Altay A. Dyussupov, Merey N. Imanbaev, Dina M. Toleutaeyva, Nazarbek B. Omarov, Zhasulan O. Kozhakhmetov, Yernur M. Kazymov, Aldiyar E. Masalov. MODERN METHODS OF SURGICAL TREATMENT OF DIABETIC FOOT SYNDROME.....	6-10
Lipatov K.V, Asatryan A.G, Vinokurov I.A, Kazantsev A.D, Melkonyan G.G, Solov'eva E.I, Gorbacheva I.V, Sotnikov D.N, Vorotyntsev A.S, Emelyanov A.Y, Komarova E.A, Avdienko E.V, Sarkisyan I.P. SURGICAL TREATMENT STRATEGIES OF DEEP STERNAL WOUND INFECTION FOLLOWING CARDIAC SURGERY.....	11-17
Yerasyl A. Mukash, Nazarbek B. Omarov, Meyrbek Zh. Aimagambetov, Altai A. Dyussupov, Tolkin A. Bulegenov, Samatbek T. Abdrakhmanov, Medet A. Auyenov, Muratkan T. Kuderbayev, Aldiyar E. Masalov. WAYS TO IMPROVE THE RESULTS OF SURGICAL TREATMENT OF DIFFUSE TOXIC GOITER.....	18-26
Hasmik G. Galstyan, Armine V. Sargsyan, Artyom A. Sahakyan, Razmik A. Dunamalyan, Siranush A. Mkrtchyan, Ganna H. Sakanyan, Rhipsime Sh. Matevosyan, Lusine M. Danielyan, Marine A. Mardiyan. QUALITY OF LIFE IN INDIVIDUALS WITH VARYING LEVELS OF TRAIT AND STATE ANXIETY.....	27-33
Abdulmajeed Alghamdi, Hashim Abdullah Saleh Alghamdi, Adel Khaled Alghamdi, Adham Mohammed H. Alghamdi, Anmar Ali Saad Alghamdi, Abdulaziz Musaad Safir Alkhatami, Abdullah Ali Abdullah Al-Mimoni, Muhannad Essa Salem Alghamdi. PREVALENCE AND RISK FACTORS OF UROLITHIASIS AMONG THE POPULATION OF AL-BAHA REGION, SAUDI ARABIA.....	34-41
Tetiana Fartushok, Dmytro Bishchak, Iryna Bronova, Olena Barabanchyk, Yuriy Prudnikov. ANALYSIS OF CHALLENGES AND POSSIBILITIES OF USING ARTIFICIAL INTELLIGENCE IN MEDICAL DIAGNOSTICS.....	42-53
Noor N. Agha, Aisha A. Qasim, Ali R. Al-Khatib. EFFECTS OF SESAMUM INDICUM (SESAME) OIL IN REMINERALIZING OF WHITE SPOT LESIONS INDUCED AFTER BRACKET DEBONDING: AN IN VITRO STUDY.....	54-60
Kordeva S, Broshtilova V, Tchernev G. GRAHAM-LITTLE-PICCARDI-LASSEUR SYNDROME (GLPLS) IN A BULGARIAN PATIENT: CASE REPORT AND SHORT PATHOGENETIC UPDATE IN RELATION TO THE CONNECTION TO ANTIGEN/ MOLECULAR MIMICRY.....	61-67
Emad A ALwashmi, Betool R Alqefari, Sadeem S Alsenidi, Eithar O Alwasidi, Yazeed M Alhujaylan, Abdullah H Alsabhawi, Monirh M Almshigh. ASSESSMENT OF THE RELATIONSHIP BETWEEN OVERACTIVE BLADDER AND FUNCTIONAL CONSTIPATION, IN QASSIM REGION, SAUDI ARABIA.....	68-74
Yeralieva B.A, Paizova M.K, Yerkinbekova G.B, Shlymova R.O, Nurgazieva G.E, Rakhmanova G.M, Nuralim M.N. COMPARATIVE ANALYSIS OF ANTIBIOTIC CONSUMPTION IN MULTIDISCIPLINARY HOSPITALS IN ALMATY PERSPECTIVES ON AWARE AND ABC ECONOMIC ANALYSIS.....	75-77
Mohammed AH Jabarah AL-Zobaidy, Sheelan Ameer Sabri, Abdulhameed Salim Barrak, Nabaa Abdulhameed Salim, Suha Ameer Sabri. A NEW COMBINATION OF KNOWN AGENTS FOR TREATMENT OF ALOPECIA AREATA: A CASE-SERIES STUDY.....	78-82
Levytska O.V, Dubivska S. S. FEATURES OF THE POSTOPERATIVE COURSE IN PATIENTS WITH DIABETIC FOOT SYNDROME AND SYSTOLIC MYOCARDIAL DYSFUNCTION AFTER LOWER LIMB AMPUTATION.....	83-87
Knarik V. Kazaryan, Naira G. Hunanyan, Margarita H. Danielyan, Rosa G. Chibukchyan, Yulia Y. Trofimova, Arusyak V. Mkrtchyan, Kristine V. Karapetyan, Tatevik A. Piliposyan. CORRELATION BETWEEN RHYTHMOGENESIS OF THE RAT URETERS UNDER HISTAMINE EXPOSURE.....	88-94
L.Ya.Abbasova, V.A. Mirzazade, I.I. Mustafayev, N.R. Ismayilova. FEATURES OF THYROID DYSFUNCTION IN PATIENTS WITH ATRIAL FIBRILLATION	95-98
Adil Khalaf Altwairgi. CHRONIC INFECTION WITH SCHISTOSOMA HAEMATOBIIUM LEADS TO THE DEVELOPMENT OF SQUAMOUS CELL CARCINOMA OF THE BLADDER.....	99-103
Shkvarkovskiy I.V, Moskaliuk O.P, Kozlovska I.M, Kolotylo O.B, Rusak O.B. PREVENTION AND TREATMENT OF PANCREATITIS AFTER ENDOSCOPIC SURGERY ON THE BILE DUCT.....	104-107
Meruert T. Orazgalieva, Meyrbek Zh. Aimagambetov, Samatbek T. Abdrakhmanov, Nazarbek B. Omarov, Medet A. Auyenov, Merkhata N. Akkaliyev, Ainash S. Orazalina, Aldiyar E. Masalov, Daniyar S. Bokin, Julia V. Omarova Aida M. Ulbauova. METHOD FOR PREVENTION OF COAGULOPATHIC BLEEDING DURING SURGERY FOR MECHANICAL JAUNDICE.....	108-114
Munther Natheer, Mohammed Tariq, Tameem Thamir, Rami Ramadhan. NURSES' KNOWLEDGE WITH REGARD PAIN AS A PART OF A VITAL SIGNS.....	115-118

Olga Kim, Zilola Mavlyanova, Bakhridin Doniyorov, Mukhayakhon Khamdamova, Fariza Khalimova. INDIVIDUAL CHARACTERISTICS OF HIGHER NERVOUS ACTIVITY AS A FACTOR IN ADAPTATION AND RECOVERY OF THE CARDIOVASCULAR SYSTEM IN ATHLETES.....	119-124
Jingjing Liu, Anli Hu, Yulei Xie. A STUDY ON THE RELATIONSHIP BETWEEN TYPE A PERSONALITY, EMPLOYMENT STRESS, AND MENTAL HEALTH OF RESIDENT PHYSICIANS IN TERTIARY HOSPITALS IN NANCHONG, CHINA.....	125-131
Rym ben Othman, Inchirah Karmous, Ramla Mizouri, Olfa Berriche, Amina Bornaz, Ines Mannai, Faten Mahjoub, Fethi Ben Slama, Henda Jamoussi. INTERMITTENT FASTING (5:2) VS. NON-FASTING: A COMPARATIVE ANALYSIS OF ANTHROPOMETRIC PARAMETERS, DEPRESSION, AND STRESS IN HEALTHY ADULTS - A CROSS-SECTIONAL STUDY.....	132-137
Noor Mohammed Mousa, Abdull Jabar Attia, Karima Fadhil Ali. DESIGN, MOLECULAR DOCKING, MOLECULAR DYNAMICS, AND EVALUATION OF NOVEL LIGANDS TARGETING BETA-2 ADRENERGIC RECEPTOR FOR ASTHMA THERAPEUTICS.....	138-147
Kolev I, Andreev A, Zazirnyi I. ARTHROSCOPIC TREATMENT OF POSTERIOR ANKLE IMPINGEMENT SYNDROME – SYSTEMATIC SURGICAL APPROACH AND CASE REPORT.....	148-153
Rusudan Devadze, Arsen Gvenetadze, Shota Kepuladze, Giorgi Burkadze. FEATURES OF DISTRIBUTION OF INTRATUMORAL LYMPHOCYTES IN OVARIAN EPITHELIAL TUMOURS OF DIFFERENT HISTOLOGICAL TYPES AND DEGREE OF MALIGNANCY.....	154-158
Merey N. Imanbayev, Altai A. Dyussupov, Yersyn T. Sabitov, Nazarbek B. Omarov, Yernur M. Kazymov, Zhassulan O. Kozhakhmetov, Dina M. Toleutayeva, Samatbek T. Abdrakhmanov, Merkhata N. Akkalyiev, Aldiyar E. Masalov. PREVENTION OF COMPLICATIONS OF SURGICAL TREATMENT OF PATIENTS WITH OCCLUSION OF THE AORTOILIAC SEGMENT.....	159-167
Salah Eldin Omar Hussein, Awadh S Alsubhi, Ammar Abdelmola, Saadalnour Abusail Mustafa, Praveen Kumar Kandakurti, Abdulrahman Algarni, Elryah I Ali, Abdelrahman Mohamed Ahmed Abukanna, Hussam Ali Osman, Ayman Hussien Alfeel. ASSOCIATION BETWEEN GLYCATED HEMOGLOBIN AND ELEVATED THYROID HORMONES LEVELS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS.....	168-172
Sami A. Zbaar, Islam K. Kamal, Atyaf Alchalabi. ASSOCIATION BETWEEN SERUM LEVELS OF ADIPOKINES IN PATIENTS WITH PROSTATE CANCER.....	173-177
Ramazanov M.A, Bogaevskaya D.V, Sobolev D.A, Riabov A.A, Vysokikh I.S, Makhmudova A.A, Eremenko A.A, Motskobili G.G, Sadkovskaia A.I, Alibekov Gulyakhmed-haji A. IMPROVEMENT OF COGNITIVE FUNCTION IN WISTAR RATS UNDER CHRONIC STRESS CONDITIONS WITH MELATONIN.....	178-180
Olena Babkina, Svitlana Danylenko, Ihor Korobko, Vadym Zozuliak, Valerii Kucher. DIAGNOSTIC OF PANCREATIC INJURY USING INFRARED THERMOMETRY.....	181-186
Takuma Hayashi, Krishna Prasad Acharya, Sarita Phuyal, Ikuo Konishi. THE IMPORTANCE OF ONE HEALTH IN PREVENTING THE SPREAD OF HIGHLY PATHOGENIC AVIAN INFLUENZA/H5N1.....	187-189

FEATURES OF THYROID DYSFUNCTION IN PATIENTS WITH ATRIAL FIBRILLATION

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Abstract.

This study explores the prevalence and clinical significance of thyroid dysfunction in patients diagnosed with atrial fibrillation (AF). A total of 134 AF patients (72 males and 62 females) were evaluated using clinical assessments, anthropometric measurements, electrocardiography, Doppler echocardiography, thyroid ultrasound, and thyroid hormone testing (TSH, free T3, free T4, anti-TPO). Participants were grouped according to thyroid gland functional, structural, and autoimmune status.

Results indicated that 61.9% had normal thyroid function, whereas 38.1% demonstrated dysthyroidism (subclinical/overt hypothyroidism, subclinical/overt hyperthyroidism, or “pseudo-dysthyroidism”). Notably, hypothyroidism was associated with a higher frequency of severe AF symptoms (EHRA class IV). However, it did not significantly influence AF type (bradysystolic, normosystolic, tachysystolic), AF form (paroxysmal, persistent, permanent), or disease duration. Hyperthyroidism also showed no statistically significant effect on AF type, form, or duration, though there was a trend toward more severe symptoms (EHRA III–IV).

The study emphasizes the importance of “pseudo-dysthyroidism,” a condition marked by secondary alterations in thyroid hormone levels due to other comorbid illnesses. Recognizing such cases is critical to prevent unnecessary thyroid-directed interventions. Overall, the findings suggest that thyroid dysfunction—particularly hypothyroidism—may exacerbate symptom severity in AF without necessarily altering the arrhythmia’s fundamental characteristics. Comprehensive thyroid evaluation, including hormone measurements and ultrasound, is recommended for all AF patients to detect both overt and subclinical thyroid disorders and guide appropriate management.

Key words. atrial fibrillation, thyroid dysfunction, “pseudo-dysthyroidism”

Introduction.

Atrial fibrillation (AF) is regarded as the most common supraventricular arrhythmia capable of causing serious hemodynamic disturbances [1]. During AF, the atria undergo irregular activation, preventing coordinated atrial contractions [1]. The principal diagnostic criteria for AF on a 12-lead electrocardiogram (ECG) include irregular R-R intervals, absence of P waves, and evidence of irregular atrial activity [1,2].

Recent years have witnessed a rise in the incidence of AF in developed countries, attributed to population aging, the spread of obesity, improved diagnosis of cardiovascular diseases, and increased survival rates due to better treatments. Studies indicate that AF prevalence reaches approximately 3% in individuals over 20 years of age, and one in three Europeans older than 55 is at lifetime risk of developing AF [2-5].

AF elevates the risk of mortality by an average of 1.5–2 times and predisposes patients to serious complications, including stroke, dementia, myocardial infarction, sudden cardiac death,

heart failure, chronic kidney disease, and peripheral artery disease [4,6-9]. The pathogenesis of AF involves both modifiable and non-modifiable risk factors [1,2,4]. Among these, the functional status of the thyroid gland has garnered particular attention [10,11]. Even subclinical thyroid dysfunction may trigger AF [10-13].

Aim of the study.

To determine the prevalence of thyroid gland dysfunction in patients with atrial fibrillation.

Materials and Methods.

The study was conducted on 134 patients with atrial fibrillation (72 of them men and 62 women) who were both inpatients and outpatients admitted to the Republican Clinical Hospital named after acad. M. Mirgasimov. AF was verified by 12-lead ECG examination. The following examinations were performed on the patients: clinical, anthropometric (height, weight, BMI) assessments, measurement of arterial pressure, ECG, Doppler echocardiography, ultrasound examination of the thyroid gland and hormonal status (free T3, free T4, TSH, and anti-TPO in the blood). The analysis of thyroid gland hormones was carried out by the IFA (Immun Ferment Analysis) method on the Bio Screen MS-500 (USA) apparatus. It should be noted that patients with clinical and laboratory hypo- and hyperthyroidism, and those taking medications (antithyroid preparations, betablockers, etc.) that could affect the functional activity of the thyroid gland, were not included in the study. Arterial hypertension (AH) was confirmed in patients who had a blood pressure level $\geq 140/90$ mmHg at two consecutive clinical visits and who were receiving an adequate dose of antihypertensive medication. Blood pressure categories were determined based on the guidelines of the European Society of Cardiology (ESC 2024).

Type 2 diabetes mellitus (T2DM) diagnosis was confirmed according to the criteria of the American Diabetes Association (IDF 2024).

Chronic heart failure (CHF) and its functional classes were determined according to the 2016 guidelines of the European Society of Cardiology, based on ejection fraction in the echocardiogram (EchoCG) and corresponding symptoms (ESC 2021).

During the analysis of the material, at the first stage of the study, the following initial groups of patients with atrial fibrillation (n=134) were grouping by Thyroid Functional Status:

1. **Normal Thyroid Function (n=83; 61.9%)** – Normal TSH, free T4 (FT4), and free T3 (FT3) levels.

2. **Dysthyroidism (n=51; 38.1%)** – Thyroid function parameters outside the normal range.

Within the Normal Thyroid Function cohort, two subgroups were identified:

• **Ideal Normal (IN) group (n=20; 14.9%).**

No structural abnormalities on thyroid ultrasound and normal anti-TPO levels.

• **Functional Normal (FuN) group (n=63; 47.0%):** Normal TSH, FT4, and FT3 levels but accompanied by autoimmune and/or structural changes:

- **FuNA:** Functional normal with autoimmune changes.
- **FuNS:** Functional normal with structural changes.
- **FuNAS:** Functional normal with both autoimmune and structural changes.

Within the dysthyroidism cohort, the following subgroups were identified:

Hypothyroidism (HypoT) group (n=21; 15.7%): Patients with TSH, FT4, and FT3 changes suggestive of hypothyroidism (subclinical or overt).

Hyperthyroidism (HyperT) group (n=11; 8.2%): Patients with elevated thyroid hormone levels (subclinical or overt hyperthyroidism).

Pseudo-dysthyroidism (PDT) group (n=19; 14.2%): Normal TSH levels but pathological changes in FT3 and/or FT4 (consistent with Euthyroid Sick Syndrome [ESS] or Non-Thyroidal Illness Syndrome [NTIS]). A key characteristic of this “pseudo-dysthyroidism” (PDT) syndrome is that the observed thyroid-related changes are secondary and appear as an adaptive response to the patient’s primary comorbid condition [22-24].

Results.

Among the participants, 61.9% had normal thyroid function (IN and FuN groups), whereas 38.1% belonged to the dysthyroidism groups (HypoT, HyperT, PDT). These findings confirm the relatively high frequency of thyroid abnormalities in AF patients.

Comparison of hypothyroidism (HypoT) and ideal normal (IN) groups.

The HypoT and IN groups were compared with respect to AF

type (bradysystolic, normosystolic, tachysystolic), AF form (paroxysmal, persistent, permanent), and disease duration (0–1 year, 1–3 years, 3–5 years, 5–10 years, over 10 years) (Tables 1 and 2).

AF Type and form: No statistically significant difference was found. Presence of hypothyroidism did not decisively influence the brady-, normo-, or tachysystolic pattern of AF, nor did it affect paroxysmal, persistent, or permanent forms.

Symptomatic severity (EHRA): In the HypoT group, the frequency of EHRA class IV (severe symptoms) was 28.6%, whereas it was only 5.0% in the IN group ($p < 0.05$). This suggests that hypothyroidism may exacerbate clinical manifestations in AF.

Disease duration: No significant intergroup difference was noted for durations of 1 year or less, 1–3 years, 3–5 years, 5–10 years, or more than 10 years.

Comorbid conditions: Patients with hypothyroidism tended to have fewer multiple comorbidities but a higher frequency of single comorbidities. This might reflect the lower co-occurrence of other risk factors in hypothyroid patients.

Conclusion: Although hypothyroidism does not significantly affect the principal forms and types of AF, it may lead to more severe symptomatology (EHRA IV).

Comparison of hyperthyroidism (HyperT) and ideal normal (IN) groups.

These two groups were similarly compared (Tables 3 and 4).

AF type and form: No statistically significant differences emerged between hyperthyroid and IN patients in terms of

Table 1. The frequency of occurrence of AF symptomatology at various degrees of severity in the IN (n=20) and HypoT (n=21) groups.

EHRA	Group % (95% CI)		P
	IN (n=20)	HypoT (n=21)	
I	5.0 (0.00 – 14.80)	9.5 (0.00 – 22.39)	> 0.05
II	50.0 (27.52 – 72.48)	28.6 (8.77 – 48.37)	> 0.05
III	40.0 (17.97 – 62.03)	33.3 (12.67 – 53.99)	> 0.05
IV	5.0 (0.00 – 14.80)	28.6 (8.77 – 48.37)	< 0.05
I - II	55.0 (32.63 – 77.37)	38.1 (16.81 – 59.38)	> 0.05
III - IV	45.0 (22.63 – 67.37)	61.9 (40.62 – 83.19)	> 0.05

Table 2. The duration of AF in the IN (n=20) and HypoT (n=21) groups.

Duration of disease	Group % (95% CI)		P
	IN (n=20)	HypoT (n=21)	
Unknown	5.0 (0.00 – 14.80)	14.3 (0.00 – 29.62)	> 0.05
0-1 year	30.0 (9.39 – 50.61)	14.3 (0.00 – 29.62)	> 0.05
1-3 years	15.0 (0.00 – 31.06)	19.0 (1.84 – 36.26)	> 0.05
3-5 years	15.0 (0.00 – 31.06)	9.5 (0.00 – 22.39)	> 0.05
5-10 years	0.0	0.0	-
More than 10 years	35.0 (13.55 – 56.45)	42.9 (21.17 – 64.56)	> 0.05

Table 3. The frequency of occurrence of AF symptomatology at various degrees of severity in the IN (n=20) and HyperT (n=11) groups.

EHRA	Group % (95% CI)		P
	IN (n=20)	HyperT (n=11)	
I	5.0 (0.00 – 14.80)	9.1 (0.00 – 26.91)	> 0.05
II	50.0 (27.52 – 72.48)	18.2 (0.00 – 42.09)	> 0.05
III	40.0 (17.97 – 62.03)	63.6 (33.82 – 93.45)	> 0.05
IV	5.0 (0.00 – 14.80)	9.1 (0.00 – 26.91)	> 0.05
I - II	55.0 (32.63 – 77.37)	27.3 (0.00 – 54.88)	> 0.05
III - IV	45.0 (22.63 – 67.37)	72.7 (45.12 – 100.00)	> 0.05

Table 4. The duration of AF in the IN (n=20) u HyperT (n=11) groups.

Duration of disease	Group %(95% CI)		p
	IN (n=20)	HyperT (n=11)	
Unknown	5.0 (0.00 – 14.80)	9.1 (0.00 – 26.91)	> 0.05
0-1 year	30.0 (9.39 – 50.61)	18.2 (0.00 – 42.09)	> 0.05
1-3 years	15.0 (0.00 – 31.06)	45.5 (14.59 – 76.32)	> 0.05
3-5 years	15.0 (0.00 – 31.06)	18.2 (0.00 – 42.09)	> 0.05
5-10 years	0.0	0.0	-
More than 10 years	35.0 (13.55 – 56.45)	9.1 (0.00 – 26.91)	> 0.05

Table 5. Occurrence frequency of comorbid diseases in the PDT (n=11) and IN (n=20) groups.

Comorbidity	Group %(95% CI)		p
	IN (n=20)	PDT (n=11)	
Obesity	30.0 (9.39 – 50.81)	10.5 (0.00 – 24.70)	> 0.05
Type 2 diabetes mellitus	20.0 (2.01 – 37.99)	21.1 (2.22 – 39.89)	> 0.05
Arterial hypertension	55.0 (33.63 – 77.37)	68.4 (46.95 – 89.90)	> 0.05
Ischemic heart disease	35.0 (13.55 – 56.45)	47.4 (24.30 – 70.44)	> 0.05
Heart failure	80.0 (62.01 – 97.99)	68.4 (46.95 – 89.90)	> 0.05

brady-/normo-/tachysystolic AF or paroxysmal/persistent/permanent forms.

Symptomatic severity (EHRA): Although EHRA III–IV was noted in 72.7% of the HyperT group vs. 45% of the IN group, this difference did not reach statistical significance ($p > 0.05$).

Disease duration: No significant differences were observed regarding the duration of AF.

The factors creating conditions for the development of PDT during AF, based on the comparison of PDT and IN groups

Literature data shows that the development of PDT is the result of acute or chronic comorbid conditions. During AF, for the analysis of the factors creating conditions for the emergence of this syndrome, by us, a comparison of the PDT (n=19) and IN (n=20) groups was carried out.

From Table 5 it becomes evident that, in the PDT group, the occurrence frequency of comorbid diseases was as follows: obesity – 30.0 (9.39 – 50.81)%, type 2 diabetes mellitus – 20.0 (2.01 – 37.99)%, arterial hypertension – 55.0 (33.63 – 77.37)%, ischemic heart disease – 35.0 (13.55 – 56.45)%, heart failure – 80.0 (62.01 – 97.99)%/ Correspondingly, in the IN group, those indicators were as follows: obesity – 10.5 (0.00 – 24.70)%, type 2 diabetes mellitus – 21.1 (2.22 – 39.89)%, arterial hypertension – 68.4 (46.95 – 89.90)%, ischemic heart disease – 47.4 (24.30 – 70.44)%, heart failure – 68.4 (46.95 – 89.90)%. In all cases, differences between the groups did not demonstrate statistical significance.

Thus, the comparative analysis of the PDT (n=19) and IN (n=20) groups did not reveal the effect of comorbidity on the “pseudo-dysthyroidism” syndrome.

Discussion

The present study investigated the prevalence and clinical significance of thyroid dysfunction in patients with atrial fibrillation (AF), focusing on both overt and subclinical forms of hypo- and hyperthyroidism as well as “pseudo-dysthyroidism” (PDT). The results showed that 38.1% of all AF patients enrolled had thyroid function parameters outside the normal range, highlighting a notable prevalence of dysthyroidism in this population.

Consistent with previous studies indicating an association between thyroid disorders and arrhythmias (particularly AF),

38.1% of our cohort exhibited some form of thyroid dysfunction (overt or subclinical hypothyroidism, overt or subclinical hyperthyroidism, or PDT) [10–13]. Although earlier large-scale studies have documented the link between hyperthyroidism and increased AF incidence, our data suggest that hypothyroidism is also relatively common among individuals with AF and may carry its own clinical repercussions [14, 15]. Notably, 14.2% of patients demonstrated PDT, signifying that various comorbid conditions can induce alterations in thyroid hormone levels not necessarily warranting direct thyroid-targeted treatments.

A salient finding in our study is the association between hypothyroidism and increased AF symptom severity. Specifically, patients in the HypoT group exhibited a significantly higher frequency of EHRA IV symptoms compared to those with ideal thyroid function ($p < 0.05$). These data resonate with existing evidence that hypothyroidism negatively affects cardiovascular hemodynamics, leading to bradycardia, reduced cardiac output, and fluid retention [17]. Such mechanisms may exacerbate the symptom burden in AF – manifesting clinically as palpitations, fatigue, and exercise intolerance – despite the absence of significant differences in AF form, type, or duration.

Importantly, our findings that hypothyroidism did not influence the brady-, normo-, or tachysystolic patterns of AF align with prior observations suggesting that, while thyroid hormones can modulate heart rate and conduction, the presence of reduced thyroid hormone levels may not necessarily alter the fundamental electrophysiological drivers of AF. Instead, the primary effect appears to manifest through more pronounced symptoms, potentially via increased susceptibility to fluid overload, decreased myocardial contractility, and impaired vascular compliance.

Despite well-known pathophysiological links between hyperthyroidism and AF – namely, enhanced sympathetic tone, increased automaticity, and shortened atrial refractoriness – lk our study did not identify a statistically significant effect of hyperthyroidism on AF type (brady-, normo-, tachysystolic), form (paroxysmal, persistent, or permanent), or disease duration [14–16]. One possible explanation is that, while overt hyperthyroidism can trigger the onset of AF, it may

not necessarily drive the persistence or specific rate-related characteristics once AF is established. Moreover, many patients with hyperthyroidism receive beta-blockers or other rate-control medications during their clinical course, potentially mitigating differences in ventricular rate or arrhythmia patterns [16].

Nevertheless, we observed a non-significant trend toward more severe (EHRA III-IV) symptomatology in the hyperthyroid group relative to the ideal normal group, indicating that hyperthyroidism may still intensify subjective complaints such as palpitations, anxiety, and reduced exercise tolerance. This observation is in line with previous reports that hyperthyroidism often leads to hyperadrenergic states and can worsen patient-perceived quality of life [14, 15].

A notable portion of the dysthyroid cohort (14.2%) comprised patients whose thyroid function test abnormalities were attributable to non-thyroidal conditions, consistent with the concept of euthyroid sick syndrome (ESS) or non-thyroidal illness syndrome (NTIS) [18, 19]. This phenomenon underscores the complexity of interpreting thyroid function tests in patients with acute or chronic comorbidities such as heart failure, ischemic heart disease, or diabetes mellitus. While such comorbidities were common in both the PDT and ideal normal subgroups, our comparative analysis did not detect a specific factor uniquely driving PDT in AF patients. Instead, these findings suggest that multiple concurrent illnesses might collectively contribute to transient thyroid hormone alterations.

Recognizing PDT has crucial clinical implications. Misinterpreting these changes as primary thyroid dysfunction could lead to unnecessary treatments (e.g., levothyroxine or antithyroid drugs). Therefore, careful correlation of laboratory data with clinical findings and imaging (thyroid ultrasound) is paramount to rule out true thyroid pathology before initiating any therapy. This recommendation aligns with existing guidelines advocating a thorough diagnostic approach for subclinical thyroid dysfunction in complicated patients [1, 2].

Recommendations

Routine Thyroid Screening in AF: Given that nearly two in five AF patients in our study had some form of thyroid function abnormality, systematic screening with TSH, free T4, and free T3—along with anti-TPO antibodies and ultrasound in select cases—appears warranted. Early identification of thyroid dysfunction can guide more nuanced therapeutic decisions, including the need for thyroid hormone replacement or antithyroid therapy and tailored AF management (rate vs. rhythm control).

Focus on Symptom Management: As hypothyroidism correlated with more severe AF symptoms, identifying and correcting even subclinical hypothyroidism could improve patient-reported outcomes. Restoring euthyroidism may alleviate some hemodynamic burdens, enhance exercise capacity, and reduce symptomatic distress.

Avoiding Overtreatment in PDT: Clinicians should differentiate pseudo-dysthyroid patterns from genuine thyroid dysfunction to avert unnecessary interventions. Inappropriate treatment can introduce iatrogenic complications without conferring genuine benefit, emphasizing the utility of repeated hormone measurements and close clinical follow-up.

Personalized AF Care: Integrating thyroid function assessment into AF care algorithms allows for individualized therapy. Patients with hypothyroidism might benefit from more aggressive symptom control measures, whereas hyperthyroid patients may require careful rate-control strategies, especially if coexisting comorbidities contraindicate certain medications.

Conclusion

In summary, this study highlights a considerable frequency of thyroid dysfunction in AF patients and the importance of distinguishing between true thyroid disorders (hypothyroidism or hyperthyroidism) and transient, illness-related alterations (PDT). Hypothyroidism was associated with more severe AF symptomatology, suggesting that timely recognition and correction of low thyroid hormone levels may alleviate symptom burden. Although hyperthyroidism did not significantly affect AF type, form, or duration, its trend toward higher EHRA III-IV classes suggests that further investigation is warranted. Finally, “pseudo-dysthyroidism” exemplifies the complexities of interpreting thyroid hormone alterations in patients with diverse comorbidities, emphasizing the need for thorough endocrine evaluation to avoid misdiagnosis and overtreatment. Enhanced screening, combined with personalized management, can ultimately refine AF care and improve clinical outcomes.

Future investigations involving larger patient cohorts may further improve AF diagnosis and inform personalized treatment strategies. Specifically, evaluating TSH, free T4, free T3, anti-TPO, and thyroid ultrasound in all AF patients – and ruling out “pseudo-dysthyroidism” when indicated – are important steps in minimizing both AF-related complications and unnecessary risks for patients.

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