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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.

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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.

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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 საქმრალდებოლ!

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, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრაფიების ფოტოასლები 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.

Hasanov N.H, Istomin A.G, Istomin D.A. MATHEMATICAL JUSTIFICATION OF THE CHOICE OF RODS FOR EXTERNAL FIXATION DEVICES FOR POLYSTRUCTURAL PELVIC INJURIES.....	6-13
B. Todorova, I. Bitoska, A. Muca, O.Georgieva Janev, T. Milenkovic. A RARE CASE OF A PATIENT WITH HYPERTHYROIDISM AFTER HYPOTHYROIDISM.....	14-16
Satyaapir Sahu, Shabir Ahmad Shah, Supriti, Apurva Kumar R Joshi, Devanshu Patel J, Asha Yadav. THE GUT-BRAIN AXIS: IMPLICATIONS FOR NEUROLOGICAL DISORDERS, MENTAL HEALTH, AND IMMUNE FUNCTION..	17-24
Sara Mohammed Oudah Al-Saedi, Israa Hussein Hamzah. THE ROLE GENE EXPRESSION OF PD-1 AND PD-L1 IN NEWELY DIAGNOSED AND TREATED PATIENTS WITH ACUTE MYELOID LEUKEMIA.....	25-29
Stepanyan L, Lalayan G, Avetisyan A. AN INVESTIGATION OF PSYCHOLOGICAL AND PHYSIOLOGICAL FACTORS AFFECTING PERFORMANCE IN ADOLESCENT JUDOKAS.....	30-36
Takuma Hayashi, Nobuo Yaegashi, Ikuo Konishi. EFFECT OF RBD MUTATIONS IN SPIKE GLYCOPROTEIN OF SARS-COV-2 ON NEUTRALIZING IGG AFFINITY.....	37-46
Yahya Qasem Mohammed Taher, Muna Muneer Ahmed, Hakki Mohammed Majdal. A CLINICO-EPIDEMIOLOGICAL STUDY OF MULTIPLE SCLEROSIS IN MOSUL CITY, IRAQ.....	47-52
Simona Kordeva, Georgi Tchernev. THIN MELANOMA ARISING IN NEVUS SPILUS: DERMATOSURGICAL APPROACH WITH FAVOURABLE OUTCOME.....	53-55
Buthaina H. Al-Sabawi, H. S. Sadoon. HISTOCHEMICAL CHANGES OF THE PULMONARY HYDATID CYSTS IN SHEEP INFECTED WITH CYSTIC ECHINOCOCCOSIS.....	56-60
Rocco De Vitis, Marco Passiatore, Vitale Cilli, Massimo Apicella, Giuseppe Taccardo. SARS-COV-2 INFECTION AND INVOLVEMENT OF PERIPHERAL NERVOUS SYSTEM: A CASE SERIES OF CARPAL TUNNEL SYNDROME AGGRAVATION OR NEW ONSET WITH COVID-19 DISEASE AND A REVIEW OF LITERATURE.....	61-66
L. Dzyak, K. Miziakina. NEURAL PROTEINS AS MARKERS FOR DIAGNOSING STRUCTURAL DAMAGE TO BRAIN MATTER IN POST-TRAUMATIC NEUROCOGNITIVE DISORDERS.....	67-70
Hiba M. Al-Khuzayy, Yasir H. Al-Juraisy, Ali H. Alwan. PURIFICATION, CHARACTERIZATION, AND IN VITRO ANTITUMOR ACTIVITY OF A NOVEL GLUCAN FROM PHOENIX DACTYLIFERA L. FRUITS.....	71-75
Natalia Stepaniuk, Oleh Piniashko, Olesia Poshvyak, Tetiana Bessarab, Natalia Hudz, Irina Gavriluk. MANAGEMENT OF RISKS OF ADVERSE DRUG REACTIONS ACCORDING TO ADR REPORT FORM DATA FROM LVIV REGION HEALTHCARE FACILITIES IN 2022.....	76-80
Ghazwan M. Radhi, Nihad N. Hilal, Mohammed M. Abdul-Aziz. TESTOSTERONE AND SERUM ZINC LEVELS IN MEN WITH BENIGN PROSTATIC HYPERPLASIA.....	81-86
Zora Khan, Deepthi Krishna, Surya Shekhar Daga, Nitin Kumar Rastogih, Rekha MM, Komal Patel. ADVANCEMENTS IN MINIMALLY INVASIVE SURGERY: A COMPREHENSIVE ANALYSIS OF ROBOTIC SURGERY, ENDOSCOPIC TECHNIQUES, AND NATURAL ORIFICE TRANSLUMENAL ENDOSCOPIC SURGERY (NOTES).....	87-92
Aditi Jane, Manoj Rameshachandra Vyas, Anil Kumar, Anurag Verma, Giresha AS, Devanshu Patel J. LIVER FIBROSIS: PATHOPHYSIOLOGY, DIAGNOSIS, AND EMERGING THERAPEUTIC TARGETS FOR A COMMON COMPLICATION OF CHRONIC LIVER DISEASES.....	93-100
Dilip Kumar Pati, Abhishek Roy, Mayur Porwal, Beemkumar N, Geetika Patel M, Sunita Bhatt. INNOVATIONS IN ARTIFICIAL ORGANS AND TISSUE ENGINEERING: FROM 3D PRINTING TO STEM CELL THERAPY.....	101-106
Nada HA. Al-Nuaimi, Saher S. Gasgoos. EFFECT OF CHICKEN EGG SHELL PASTE ON ENAMEL SURFACE MICROHARDNESS AND COLOUR CHANGE OF ARTIFICIAL CARIOUS LESIONS CREATED ON PERMANENTLY EXTRACTED TEETH.....	107-112
Ali Sabah Abbas, Hind Taher Jarjees. EVALUATION THE EFFECT OF THE ADDITION OF ZIRCONIUM OXIDE AND TITANIUM DIOXIDE NANOPARTICLES ON SHEAR BOND STRENGTHS OF ORTHODONTIC ADHESIVE: IN-VITRO STUDY.....	113-121

Marwa H. Abdullah, Sawsan H. Aljubori. EVALUATION OF THE EFFECT OF DIFFERENT INTRAORIFICE BARRIER MATERIALS ON CORONAL MICRO LEAKAGE OF ENDODONTIC ALLY TREATED TEETH BY USING MICRO-COMPUTED TOMOGRAPHY TECHNOLOGY (A COMPARATIVE IN VITRO STUDY).....	122-130
Makhlynets NP, Prots HB, Ozhogan ZR, Pantus AV, Yatsynovych VI. PREVENTIVE PLASTIC OF BUCCAL FRENUM IN COMPLEX TREATMENT OF PATIENTS WITH ACQUIRED MAXILLOMANDIBULARANOMALIES.....	131-135
Geetika Patel M, Nidhi, Karan Ramlal Gupta, Manish Kumar Gupta, Sudhir Kumar Gupta, Krupa S. THE IMPACT OF CLIMATE CHANGE ON INFECTIOUS DISEASES: A COMPREHENSIVE ANALYSIS OF VECTOR-BORNE DISEASES, WATER-BORNE DISEASES, AND PUBLIC HEALTH STRATEGIES.....	136-142
Volodymyr Gavrysyuk, Ievgeniia Merenkova, Yaroslav Dziublyk, Galyna Gumeniuk, Mykola Gumeniuk. REFRACTORY PULMONARY SARCOIDOSIS: INCIDENCE AFTER TREATMENT WITH METHYLPREDNISOLONE AND/OR METHOTREXATE IN PATIENTS WITH NEWLY DIAGNOSED DISEASE.....	143-147
Tsvetkova M.A., Kovalenko A.Yu. ORTHODONTIC TREATMENT ALGORITHM OF PATIENTS WITH A BURDENED DRUG ANAMNESIS. DRUGS THAT REDUCE BONE MINERAL DENSITY.....	148-152
Devanshu Patel J, Aparna vikal, Vinay Kumar HK, Aejaz Ahmadh, Krishana Kumar Sharma, Asha K. THE MICROBIOME AND METABOLIC DISORDERS: THE LINK BETWEEN THE GUT MICROBIOTA AND METABOLIC SYNDROME.....	153-158
Liubov Kobak, Orest Abrahamovych, Uliana Abrahamovych, Andriy Maksymuk, Ruslana Ivanochko. DIAGNOSTIC VALUE OF LABORATORY MARKERS OF SYNTROPIC LESIONS OF THE CIRCULATORY SYSTEM ORGANS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS.....	159-164
Sriniwas Vishnu Yadkikar, Komal Patel, Renuka Jyothi R, Richard Swami, Syam Bhargavan, Sandeep Bishnoi. INNOVATIONS IN ORTHOPEDIC SURGERY: MINIMALLY INVASIVE TECHNIQUES FOR JOINT REPLACEMENT AND REPAIR.....	165-169
Kordeva S, Tchernev G, Ivanov L, Broshtilova V. "THE DANGEROUS BRASSIERE" AND THE NEVUS ASSOCIATED POLYPOID MELANOMA: CONNECTION SEEMS PLAUSIBLE?.....	170-175
Kavyn Vasyl. COMPARISON OF THE RESULTS OF STUDYING BY THE STUDENTS OF THE "CLINICAL ANATOMY AND OPERATIVE SURGERY" MODULE WITH DIFFERENT FORMS OF THE EDUCATIONAL FORMS OF THE EDUCATIONAL PROCESS IN CONDITONS OF SOCIAL SHOCKS IN UKRAINE.....	176-179
N.P. Voloshina, V.V. Vasilovsky, T.V. Negreba, V.M. Kirzhner, I.K. Voloshyn-Haponov. THE RELATIONSHIP BETWEEN THE DURATION OF REMISSIONS AFTER THE ONSET, THE SEVERITY OF THE RELAPSES AGAINST THE BACKGROUND OF DIFFERENT DURATION OF THE RELAPSING STAGE AND THE NATURE OF THE PROGNOSIS IN SECONDARY-PROGRESSIVE MULTIPLE SCLEROSIS.....	180-184
Phool Chandra, Natwar lal Vyas, Geetika Patel M, Malathi H, Radhika, Vinay Kumar HK. CARDIAC REHABILITATION: IMPROVING OUTCOMES FOR PATIENTS WITH HEART DISEASE.....	185-190
N.V. Avramenko, G.V. Bachurin, Yu.S. Kolomoets, O.A. Nikiforov. REPRESENTATION OF KIDNEY DAMAGE AT THE MOLECULAR LEVEL IN PATIENTS WITH UROLITHIASIS BASED ON THE STUDY OF ENZYMATIC TEST INDICATORS.....	191-197
Teremetskyi VI, Rusnak LM, Avramova OYe, Gorbenko AS, Kyrychenko TS. CORRELATION BETWEEN THE RIGHT TO HEALTH CARE AND THE RIGHT TO HOUSING WITHIN MEDICAL AND LAW- ENFORCEMENT PRACTICE IN TERMS OF THE COVID-19 PANDEMIC.....	198-204
Dilip Kumar Pati, Piyush Mittal, Arvind Verma, Devanshu Patel J, Asha. K, Kanika Pundir. PSORIASIS PATHOGENESIS: INSIGHTS FROM TRANSCRIPTOMICS AND PROTEOMICS STUDIES OF KERATINOCYTES....	205-211
Garashchenko O.O., Konovalenko V.F. ANALYSIS OF PLASMA MIRNA-497 LEVELS IN THE BLOOD OF PATIENTS WITH BREAST CANCER.....	212-216
Geetika Patel M, Varshini B, Anju Mandal, Deepthi Krishna, Vaibhav Rastogi, Madhumati Varma. THE ROLE OF GENETICS IN DISEASE DIAGNOSIS AND TREATMENT MITOCHONDRIAL RESPIRATORY CHAIN DYSREGULATION IN GENOMIC MEDICINE.....	217-226
Kordeva S, Broshtilova V, Batashki I, Tchernev G. BULGARIAN PATIENT WITH ATROPHODERMA OF PASINI AND PIERINI-DESCRIPTION OF A CASE AND SHORT UPDATE.....	227-231

Shypunov V.G, Strafun S.S, Borzykh A.V, Borzykh N.A, Zahovenko M.A. PECULIARITIES OF USING A NEUROVASCULARIZED FLAP ON THE SURAL ARTERY IN PLASTIC SURGERY OF GUNSHOT DEFECTS ON THE FOOT AND LOWER LEG.....	232-236
Igor Morar, Oleksandr Ivashchuk, Sergiy Ivashchuk, Volodymyr Bodiaka, Alona Antoniv. MICROBIOLOGICAL FEATURES OF A LAPAROTOMY WOUND COMPLICATED BY POSTOPERATIVE EVENTRATION AGAINST THE BACKGROUND OF AN ONCOLOGICAL PROCESS.....	237-242
Vadim V. Klimontov, Kamilla R. Mavlianova, Jilia F. Semenova, Nikolay B. Orlov. CIRCULATING PEPTIDES OF THE TNF SUPERFAMILY AND TNF RECEPTOR SUPERFAMILY IN SUBJECTS WITH TYPE 1 DIABETES: RELATIONSHIPS WITH CLINICAL AND METABOLIC PARAMETERS.....	243-248
Rurua Magda, Sanikidze Tamar, Machvariani Ketevan, Pachkoria Elene, Ormotsadze Gorge, Intskirveli Nino, Mikadze Ia, Didbaridze Tamar, Ratiani Levan. CORRELATIVE ASSOCIATION OF OXYGENATION AND SEPSIS PANELS WITH THE USE OF ACE2 INHIBITORS AND WITHOUT IT IN THE CONDITIONS OF SEPTIC SHOCK IN COVID-19-INFECTED AND NON-INFECTED PATIENTS (COHORT STUDY).....	249-253
Vladyslava Kachkovska. ASSOCIATION BETWEEN GLN27GLU POLYMORPHISM IN THE B2 ADRENERGIC RECEPTOR GENE AND OBESITY RISK IN PATIENTS WITH EARLY-ONSET AND LATE-ONSET BRONCHIAL ASTHMA.....	254-258
Lazarenko H.O, Lazarenko O.M, Shaprinskyi V.V, Semenenko N.V. INFLUENCE OF VASCULAR STENT SURFACE TREATMENT WITH AN ADAPTIVE COMPOSITION (ADC) FOR IMPROVING ITS BIOCOMPATIBILITY AND RESTENOSIS PREVENTION.....	259-263
Duve K.V. THE PREVALENCE OF C3953T IL1B GENE AND G308A TNFA GENE POLYMORPHIC VARIANTS IN THE PATIENTS WITH DIFFERENT TYPES OF ENCEPHALOPATHIES.....	264-269
Levandovskiy R, Belikova N, Belikov O, Sorokchan M, Roschuk O. EVALUATION OF THE CLINICAL CONDITION OF THE ORAL CAVITY BEFORE ADHESIVE SPLINTING OF MOVABLE TEE TH.....	270-274
Bakhtiyarov Kamil Rafaelevich, Ivantsova Margarita Vladimirovna, Kukes Ilya Vladimirovich, Ignatko Irina Vladimirovna, Glagovsky Pavel Borisovich. METABOLOMIC MARKERS OF ENDOMETRIOSIS: PROSPECTS.....	275-279
Jain SK, Komal Patel, Kavina Ganapathy, Firoz Khan, Satyaapir Sahu, Ashok Kumar Singh. LAPAROSCOPIC APPROACH TO A GIANT RUPTURED SPLENIC CYST: A CHALLENGING CASE REPORT.....	280-283
ManojRameshachandra Vyas, Phool Chandra, Rachit Jain, Devanshu Patel J, Manashree Avinash Mane, Shaily. CLINICAL AND OBJECTIVE TEST CHARACTERISTICS OF VESTIBULAR MIGRAINE: IMPLICATIONS FOR DIAGNOSIS AND MANAGEMENT.....	284-289
Vipin Kumar, Rakesh Ashokrao Bhongade, Vipin Kumar, Praveen Mathur, Komal Patel, Renuka Jyothi R. POSTCHOLECYSTECTOMY SYNDROME: UNDERSTANDING THE CAUSES AND DEVELOPING TREATMENT STRATEGIES FOR PERSISTENT BILIARY SYMPTOMS AFTER GALLBLADDER REMOVAL.....	290-296
Georgi Tchernev. LOSS OF EFFICACY OF ADALIMUMAB IN HIDRADENITIS SUPPURATIVA: FOCUS ON ALTERNATIVES.....	297-300

NEURAL PROTEINS AS MARKERS FOR DIAGNOSING STRUCTURAL DAMAGE TO BRAIN MATTER IN POST-TRAUMATIC NEUROCOGNITIVE DISORDERS

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Abstract.

Objectives: To determine the role of neural markers for brain matter damage in cognitive dysfunction after severe traumatic brain injury.

Materials and Methods: A comprehensive study included clinical and laboratory examination, neuropsychological testing, MRI. To identify markers of structural changes in brain substance, neural proteins were identified: glial fibrillary acidic protein and neural cell adhesion molecule. Neural proteins quantification was performed using an enzyme-linked immunosorbent assay.

280 patients with severe traumatic brain injuries and moderate neurocognitive disorders (MND) (DSM-V) were examined and divided into two groups according to the pathogenetic mechanisms and neuropsychological profiles. The first group included subjects with MNDs of a primary dysmnesic type (73 persons), the second group presented MNDs of a neurodynamic-dysregulatory type (207 persons). The follow-up period was 6 months, 1 year and 3 years.

Results: By the third year, progression of cognitive disorders in the first group was detected in 5% of cases, in the second group - in 10% of cases.

Conclusion: The revealed NP dynamic disturbances and their quantitative assessment in clinical groups during the examination allowed specifying structural and functional brain changes to characterize mechanisms of neurocognitive disorder development in traumatic brain injuries.

The findings have demonstrated neural proteins can be considered as markers for not only structural, but also neurodynamic processes.

Key words. Severe traumatic brain injury, neurocognitive disorders, neural proteins, GFAP, NCAM.

Introduction.

Treatment and rehabilitation of patients with traumatic brain injury (TBI) is one of the urgent problems of public health. The overall frequency of BI in European countries is 262 hospitalizations per 100,000 people per year [1]. At the same time, severe TBI accounts for only about 10% of all cases which lead to the most serious consequences: early death in 39% of cases and poor outcomes in 60% of cases [2].

It should be noted that traumatic brain injury is also a serious medical problem in Ukraine. Thus, the prevalence of this pathology is 1.99–3.0 cases per 1000 adults per year [3].

It has been established that the causes determining the disability status in this category of patients include disorders of higher mental functions [4–6].

Besides, it has been found that pathognomonic nature of psychopathological consequences does not depend on the injury severity, intensity of neurological disorders, and the structural

brain damage pattern and can be formed both in the acute, intermediate, and long-term periods of BI. It has been shown that more than 35% of patients with mild brain injury subsequently develop cognitive impairments of varying severity. After a severe injury, this number is significantly higher [7]. It has been established that cognitive dysfunctions are formed in 68.4% of patients with past severe closed brain injury. At the same time, 3–10% of patients with a history of severe BI develop dementia [8–10].

In this regard, the mechanisms of cognitive processes in persons with a history of severe BI is an urgent subject to investigate.

Having stated that, there is a particular interest and importance of searching markers for structural and functional changes in the brain involved in this process in people with a history of severe BIs.

Studies have shown that neural proteins (NPs) of neuronal, glial, and microglial origin are general markers reflecting the objective state of the brain [11].

It is known that the neural cell adhesion molecule (NCAM) is involved in neuron-neuron interaction, regulates synaptic plasticity, and forms neuronal connections. Glial fibrillary acidic protein (GFAP), a specific marker of astrocytes involved in formation of glial filaments, is involved in molecular mechanisms of neuron-astrocytic interactions. It is known that one of the functions of NCAM and GFAP is implementation of neurocognitive processes. The current data about the role of NCAM and GFAP as prognostic markers for developing neurocognitive deficit are rather contradictory [12].

Objectives.

To determine the role of neural markers for brain matter damage in cognitive dysfunction after severe traumatic brain injury.

Materials and Methods.

This study is based on the findings of a comprehensive clinical and neuropsychological examination of 310 patients with severe traumatic brain injuries (210 men and 100 women) aged from 20 to 55 years. 280 patients were diagnosed with moderate neurocognitive disorders (MND). Neurocognitive deficit was assessed according to DSM–V criteria (2013) [13]. To assess heterogeneity of the identified neurocognitive disorders, the following scales were used: Mini-mental state examination (MMSE) (Folstein M. et al. 1975); Montreal Cognition Assessment (MoCA) (Nasreddine Z. et al. 2005); Frontal Assessment Battery (Dubois B. et al. 1999); Clock drawing test (Sunderland T. et al. 1989); Digit Spans (Wechsler D. 1945); character-digital test (Smith A. 1982), Shulte Table (Bleicher V. M. et al. 2002); Trail Making Test (Reitan, 1958); 10-item test (S. Ya. Rubinshtein, 1970); 10-item test (A.R. Luriya, 1976);

test of "verbal associations" (Borkowski J. et al. 1967).

All patients signed informed consent to participate in the study. The exclusion criteria: somatic, neurological pathologies leading to cognitive impairment, mental illnesses; therapy with psychotropic drugs with a proven cognitive-stimulating effect in the period of 6 months before the study; pregnancy and lactation; neoplasms of any localizations; a past history infectious diseases leading to cognitive deficits; age under 20 and over 55 years old. The control group included people aged from 20 to 55 years old.

Quantitation of immunological markers for development of structural and functional brain disorders (MANK and GFAP) was performed using an enzyme-linked immunosorbent assay including inhibition of antibodies by antigen in blood serum [14].

The follow-up period was 6 months, 1 year and 3 years.

The Cramer-Welch (T) criterion is based on the approach of assessing the equality of mathematical expectations of the general populations from which the samples are taken. The statistics of the criterion is as follows:

$$T = \frac{\sqrt{n_1 n_2} (\bar{X}_1 - \bar{X}_2)}{\sqrt{n_1 s_1^2 + n_2 s_2^2}}$$

Unknown variances are replaced by their sample estimates (s_1^2 and s_2^2), n_1 n_2 are sample sizes, \bar{X}_1 and \bar{X}_2 - are sample means. As the sample sizes increase, the distribution of the Cramer-Welch T statistic converges to the standard normal distribution with a mathematical expectation of 0 and a variance of 1. In respect to the asymptotic normality of the T statistic, the decision rule for the Cramer-Welch test is as follows:

if $|T_{em}| < z(1-\alpha/2)$, then

The null hypothesis H_0 , a hypothesis of homogeneity (equal mathematical expectations), is accepted at the significance level α .

The study used a significance level of 0.05. The value of the Cramer-Welch statistics module $T = 3.8$ for "Changes in the concentration of NCAM protein (mkg/ml) in blood serum of patients in clinical groups at the follow-up stages depending on the type of GSU syndrome in 6 months" **was compared with the critical value $Z_{kr}=1.96$** . Since $T > Z_{kr}$ ($3.8 > 1.96$), the null hypothesis H_0 is rejected at the significance level of 0.05, so there are significant differences between groups 1 and 2.

Results.

When evaluating the findings of the comprehensive clinical and neuropsychological examination, cognitive deficits were identified in 90.3% of patients with a history of STBI (Table 1).

The analysis of cognitive indicators in subjects showed heterogeneity of the clinical signs of cognitive disorders. The overall assessment of their characteristics was consistent with the generally accepted diagnostic criteria for moderate neurocognitive disorder according to DSM-V. The semiotic analysis of the impaired cognitive functions clarified the cognitive profile of patients and distinguished two types of moderate neurocognitive disorders: polyfunctional MND without primary memory impairments and polyfunctional MND with primary memory impairments.

Table 1. Characteristics of STBI Subjects of Comprehensive Clinical and Neuropsychological Examination.

Examination Group	Patient Number	%
Patients with no post-traumatic neurocognitive disorders	30	9,7
Patients with post-traumatic neurocognitive disorders	280	90,3

Table 2. Subjects in Clinical Groups according to MND Type.

Clinical Group	MND Type	Patient Number	%
Group I	polyfunctional MND with primary memory impairments	73	26
Group II	polyfunctional MND with no primary memory impairments	207	74

Based on the data obtained, the subjects were divided into 2 clinical groups (Table 2).

The detected clinical heterogeneity of neurocognitive deficit allowed for distinguishing two types of neurocognitive disorder development: primary dysmnesic and neurodynamic-dysregulatory.

When assessing blood serum NP level in the subjects, the results were compared with the indicators characterizing neural protein concentrations (NCAM and GFAP) in blood serum of practically healthy individuals with 90-100% correct answers during cognitive tests and no pathology (20 persons) and patients with a history of STBI and no neurocognitive disorders (30 persons).

There were no significant differences in NCAM, GFAP proteins content in patients with no post-traumatic cognitive disorders against the controls at the follow-up points.

But when studying content evolution of immunological markers characterizing the structural and functional state of the brain (NCAM and GFAP) in patients with post-traumatic neurocognitive disorders in clinical groups, heterogeneous indicators were detected depending on the type of neurocognitive deficit (Figures 1 and 2).

It has been established that traumatic brain injuries induce pathobiochemical reactions that occur in the main cell pools of nervous tissue and cause neuronal disorders involving astrocytes and neuroglia.

When studying concentrations of neural proteins (NCAM and GFAP) in Clinical group I, a sharp increase in GFAP, a marker of astrocytes, was detected in blood serum at all follow-up stages. While the increased concentrations of NCAM involved in regulation of synaptic plasticity and formation of neural networks is observed at the initial follow-up points (6 months, 1 year), and then significant decrease is demonstrated (2 years, 3 years).

When studying concentrations of neural proteins (NCAM and GFAP) in Clinical group II, a significant increase in NCAM concentrations was detected throughout the entire study period, and a significant decrease in this indicator was detected at the 3rd year of follow-up. When studying evolution of GFAP concentrations in this clinical group, a significant uniform

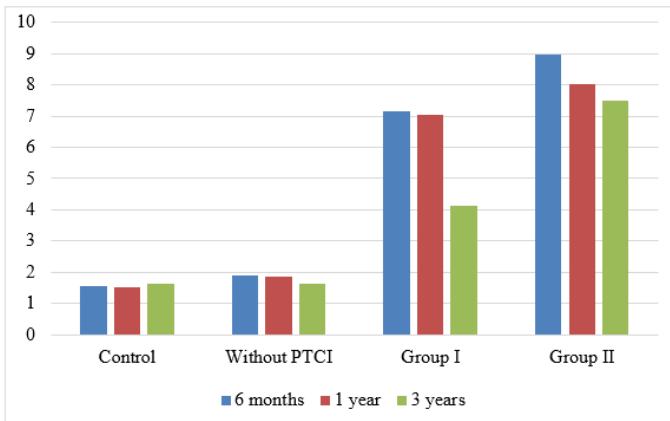


Figure 1. Changes in Blood Serum NCAM Concentrations (mkg/ml) in Clinical Groups at Follow-up Points.

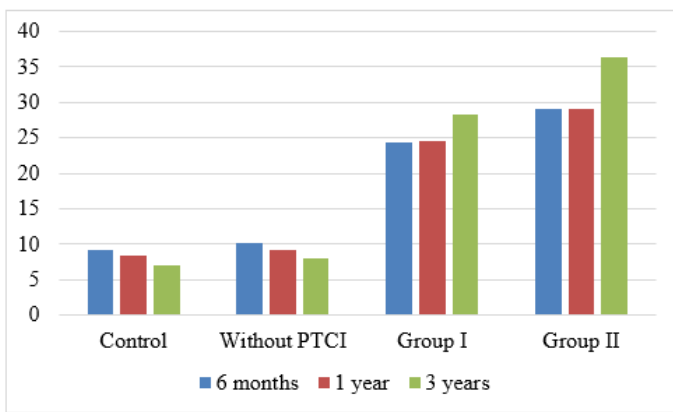


Figure 2. Changes in Blood Serum GFAP Concentrations (ng/ml) in Clinical Groups at Follow-up Points.

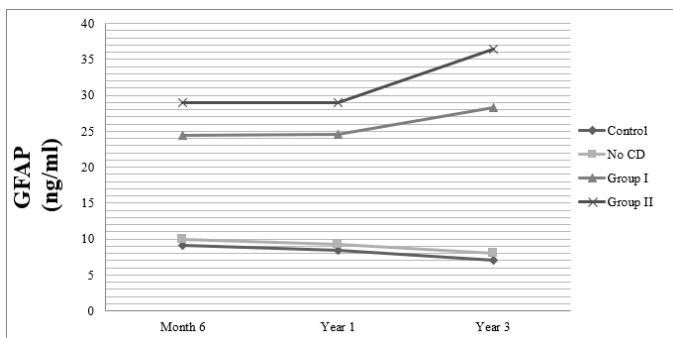


Figure 3. Changes in Blood Serum GFAP Concentrations in Patients with a History of STBI at Follow-up Points in Clinical Groups.

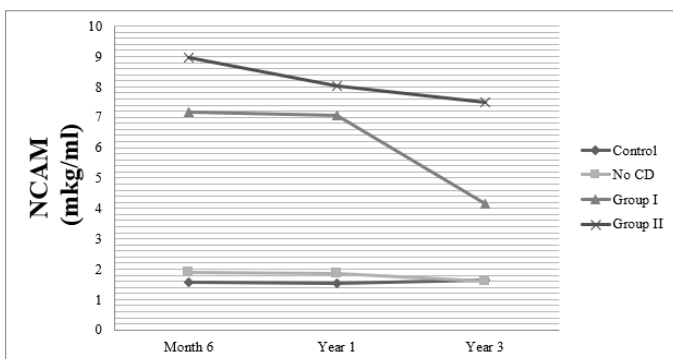


Figure 4. Changes in Blood Serum NCAM Concentrations in Patients with a History of STBI at Follow-up Points in Clinical Groups.

increase in the studied indicator was detected at all follow-up points.

Evolution of blood serum GFAP concentrations in patients after STBI at the follow-up points in clinical groups is shown in Figure 3.

The increased concentration of serum NCAM localized in the neuronal pool of cells was accompanied by progression of cognitive impairment in patients. The established positive correlation between NCAM expression and severity of memory impairment may indicate NCAM involvement in plastic rearrangements of neural patterns associated with formation of cognitive function. Taking into account the significant content of NCAM in synaptic contact zones, it should be recognized that NCAM is involved in the structural and molecular rearrangements of synapses and in intensity changes of synaptic transmission, modification of the synapses morphology.

Evolution of blood serum NCAM concentrations in patients after STBI at the follow-up points in clinical groups is shown in Figure 4.

The detected shifts in blood serum GFAP content was characterized by the sharp increase in the level of the marker of astrocytic cells which perform trophic functions in relation to neurons in patients with post-traumatic moderate cognitive disorders with MND of a polyfunctional type both with and without memory impairments. The high level of this indicator has been observed within 6 months after the injury and significantly increases at all follow-up points. This may indicate the intensive processes of plastic restructuring of synaptic connections as early as 6 months after a severe brain injury. The increase in GFAP expression at further follow-up points is consistent with the current data that astroglial cells are closely associated with synaptic structures of neurons and have receptors responding to neurotransmitter stimulation.

Besides, the increased level of blood serum GFAP confirms the concept that astrocytes having this protein as a structural component, are among the cellular elements of the blood-brain barrier thereby participating in dysregulation of its permeability, starting from the very early stages of the disease.

Thus, the use of neural proteins (NCAM and GFAP) to clarify the structural and functional characteristics of the brain which lead to development of cognitive impairment in patients with severe traumatic brain injury, can present diagnostic "markers" of damage and "indicators" of neurocognitive disorders both for primary dysmnesic and for neurodynamic-dysregulatory types of neurocognitive disorders.

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რეზიუმე

კვლევის მიზანი. დაზუსტდეს ნეიროსპეციფიური ცილების (ნერვული უჯრედების ადგეზიის მოლეკულა (ნუამ) და გლიალური ფიბრილალური მჟავე ცილა (გფმც)) როლი ნეიროკოგნიტიური დარღვევების ფორმირების მექანიზმებში იმ პირებთან, რომელთაც გადაიტანეს აქვთ ქალა-ტვინის მძიმე ტრავმა.

მასალები და მეთოდები. კვლევაში ჩართული იყო 310 ავადმყოფი, რომელთაც გადაიტანეს ქალა-ტვინის მძიმე ტრავმა. მათგან 280 პაციენტი პოსტრავმატული ნეიროკოგნიტიური დარღვევებით, 30 კი პოსტრავმატული ნეიროკოგნიტიური დარღვევების გარეშე. კონტროლის ჯგუფი შედგებოდა 20 კაცისგან. ყველა პაციენტს ჩატარდა კომპლექსური კლინიკურ-ნეიროფსიქოლოგიური გამოკვლევა, სისხლის შრატის ბიოქიმიური კვლევა. დაკვირვების პერიოდი შეადგენდა 6 თვეს, 1, 2, 3 წელს.

შედეგები. იმუნოლოგიური მარკერების შინაარსის დინამიკის შესწავლისას, რომელიც ახასიათებს თავის ტვინის სტრუქტურულ-ფუნქციონალურ მდგომარეობას (ნუამ და გფმც) პოსტრავმატული ნეიროკოგნიტიური დარღვევების მქონე პირების კლინიკურ ჯგუფებში, გამოვლენილი იყო ჰეტეროგენული მაჩვენებლები ნეიროკოგნიტიური დეფიციტის ტიპისდა მიხედვით.

დასკვნა. ჩატარებულმა კლინიკურმა მონიტორინგმა შესაძლებლობა მოგვცა დადგენილიყო ნსც-ის (ნუამ და გფმც) დონის ცვლილებების დინამიკა გამოსაკვლევ ჯგუფებში და შედარებულიყო პათოფიზიოლოგიური ცვლილებების ხასიათი, გამოვლენა და მიმართულება, რაც განაპირობებს ნეიროკოგნიტიური დარღვევების ფორმირებასა და პროგრესირებას პირებში, რომელთაც გადაიტანეს ქალა-ტვინის მძიმე ტრავმა.

საკვანძო სიტყვები: მძიმე ქალა-ტვინის ტრავმა, ნეიროკოგნიტიური დარღვევები, ნეიროსპეციფიური ცილები, ნუამ და გფმც.