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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 ВИНИТИ 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 ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

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. Корректура авторам не высылается, 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 compu-ter-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 საყურადღებოდ!

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. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის 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 ფჩხილებში 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.

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PSYCHOTHERAPEUTICAL FEATURES OF PERSONS WITH MULTIPLE SCLEROSIS AND HEPATOCEREBRAL DEGENERATION

Voloshyn-Gaponov I.K[1,2], Lantukh I.V[1], Mikhanovska N.G[3,4], Gulbs O.A[5], Malieieva O.V[6], Dikhtiarenko S.Yu[5], Kobets O.V[5], Malieiev D.V[6].

[1]*Kharkiv Institute of Interregional Academy of Personnel Management, Kharkiv, Ukraine.*

[2]*Institute of Neurology, Psychiatry, and Narcology under the National Academy of Medical Sciences of Ukraine, Kharkiv, Ukraine.*

[3]*V. N. Karazin Kharkiv National University, Kharkiv, Ukraine.*

[4]*State Institution "Institute of Children and Adolescent Health Care Academy of Medical Sciences of Ukraine", Kharkiv, Ukraine.*

[5]*Pavlo Tychyna Uman State Pedagogical University, Uman, Ukraine.*

[6]*Donetsk Regional Institute of Postgraduate Pedagogical Education, Kramatorsk, Ukraine.*

Abstract.

Physiological process of aging causes a slight deterioration in memorization, learning, and the speed of cognitive processes. There is often a big gap between established standards, norms that work for mass and individual cases, for which standards are no more than guidelines, not prescriptions and dementia could be caused by many factors, the most important of which are degenerative, vascular, and toxic. Multiple sclerosis (MS) is the commonest non-traumatic disabling disease to affect young adults. Wilson's disease (WD) is an uncommon hereditary disorder caused by a deficiency in the ATP7B transporter. All of the above realized in the aim of our work: performing comprehensive clinical and laboratory examination of patients with multiple sclerosis and patients with Wilson's disease in order to study the problem of neurodegenerative diseases and their prevention.

Materials and methods: The "Mini Mental Status Exam" scale was used for the screening assessment of the state of cognitive functions and the study of the level of intellectual productivity of patients. To determine the verbal memory, the method: "memorizing 10 words" was used, and the Derogatis SCl-90-P method was used to study the personality and emotional sphere. Psychodiagnostic examination was performed for 111 patients with MS and 33 patients with WD.

Results: Cognitive disorders were found in less than half of the patients (44.9%) in young patients with multiple sclerosis, whereas in the older age group, almost all patients (91.2%) had various cognitive disorders. Violation of verbal memory, of varying degrees of severity, was found in 69.1% of patients with multiple sclerosis.

More than half of the patients with Wilson's disease (66.7%) had impaired productivity of cognitive functions of various degrees of severity. Identified violations were noted in 27.3% of patients (10 patients), among them 7 were aged 40 years or older and had not received pathogenetic treatment for a long time. Violations of mental functions of various nature and severity were found in 78.8% patients with Wilson's disease. They are characterized by dynamic and voluntary-regulatory thinking disorders, such as exhaustion of mental capacity (88.0%), a decrease in its speed characteristics (39.4%) and general productivity, as well as inertia (48.5 %), impulsiveness (27.3%) of mental processes. Decrease in criticality of thinking was noted in 33.3% of patients.

Conclusions: The structures that take participation in cognitive functions of the brain are always involved (sooner or later) in the pathogenetic process in patients with multiple sclerosis and hepatocerebral degeneration, that leads to the development of their defects. Therefore, for the treatment of these patients, a comprehensive, pathogenetically justified and personal fixed therapy is necessary. The development of palliative therapy, which will prevent the transformation of cognitive disorders into dementia, may become a prospect for further research.

Key words. Dementia, Alzheimer's disease, cognitive impairment, hepatocerebral degeneration, multiple sclerosis, psychology, prevention.

Introduction.

The most recent data indicate that, by 2050, the prevalence of dementia will double in Europe and triple worldwide, and that estimate is 3 times higher when based on a biological (rather than clinical) definition of Alzheimer's disease [1]. However, there is an increase in dementias of various genesis all over the world, which is largely related to the aging of the global population, as well as adverse environmental factors and pathogenesis could be different for variable etiological factor for multiple sclerosis and hepatocerebral degeneration as example [2,3] and several years of pandemic covid realized in inconsolable prognosis [4,5] with data about influence of virus for cerebral tissue and organism [6-8]. Dementia also occurs at a young, working age, which makes it not only a medical problem, but also a very important social problem. Although dementia has been described in ancient texts over many centuries (e.g., "Be kind to your father, even if his mind fails him." - Old Testament: Sirach 3:12), our knowledge of its underlying causes is little more than a century old. Alzheimer published his now famous case study only 110 years ago, and our modern understanding of the disease that bears his name, and its neuropsychological consequences, really only began to accelerate in the 1980s. Since then, we have witnessed an explosion of basic and translational research into the causes, characterizations, and possible treatments for Alzheimer's disease (AD) and other dementias [9]. Dementia is an acquired generalized impairment of the patient's intellect, memory, and personality, but without a violation of his consciousness. The most complete definition of dementia or "frailty" is given by the Royal College of Physicians (Royal College of Physicians, 1982), which states that dementia is an acquired global impairment of all higher

cortical functions of the brain, including memory, ability to solve problems of everyday life, performing acquired complex actions, correct application of social skills, all aspects of speech and communication, control of emotional reactions in the absence of gross loss of consciousness. This condition almost always progresses but is not always irreversible.

Clinicians can diagnose the syndromes of dementia (major neurocognitive disorder) and mild cognitive impairment (mild neurocognitive disorder) based on history, examination, and appropriate objective assessments, using standard criteria such as DSM-5. They can then diagnose the etiological subtypes of these syndromes using standard criteria for each of them. Brain imaging and biomarkers are gaining ground for the differential diagnoses among the different disorders. Treatments for the most part are still symptomatic [10].

However, in people who do not suffer from diseases that affect the central nervous system, age-related changes in cognitive functions are insignificant and their impact on everyday life activity and social contacts is insignificant. Physiological process of aging causes only a slight deterioration in memorization, learning, and the speed of cognitive processes. There is often a big gap between established standards, norms that work for mass and individual cases, for which standards are no more than guidelines, not prescriptions. After all, with any pathology, biological rather than calendar age is more important, because aging is a heterochronic process. As we said above, dementia is caused by many factors, the most important of which are degenerative, vascular, and toxic [10].

Multiple sclerosis (MS) is the commonest non-traumatic disabling disease to affect young adults. The incidence of MS is increasing worldwide, together with the socioeconomic impact of the disease. The underlying cause of MS and mechanisms behind this increase remain opaque, although complex gene-environment interactions almost certainly play a significant role. The epidemiology of MS indicates that low serum levels of vitamin D, smoking, childhood obesity and infection with the Epstein-Barr virus are likely to play a role in disease development [11].

Wilson's disease (WD) (hepatocerebral degeneration) is an uncommon hereditary disorder caused by a deficiency in the ATP7B transporter. The protein codified by this gene facilitates the incorporation of the copper into ceruloplasmin [12]. Cognitive impairment is common in neurological presentations of Wilson's disease (WD). Various domains can be affected, and subclinical deficits have been reported in patients with hepatic presentations. Associations with imaging abnormalities have not been systematically tested [3]. Our previous experience proves necessity of psychological management of patients with cognitive disorder [13]. Multiple sclerosis and Wilson's disease are conditions which could be manifested with cognitive disorder, both are relatively often in psychological care, both have unclear development of mental dysfunction. All of the above realized in the aim of our work: performing comprehensive clinical and laboratory examination of patients with multiple sclerosis and patients with hepatocerebral degeneration in order to study the problem of neurodegenerative diseases and their prevention.

Materials and Methods.

The "Mini Mental Status Exam" (MMSE) scale was used for the screening assessment of the state of cognitive functions and the study of the level of intellectual productivity of patients [14], Wisconsin Card Sorting Test-Perseverative Response Score (WCST-PR) was performed also. The study was performed in accordance with the principles of the Helsinki Declaration of the World Medical Association "Ethical Principles of Medical Research Concerning Human Subjects" (2013) with written informed consent. To determine the verbal memory, the method: "memorizing 10 words" was used, and the Derogatis SCl-90-P method was used to study the personality and emotional sphere. Psychodiagnostic examination was performed for 111 patients with MS and 33 patients with WD. Among patients with MS, 72 people were aged from 19 to 45 years and 39 - from 45 to 60 years old. The duration of the disease ranged from 2 months to 21 years. The age of the patients with WD ranged from 17 to 55 years and averaged 30.06±7.53 years. The average age of disease onset was 23.71±7.52 years.

Statistical processing of the data was performed using the Statistica for Windows 8.0 software package [13]. Methods of descriptive statistics (determination of numerical characteristics of variables - arithmetic mean (M), mean sampling error (m), determination of the reliability of differences (p), which were tested via the Student-Fisher t-test in representative samples) were used. Correlation between indicators was assessed using Spearman's correlation coefficient (r). The difference in values between comparative indicators was considered significant at $p < 0.05$.

Results.

Performed Mini Mental Status Examination and Wisconsin Card Sorting Test-Perseverative Response Score were characterised by reduced scores in patients with Multiple sclerosis and Wilson's disease (Table 1).

Table 1. *Results of mental status examination and neuropsychological testing.*

Method examination	Multiple sclerosis		Wilson's are disease	
	M	**SD**	**M**	**SD**
MMSE	26.38	3.42	29.12	4.11
WCST-PR	22.17	13.01	27.02	6.86

As the analysis of the obtained data shows, cognitive disorders were found in less than half of the patients (44.9%) in young MS patients. Whereas in the older age group, almost all patients (91.2%) had various cognitive disorders. The level of intellectual productivity in some patients of the older age group decreased below 20 points (the norm is 30 points). At the same time, it should be noted that not only the age of the patient affected the decline of cognitive functions, but also the duration of his illness.

Violation of verbal memory, of varying degrees of severity, was found in 69.1% of MS patients. In patients with a remitting type of the course of the disease, mild disorders dominated, and in patients with a progressive type of the course, pronounced memory disorders dominated. The frequency and severity of memory disorders also increased with the age of the patients.

This is especially evident in patients with a remitting type of the course of the disease.

MS patients are also characterized by such personality changes as rigidity, inertia, difficulty in making decisions, anxiety, and depression. With age and the duration of the disease, these personal disorders intensify.

Thus, MS patients are characterized by cognitive impairments of different nature and severity. The level of general intellectual productivity is within the range from normative indicators to very pronounced systemic disorders of cognitive functions. With the age of the patients and the duration of the disease, the expression of these disorders increases.

A comprehensive, including psychodiagnostic, examination was conducted in 33 patients with WD. The results of the study showed that more than half of the patients (66.7%) had impaired productivity of cognitive functions of various degrees of severity. Identified violations (less than 20-24 points) were noted in 27.3% of patients (10 patients), among them 7 were aged 40 years or older and had not received pathogenetic treatment for a long time.

Also, the results of the research of verbal and visual memory showed that patients with CVC are characterized by a narrowing of the volume and a decrease in the strength of voluntary memorization.

Violations of mental functions of various nature and severity were found in 78.8% of WD. They are characterized by dynamic and voluntary-regulatory thinking disorders, such as exhaustion of mental capacity (88.0%), a decrease in its speed characteristics (39.4%) and general productivity, as well as inertia (48.5 %), impulsiveness (27.3%) of mental processes. In 33.3% of patients, a decrease in criticality of thinking was noted.

Thus, it should be noted that for patients with WD, the most diverse in structure and by the degree of expression of the disturbance of higher mental activity. However, it should also be noted that the severity of these violations could largely be due to their large motor and speech disorders, i.e., executive branch defects.

In order to clarify the pathogenesis of dementing processes in the brain in patients with WD and MS, we used the classic form of dementia - Alzheimer's disease (AD) - as the basis of comparison.

Discussion.

The comparative analysis showed that the instrumentally determined markers in Alzheimer's disease are to one degree or another pronounced in patients with WD and MS. A comprehensive study of various parameters and factors affecting the state of cerebral blood circulation showed that patients with WD have both structural and functional disorders in the system of ensuring cerebral hemodynamics. They develop a syndrome of early vascular aging. In that relation, necessary to remind about participation of microcirculatory disorders in various pathological processes [15,16] and reducing of cellular density in brain connected with it in elderly person [17].

Last decade is characterized significant success in development of medical technology that allows to obtain more precise diagnosis [18,19] in different branches of medicine [20]. Currently, in addition to classical cognitive impairments, the generally recognized markers of dementia development in patients with AD also include such markers that are determined instrumentally. These are atrophy of the medial parts of the temporal lobe of the brain, disturbances in the structure of sleep, an increase in the content of tau protein in the cerebrospinal fluid and blood plasma, vascular and metabolic disorders of the brain, and disorders of functional intersystemic cerebral connections. According to magnetic resonance imaging (MRI), patients with bronchial asthma develop a kind of multi-system atrophy of the brain. It should also be noted that studies in recent years show that tau protein can be not only a morphological equivalent of the death of cerebral neurons, but also participate to a large extent in the pathogenesis of AD.

Disruption of metabolic processes in the structures of the brain in patients with WD is evidenced by MR spectroscopic studies based on the data of a neuronal marker (NAA) and a marker of cell membranes (Cho). We found pathological metabolic changes both in the gray matter of the subcortical nodes and in the white matter of the cerebral hemispheres. Also, in these patients, a significant increase in tau protein, a glutamate mediator, and a decrease in the level of the neurotransmitter Gamma-aminobutyric acid (GABA) were found. That data correlate with hepar injury of other etiology [21] and pathological process in organism under influence of harmful substances [22,23].

Data from instrumental examination methods show that, in addition to foci of demyelination in various structures of the brain ("black holes"), patients with MS also have subcortical and cortical atrophy of the brain. The study made it possible to come to the conclusion that the atrophic process intensifies with increasing age and duration of the disease.

Various parameters of cerebral hemodynamics (endothelial dysfunction, thickening of the intima-media, decreased blood flow velocity) indicate that patients with MS have chronic insufficiency of cerebral circulation. A significant increase in plasma tau protein and a significant heterochromatization, i.e., a decrease functional activity of chromatin of cell nuclei.

Thus, MRI data, hemodynamic parameters, a high content of tau protein in the blood plasma, a high content of heterochromatin in the nuclei of cells, the state of the cognitive and emotional and personal spheres give reason to assume that in MS patients there is an accelerated process of aging of cells and everything organism in general [2,11].

As our researches have shown, in disorders of cognitive functions in patients, a disorder of night sleep plays an important role. After all, high-quality restorative sleep is an indicator of well-balanced, rhythmic functioning of the brain. Sleep is considered as one of the forms of adaptation of the body to the environment. Systems that support the full functioning of the sleep-wake cycle are structurally and functionally interconnected with systems that provide an optimal level of intellectual-mnestic functions and emotional response.

Thus, the sleep disturbances noted by us in all patients with elements of dementia may be an indicator of dysfunction of both somnogenic and cognitive structures of the brain.

Our quantitative computer EEG study showed that in patients with severe cognitive impairment, there is a violation of

functional intersystem cerebral connections, both cortically organized, i.e. cortical-thalamic stem, and horizontally oriented, that is, interhemispheric- transcallosal.

Thus, we can see that patients with WD and MS actually have almost all generally recognized markers of dementia development in patients with AD. A distinctive feature is only the time and severity of cognitive impairments and their transformation into dementia.

In our opinion, the time of appearance of cognitive disorders and the speed of their transformation into dementia depends on three groups of factors: genetic predisposition, natural (biological) aging, and endo- and exogenous factors of pathogenic influence on the brain. An example of a synergistic interaction of all three factors can be Alzheimer's disease, in which cognitive impairments are quickly transformed into dementia and the patient becomes almost completely dependent on others.

In Wilson disease and multiple sclerosis, a synergistic interaction of natural aging factors and the influence of endo- and exogenous pathogenic factors on the brain can be assumed. In these patients, the time and speed of dementia development depends on the nature and duration of the effect of the pathogenic factor on the brain. At the same time, as in people even with a large calendar age, but with a relatively slow current biological age, the physiological aging process proceeds with a gradual and slight deterioration of cognitive functions.

Therefore, on the basis of the data we received, as well as the data of the scientific literature, we can state that dementia is an organic pathophysiological syndrome of the destruction of a critical mass of structural and functional blocks and systems of cognitive mechanisms of the brain. Each individual has his own genetically determined critical mass of cognitive mechanisms. Like any faulty system, this one is ultimately prone to both natural (slow) decay and pathological (accelerated) decay due to the death of neurons, both by the type of apoptosis and by the type of necrosis. There is a growing discrepancy between the function, on the one hand, and its structural support, on the other. The time and speed of this imbalance depends both on the size of the critical mass and the size of the zone of its reserve capabilities, determined by lifestyle and education, and on various endogenous and exogenous factors of influence.

Thus, dementia depends not only, and perhaps not so much, on age, but on other factors that lead to the degeneration of brain structures. Therefore, treatment of dementia must be carried out at its early stage, at the stage of cognitive impairment. The common principles of management of patients with cognitive disorder (CD) are the determination of the etiopathogenetic cause underlying the development of cognitive disorder, the reduction of the degree and prevention of the progression of cognitive deficits, and the impact, if possible, on risk factors. Also, at all stages of cognitive impairment, treatment of concomitant somatic diseases and correction of emotional state are relevant.

However, there are certain difficulties in early diagnosis and correction of these disorders. On the one hand, patients and those around them believe that these are age-related changes and, therefore, not curable. On the other hand, a kind of closed cause- and-effect circle results. After all, a defect in cognitive functions reduces the possibility of realizing this defect.

Speaking about cognitive disorders in patients with WD and MS, in which neurodystrophic processes are primarily caused by genetic factors, it is necessary to apply standard treatment of these diseases with a personalized approach. Therefore, the doctor needs to pay attention not only to motor disorders, which the patient complains about, but also to non-motor cognitive disorders, which, to a lesser extent, disable patients. In this regard, compliance is very important - the patient's consent to the complex treatment proposed by the doctor, taking into account intellectual and cognitive disorders, which the patient is often not aware of.

Thus, patients with WD and MS throughout their lives, in addition to the specific pathogenetic therapy of these diseases, depending on the clinical picture and the data of laboratory studies, should carry out at least 1-2 times a year course therapy with the use of neurotrophic and vasoactive character. Cerebrolysin is an effective neurotrophic agent. Cerebrolysin is a drug with proven effectiveness in the treatment of cognitive disorders (level of evidence B). Numerous studies have proven that cerebrolysin has neurotrophic effects that are similar to the effects of endogenous neurotrophic factors [24]. As vasoactive drugs, the following are recommended: phosphodiesterase inhibitors - pentoxifylline, vinpocetine and alpha 2-adrenoceptor blocker - sermion (nicergoline). The latter improves not only cerebral hemodynamics, but also normalizes the exchange of neurotransmitters. To improve general metabolism in nervous tissue and antihypoxic activity, gopanthenic acid, piracetam, meldonium drugs are used. Also, in the treatment of cognitive disorders, the drug Memantine is regularly used (level of evidence A), which contributes to the improvement of motor functions [25].

Cognitive disorders are often combined with emotional disorders of the anxiety-depressive range. The relationship between cognitive and emotional disorders can be both causal and parallel in nature. It is known that severe depression can contribute to the development of cognitive disorders. On the other hand, the dementing process is often accompanied by anxiety-depressive disorders as a reaction to the disease with relatively preserved criticism. In some cases, cognitive and emotional disorders are not connected by causal relationships but are united by a common psychophysiological substrate. The presence of emotional disorders certainly requires pharmacological correction. However, antidepressants with a cholinolytic effect should be avoided [26,27].

In order to prevent the transformation of cognitive disorders into dementia, it is necessary to normalize sleep as well [28,29]. Of the drugs that affect the structure of the sleep-wake cycle, preference should be given to melatonin drugs, since barbiturates and benzodiazepines cause addiction and increase the risk of apnea, which increases hypoxia and accelerates the development of CD, and monoamines have a number of side effects. which limit their use in patients with WD and MS. To restore the reserve capabilities of the brain, a non-medicinal approach is also recommended, namely regular cognitive stimulation (cognitive training and rational physical exercises)

[30,31]. The patient should also be actively involved in therapy, which includes creative therapy (musical, artistic, aromatic), various activities, comprehensive communication, physical activity (walking, physical work, dancing). Wholesome and varied nutrition, moderate physical activity, social activity, mental work, and hobbies will also prevent the transformation of cognitive disorders into dementia. Preventively, in order to protect oneself from the disease, the patient should pay special attention to the level of cholesterol and arterial hypertension, which are the two most significant risk factors for the development of the disease.

Both classical and modern methods of investigation could be useful in detection of pathogenesis described pathology [32,33] with development of imaging research [34] and especially different parts of scull investigation [35-38]. Simultaneously there are data that maintaining a good lifestyle helps prevent dementia. In particular, good eating habits, both in terms of types of food and eating routines, are essential to avoid the development of dementia [39] with unclear influence of harmful habits [40] that require additional research.

Conclusion.

The structures that take participation in cognitive functions of the brain are always involved (sooner or later) in the pathogenetic process in patients with multiple sclerosis and hepatocerebral degeneration, that leads to the development of their defects. Cognitive disorders were found in less than half of the patients (44.9%) in young patients with multiple sclerosis, whereas in the older age group, almost all patients (91.2%) had various cognitive disorders. Violation of verbal memory, of varying degrees of severity, was found in 69.1% of patients with multiple sclerosis.

More than half of the patients with Wilson's disease (66.7%) had impaired productivity of cognitive functions of various degrees of severity. Identified violations were noted in 27.3% of patients (10 patients), among them 7 were aged 40 years or older and had not received pathogenetic treatment for a long time. Violations of mental functions of various nature and severity were found in 78.8% patients with Wilson's disease. They are characterized by dynamic and voluntary-regulatory thinking disorders, such as exhaustion of mental capacity (88.0%), a decrease in its speed characteristics (39.4%) and general productivity, as well as inertia (48.5 %), impulsiveness (27.3%) of mental processes. Decrease in criticality of thinking was noted in 33.3% of patients.

Therefore, for the treatment of these patients, a comprehensive, pathogenetically justified and personal fixed therapy is necessary. The development of palliative therapy, which will prevent the transformation of cognitive disorders into dementia, may become a prospect for further research.

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Conflict of interest statement.

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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