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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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STROKE AS A LIFE-THREATENING COMPLICATION IN CHILDREN WITH LINEAR SCLERODERMA OF FACE

Osminina M.K., Podchernyaeva N.S., Khachatryan L.G., Shpitonkova O.V., Velikoretskaya M.D, Chebysheva S. N., Polyanskaya A.V., Gugueva E. A.

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Abstract.

Objective: To investigate the spectrum of neurological disorders in children with juvenile localized scleroderma (JLS) on face and JLS without plaques on face and head.

Materials and Methods: 156 children with JLS were examined were with a neurological examination MRI, EEG, genetic thrombophilia markers detection.

Results: Neurological disorders (ND) were found in 56 from 114 (49%) of the patients with scleroderma of head and face (LSH)(group1) and in 30% (13 from 42) with JLS without plaques on face (Group 2). Headaches were detected in Group 1 in 43,8%, in Group 2-in30%. In Group 1 other disorders were detected: epileptic seizures (in 25%), cranial nerve involvement (12.5%), stroke (5.3%), tics (5.3%), none of named in Group 2. Out of 56 children with ND changes on brain magnetic resonance imaging (MRI) were detected in 47 (84%). white matter lesions and less commonly gliotic changes in the periventricular area. Electroencephalography (EEG) monitoring revealed typical patterns of epileptiform activity in the majority of cases.

These three cases of ischemic stroke in children with LSH are the first to be presented. All patients with stroke were diagnosed with genetic thrombophilia, two had cerebral vascular malformations, and one had antiphospholipid syndrome.

Conclusion: Our data suggest that sclerodermic vasculopathy, cerebral vascular anomalies and genetic thrombophilia are rick factors for stroke in children with LSH. Mandatory MRI angiography and screening for genetic thrombophilia could identify risk group for stroke in children with LSH.

Key words. Juvenile localized scleroderma of head and face, juvenile localized scleroderma without plaques on face, neurological disorders, headaches, epileptic seizures, cranial nerve involvement, ischemic stroke, MRI and EEG changes, genetic thrombophilia, brain vessels malformation.

Introduction.

Pediatric stroke is a rare condition, but it could cause death and disability in children. Stroke in childhood is distinguished from perinatal stroke, defined as stroke before 29 days of age, because of its pathogenesis reflecting the maternal-fetal unit. In children incidence of stroke has been reported as 2,5 per 100000 both in Rochester [1]. The worldwide incidence of stroke in young adults (aged from 15 to 44 years) is estimated to 9-11 per 100000 people [2]. The Canadian Pediatric Stroke Register (N=681) found that among infants and children with ischemic stroke 69% died or had a neurologic deficit [3].

According to Mario Mastrangelo, the incidence of perinatal stroke is 5-13 per 100,000 live births, 0.38 per 100,000 per year in children under 5 years of age and 0.48-0.6/100,000 per year in adolescence [4]. Recent studies reported a mortality rate after childhood stroke between 2,6 and 5 % [5,6]. Post stroke seizures are more common in children in comparison to adults, with an incidence of 30% at 10 years [3].

The most common causes of strokes in children are non-atherosclerotic arteriopathies, heart disease, disorders in the haemostasis [7-9]. Non-atherosclerotic arteriopathies may develop due to structural abnormalities of cerebral blood vessels, as a result of infection, post varicella arteriopathy or cerebral vascular malformation, fibromuscular dysplasia, sickle-cell disease [10].

Prothrombotic conditions are highly associated with recurrent episodes of stroke. [11]. Numerous abnormalities in blood coagulation system (genetic thrombophilia-GT) are described, among them- protein C deficiency, protein S deficiency, factor V Leiden mutation (G1691A), prothrombin mutations (PT G 20210A), antithrombin III deficiency, increased factor VII, plasminogen activator inhibitor-1 4G/5G polymorphism. Antiphospholipid antibodies and elevated homocysteine are also associated with stroke [12,13]. An association of the thermolabile *MTHFR C677T* genotype with stroke is controversial in both adults and children [13,14]. The presence of a prothrombotic abnormality has also been shown to increase the risk of recurrent stroke in children [11].

Additionally, collagen vascular diseases with central nervous system vasculitis are reported as risk factors for stroke [15-18].

Linear scleroderma of head and face (LSH), also known as ‘sabre blow’ scleroderma, and Parry-Romberg syndrome are the most severe variants of juvenile localized scleroderma. LSH results in deforming lesions of soft tissues of the face and bones of the facial skeleton, hemiatrophy of the jaws and dental apparatus, and often neurologic disorders (ND).

In a large multicentre study of localized scleroderma (750 children), nervous system involvement in LSH was observed in 18.5% of children [19]. Cephalgias and seizures account for 40-60% of all neurological disorders [20,21].

The development of hemiplegic migraine, cranial nerve damage, neuropsychiatric disorders, partial neurological deficit, and Rassmusen's encephalitis have been also described in LSH [22,23].

ND in LSH are accompanied by changes on brain MRI. In a study [24] among 21 children with LSH, MRI changes were detected in 19% of cases. Hyperintense signals from fluid in the brain tissue on the side of the skin lesion are more often detected [25,26].

Purpose of the study.

To investigate the spectrum of neurological disorders in children with juvenile localized scleroderma (JLS) localized on face and head and JLS without plaques on face and head.

Patients and Methods.

156 children with juvenile localized scleroderma (JLS)were examined at the University Children's Clinical Hospital) of Moscow Sechenov University between 2009 and 2024 year.

The diagnosis of JLS was established according to the preliminary classification criteria of localized scleroderma in children JLS [27].

Clinical evaluation of somatic, neurological and psychological status of patients was carried out.

Instrumental methods of investigation were performed: MRI of the brain on a Siemens 'Magnetom Verio' with a magnetic field of 3 Tesla, EEG in mono/bipolar leads with electrode placement according to the international system '10-20' on an 'Encephalan-131-03' device.

Results.

We examined 114 patients with LSH (group I), among them 46(40%) boys and 68 (59.6%) girls and 42 children with linear and plaque localized scleroderma (29 girls and 13 boys)-group II. The age of the patients ranged from 3 to 16 years. The mean age of disease debut was 6.64±0.53 years.

Neurological examination revealed ND in half of the patients with LSH (n=56; 49%) and in 13 (30%) with JLS without plaques on face. Among patients in group I, the disease debut was characterised by typical skin changes in 106 children (92,9%) and neurological signs in 8 (7,1%). In Group II all patients manifested with skin lesions.

The most frequent neurological signs were headaches, epileptic seizures, crainial nerve disorders, tics and acute cerebral circulatory failure (stroke).

Children with LSH had ND more frequently than children with JLS without plaque on face and head, more over among the latter we did not diagnose epileptic seizures, crainial nerve disorders, tics, hearing loss, and stroke.

Headaches were the most frequent (43.8%). The majority of patients had tension cephalgia, the genesis of which remains unclear. Epileptic seizures were the second most frequent, occurring in 33 patients (25%), with 11 patients having repeated seizures over a number of years and generalised seizures, and three patients having seizures preceding cutaneous manifestations of the disease. EEG monitoring revealed typical pattern of epileptiform activity in the majority of cases. Focal lesions of cranial nerves were observed in 14 patients (12,5%), including *n. facialis, n. trigeminis*, eyelid ptosis. In all cases, the changes occurred more than three months after the cutaneous debut.

Out of 56 children with ND changes on brain MRI were detected in 47 (84%). Foci in the white matter were more frequent (63%), glial changes in the periventricular region were less frequent (31%). Three children were verified to have changes characteristic of the consequences of stroke.

The obtained data on the nature and frequency of ND coincide with the available data in the literature, with the exception of cases of stroke. Numerous publications demonstrate the frequent development of epileptic attacks, cranial nerve disorders, headaches, and foci in the white matter of brain in patients with LSF, both children and adults. Cases of stroke are also repeatedly named in literature in adult patient with systemic sclerosis, with a link between cerebral autoimmune scleroderma microangiopathy and high frequency of stroke. In contracts to this we have met only two descriptions in the literature on stroke in LSH. Cases are devoted to the stroke in 73- and 37-year-old persons with LSH, the latter had malformation of brain vessels. The authors see a pathogenetic connection between scleroderma and stroke.

We would like to describe for the first time three cases of strokes, including recurrent strokes, in children with LSH.

Detailed description of cases is summarized in Table 2.

The clinical manifestations of transient ischemic attacks (TIA) and stroke in children were movement limitations, paretic gait, dyscoordination, dysarthria, and epileptic seizures.

In one patient the disease started with a skin process, in two cases it debuted with stroke combined with epileptic seizures a year or more before the facial skin lesions (Figure 1), later symptomatic focal epilepsy was formed.

In all three cases, the nature of the stroke was ischaemic. Analysis of cases revealed that two patients (S. M. and F. E.) had vascular malformation of brain vessels (VMBV) detected on MRI in vascular mode. In patient F.E., signs of arteriovenous malformation of the right posterior cerebral and right middle cerebral arteries were established. In the acute period, an ischaemic focus with haemorrhagic component was detected in the area of the subcortical nuclei of the right hemisphere (Figure 2). Later, multiple foci of gliosis were formed in this area.

Patient S. M. showed a picture of posterior trifurcation of the left internal carotid artery. In patients with VMBV episodes of TIA were repeated and occurred long before the appearance of scleroderma skin lesions. In a patient (S. S.) with a single stroke without VMBV, the disease debuted with a skin syndrome. An MRI scan one year before stroke revealed focal changes in the white matter of the left hemisphere. There were no epileptic seizures, but EEG revealed epileptiform activity in the frontal temporal region of the left hemisphere. Markers of antiphospholipid syndrome were also detected: antibodies to cardiolipin and lupus anticoagulant.

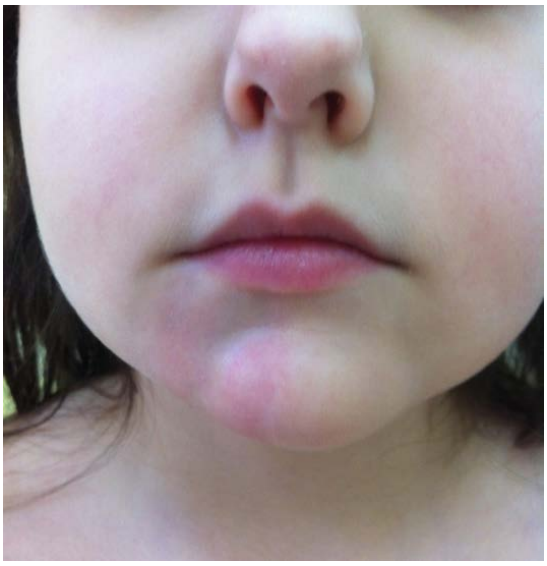

Figure 1. *Scleroderma skin lessions- erithema, fibrosis in the middle of plaque.*

Table 1. *Structure of neurological disorders in LSH children.*

Neurological disorders	**Children with LSH (group I) and neurological disorders (n=56)**	**Children with juvenile linear and plaque scleroderma (group II) and neurological disorders (n=13)**
Headaches	69 (43,8%)	4 (30%)
Epileptic seizures	33 (25%)	-
Crainial nerve disorders	14(12,5%)	-
Tics	3 (5,3%)	-
Stroke	3 (5,3%)	-
Hearing loss	1 (1%)	-
Parasomnias and insomnias	3 (5,3 %)	2 (15,4%)
Astheno-neurotic syndrome	3 (5,3%)	5 (38%)
Attention deficit and hyperactivity disorder	2 (3,5%)	2(15,4)

Table 2. *Characteristics of patients with stroke.*

	Patient S.S.	**Patient S.M.**	**Patient F.E.**
Sex, age at the time of stroke	Boy,10 years old	Boy,7 years old	Girl,6 years old
Initial clinical manifestations	Cutaneous syndrome	Stroke	Stroke
Localization of stroke and scalp lesions	Coincides	Coincides	Coincides
Number of strokes / transient ischaemic attack	1	1\13	1\5
Seizures	no	yes	yes
Neurological deficit	Right-sided hemiparesis	Alteration syndrome: nasolabial fold asymmetry on the left and hemiplegia of the right hand.	Left-sided hemiparesis
Genetic thrombophilia	Identified	Identified	Identified
Antiphospholipid syndrome	Identified	no	no
MRI of the brain	Gliosis foci in the white matter of the frontal and parietal lobes periventricularly with detection of gliosis and cavitation areas in the left thalamus and basal nuclei.	Foci of 2-6 mm merging with each other in the white matter of the brain periventricularly in the area of the anterior horn and body of the left lateral ventricle and convexitally.	Multiple foci of gliosis of the white matter of the right hemisphere, foci with marked haemorrhagic impregnation in the area of the head of the caudate nucleus.
Vascular malformation of brain vessels	No	Posterior trifurcation of the left internal carotid artery	Arterio-venous malformation of the right posterior cerebral artery and right middle cerebral artery
EEG	Epileptiform activity in the frontal temporal, central part of the left hemisphere	Epileptiform activity in frontal-parietal region of the left hemisphere	Dysfunction of diencephalic brain structures

It is noteworthy that brain ischaemic lesions in all cases were detected on the side of the cutaneous lesion, in two cases coinciding with side of abnormalities vascular structure.

Since it is known that the factor predisposing to stroke is hereditary thrombophilia, we examined patients for mutations in blood clotting genes, data are summarized in Table 3.

Patient S.S. has a homozygous mutation in the PAI-I gene, heterozygous mutations in the folate cycle genes *MTR, MTRR, MTHFR*, fibrinogen beta, integrin alpha-2. Patient S. M. had heterozygous mutations in the genes of coagulation factors VII and XII, integrin alpha-2, *MTRR*; patient F. E. had a homozygous mutation in the gene of platelet glycoprotein 1B, heterozygous mutations in the genes of coagulation factor XII, *PAI-1, MTR, MTRR, MTHFR*, integrin alpha-2 (Table 3). Multiple heterozygous mutations in genes encoding folate cycle enzymes, heterozygous mutation in the gene of integrin alpha-2 were detected in all cases. Homo- and heterozygous mutation of PAI-1 was detected in two children.

Results and Discussion.

In the whole group of 114 patients with LSH ND were detected in 56 (49%), in comparison to 30% (13 from 42) in children with JLS without plaque on face and head. ND in LSH were severe, including epileptic seizures, crainial nerve disorders, tics, hearing loss, and stroke. The most frequent ND were headaches (43%), epileptic seizures (25%) and focal lesions on brain MRI on the side of skin damage (84%). Similar results were obtained in a meta-analysis in 2013 [28] and according to a number of original studies in adult and children with LSH [19,28-30].

In adults with LSH, there is a case report of ischemic stroke (IS) and recurrent haemorrhagic strokes. An IS with left-sided hemiparesis was described in a 73-year-old woman with LSH [30], the focus was located in the basin of the right middle

Table 3. *Mutations in blood clotting genes in patients with ischemic stroke.*

Factor/mutations in gene	**Patient S.S.**	**Patient S.M.**	**Patient F.E.**
Prothrombin (coagulation factor II) (F II c *96 G > A)	GG	GG	GG
Factor V Leiden (F V *Arg506Gin* G > A)	GG	GG	GG
Coagulation factor VII (F V *Arg353Gin* G > A	GG	***GA***	GG
Coagulation factor XII (F XII c.-4 C > T)	CC	***CT***	***CT***
Coagulation factor (XIII *Val35Leu* G > T)	GG	GG	GG
Fibrinogen beta (FGB beta s.-455 G > A)	***GA***	GG	GG
Platelet glycoprotein 1 B (GPIBA c.-5 T > C)	TT	TT	TT
Platelet glycoprotein 1 B (GPIBA *Thr145Met T* > C)	TT	TT	***CC***
Integrin Alpha-2 (*ITGA2(GPIA)807* C >T	CT	***CT***	***CT***
Platelet fibrinogen receptor (*ITGB3(GPIIIa)Leu59Pro* T > C)	TT	TT	TT
Methylenetetrahydrofolate reductase (*MTHFR* Ala222Val C > T)	***CT***	CC	***CT***
Methionine synthase (*MTR* Asp919Gly A > G)	***AG***	***AG***	***AG***
Methionine synthase reductase (*MTRR* IIe22Met A > G	***AG***	***AG***	***AG***
Plasminogen activation inhibitor (*PAI-I* (SERPINE)-675 5G > 4G)	***4G/4G***	5G/5G	***5G /4G***
P-selectin glycoprotein ligand (*SELPLG* Met6211c G > A)	GG	GG	GG
Janus kinase 2 (JAK 2 Val617Phe G > T)	GG	GG	GG

Note: *Genes with mutation are highlighted in bold.*

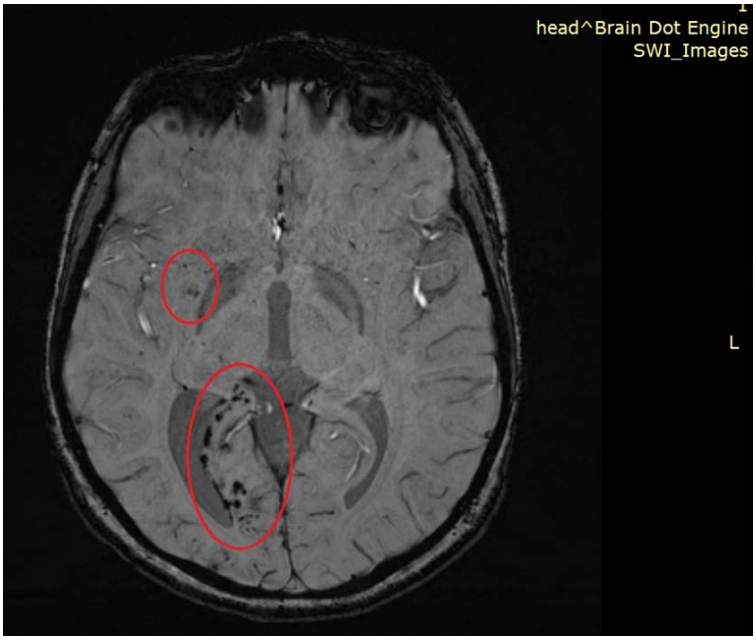

Figure 2. *Brain MRI. Foci of haemorrhagic impregnation.*

cerebral artery, where stenosis was detected. The authors attributed the development of AI to cerebral vascular sclerosis due to scleroderma. However, it is known that vascular sclerosis in old age can be caused by atherosclerotic processes.

The second observation presents episodes of recurrent haemorrhagic strokes in a 39-year-old man with LSH [31]. The authors see a pathogenetic link between stroke and scleroderma. The third case is devoted to the stroke in adult with scleroderma plaques on the trunk [32].

No cases of stroke in children with hemiscleroderma of face have been described in the literature to date.

Despite numerous descriptions of ND in LSH, it's pathogenesis is not completely clear. Two main concepts are presented in the literature. The first one is vascular. It is believed that vasculopathy, endotheliopathy, sclerotic changes of brain vessels and neuroglia are due to changes within the framework of scleroderma. Autopsy data from patients with LSH indicate inflammatory infiltration of both brain parenchyma and vessel walls in histological studies of the brain [33]. In small calibre vessels, signs of vasculitis, glial changes as a sign of chronic inflammation, leptomeningeal ribbon-like sclerosis, and thickening of brain vessel walls are detected [34,35]. The second hypothesis of pathogenesis is related to the fact that facial tissues and the underlying brain parenchyma have a single cellular precursor, the early damage of which on one side of the rostral neural tube may subsequently cause unilateral lesions [36].

According to our data, focal changes in the brain tissue and localization of stroke foci were noted on the side of skin lesions, which reflects vascular lesions within scleroderma and is consistent with the data of other authors [15,16].

It is important to note that two out of three patients with stroke had VMBV, which aggravated scleroderma vasculopathy. The identified mutations in blood coagulation system (GT) were predisposing factors for stroke. Another provoking factor is immunological and inflammatory activity of scleroderma, which reduces the antithrombotic potential of the endothelium, increasing the risk of thrombosis.

Conclusion.

The results of the study revealed nervous system involvement in half of the patients with LSH, similar to existing data in the literature. These three cases of ischemic stroke in children with LSH are the first to be presented. We speculate that the causes of stroke in patients have a multifactorial nature due to a combination of brain vascular anomaly, mutations in blood coagulation genes leading to hypercoagulability and scleroderma vasculopathy. Frequency of GT in patients with LSH is not described in current literature and is still a subject for investigation. We consider that coincidence of three predisposing factors (GT, VMBV and scleroderma vaculopathy) in presented cases led to IS. VMBV is well known risk factor of stroke, additionally probably unilateral brain vessel anomaly predisposes to hemitype of scleroderma lesions on head, due to inborn anomalies of rostral neural tube. Ischemic origin of stroke to our opinion is caused by the course of ischaemic vasculopathy of cerebral vessels due to scleroderma.

Life-threatening complications of stroke in children make it advisable to perform mandatory MR angiography to detect possible VMBV and screening for genetic thrombophilia. This will allow timely prescription of basic therapy for LSG to prevent ischemic attack and control vasculitis activity, as well as replacement therapy with folic acid and B vitamins.

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Резюме статьи

Инсульт как жизнеугрожающее осложнение склеродермии лица у детей.

Цель исследования: Изучить спектр неврологических нарушений у детей с ювенильной локализованной склеродермией (ЮЛС), локализованной на лице и голове, и ЮЛС без бляшек на лице и голове. **Материалы и методы:** Обследовано 156 детей с ЮЛС с проведением неврологического осмотра, МРТ, ЭЭГ, определением генетических маркеров тромбофилии. **Результаты:** Неврологические нарушения были выявлены у 56 из 114 (49%) пациентов со склеродермией на лице (группа1) у 30% (13 из 42) без бляшек на лице (группа 2). Головные боли выявлены в 1-й группе в 43,8%, во 2-й группе - в 30%. В 1-й группе были выявлены другие нарушения: эпилептические припадки (в 25%), поражение черепных нервов (12,5%), инсульт (5,3%), тики (5,3%), во 2-й группе - ни одного из названных. Из 56 детей с неврологическими нарушениями изменения на МРТ головного мозга были выявлены у 47 (84%) ввиде очагового поражение белого вещества и реже глиозные изменений в перивентрикулярной области. ЭЭГ-мониторинг выявил типичный паттерн эпилептиформной активности в большинстве случаев. Впервые представлены три случая ишемического инсульта у детей с ЮЛС лица. У всех пациентов с инсультом была диагностирована генетическая тромбофилия, у двух - мальформации сосудов головного мозга, у одного - антифосфолипидный синдром. **Выводы:** Наши данные свидетельствуют о том, что склеродермическая васкулопатия, церебральные сосудистые аномалии и генетическая тромбофилия являются факторами риска развития инсульта у детей с гемисклеродермией лица. Обязательная МР-ангиография и скрининг на генетическую тромбофилию могут выявить группу риска развития инсульта у детей с гемисклеродермией лица.
Ключевые слова: ювенильная локализованная склеродермия лица, ювенильная локализованная склеродермия без бляшек на лице, неврологические нарушения, головные боли, эпилептические припадки, поражение черепных нервов, ишемический инсульт, изменения МРТ и ЭЭГ, генетическая тромбофилия, мальформация сосудов головного мозга.

ინსულტი, როგორც სიცოცხლისათვის საშიში გართულებაბავშვებშისახისხაზოვანისკლეროდერმიით ობიექტური. კვლევის მიზანი: გამოიკვლიოს

ნევროლოგიური დარღვევების სპექტრი ბავშვებში არასრულწლოვანთა ლოკალიზებული სკლეროდერმიით სახეზე და არასრულწლოვანთა ლოკალიზებული სკლეროდერმიით სახეზე და თავზე ნადების გარეშე.

მასალები და მეთოდები. გამოკვლეული იქნა არასრულწლოვანთა ლოკალიზებული სკლეროდერმიით დაავადებული 156 ბავშვი ნევროლოგიური გამოკვლევით MRI, EEG, გენეტიკური თრომბოფილიის მარკერების გამოვლენით.

შედეგები.. ნევროლოგიური დარღვევები აღმოჩნდა 56-ში 114 (49%) პაციენტში სახეზე სკლეროდერმიით (ჯგუფი 1) და 30%-ში (13 42-დან) JLS-ით სახეზე დაფების გარეშე (ჯგუფი 2). თავის ტკივილი გამოვლინდა 1 ჯგუფში 43,8%-ში, 2 ჯგუფში - 30%-ში. 1 ჯგუფში გამოვლინდა სხვა დარღვევები: ეპილეფსიური კრუნჩხვები (25%), კრანიალური ნერვის ჩართვა (12.5%), ინსულტი (5.3%), ტიკები (5.3%), არცერთი დასახელებული 2 ჯგუფში. ნევროლოგიური დარღვევების მქონე 56 ბავშვიდან. ცვლილებები ტვინის MRI-ზე გამოვლინდა 47-ში (84%). თეთრი ნივთიერების დაზიანებები და ნაკლებად ხშირად გლიოზური ცვლილებები პერივენტრიკულარულ მიდამოში. EEG მონიტორინგმა აჩვენა ეპილეფსიური აქტივობის ტიპიური ნიმუში უმეტეს შემთხვევაში.

იშემიური ინსულტის ეს სამი შემთხვევა ბავშვებში სახის ლოკალიზებული სკლეროდერმიით

პირველები არიან წარმოდგენილი. ინსულტის მქონე ყველა პაციენტს დაუდგინდა გენეტიკური თრომბოფილია, ორს აღენიშნებოდა ცერებრალური სისხლძარღვთა მანკი, ხოლო ერთს - ანტიფოსფოლიპიდური სინდრომი.

დასკვნა. ჩვენი მონაცემები ვარაუდობს, რომ სკლეროდერმული ვასკულოპათია, ცერებრალური სისხლძარღვთა ანომალიები და გენეტიკური თრომბოფილია არის ინსულტის რიკ-ფაქტორები ბავშვებში სახის ლოკალიზებული სკლეროდერმიით. სავალდებულო MR ანგიოგრაფია და გენეტიკური თრომბოფილიის სკრინინგმა შეიძლება გამოავლინოს ინსულტის რისკის ჯგუფი.

საკვანძო სიტყვები: არასრულწლოვანთა ლოკალიზებული სახის სკლეროდერმია, არასრულწლოვანთა ლოკალიზებული სკლეროდერმია სახეზე დაფების გარეშე, ნევროლოგიური დარღვევები, თავის ტკივილი, ეპილეფსიური კრუნჩხვები, კრანიალური ნერვის ჩართვა, იშემიური ინსულტი, MRI და EEG ცვლილებები, გენეტიკური თრომბოფილია, ტვინის გემების მალფორმაცია.