 ISSN 2221-2582

UDC 616.127+616.831-005.4-053.7+617.754.5-031.41
DOI: 10.56871/CmN-W.2024.71.47.016

ISOLATED VISUAL FIELD IMPAIRMENT IN REPEATED CEREBRAL CIRCULATORY DISORDERS IN THE BASIN OF THE POSTERIOR CEREBRAL ARTERY IN A TEENAGER

© Zara G. Tadtayeva[1], Anna N. Galustyan[1], Vladislav O. Zavialov[2], Elizaveta S. Shanina[1], Adelia R. Iskalieva[1], Ivan S. Sardaryan[1]

[1] Saint Petersburg State Pediatric Medical University. 2 Lithuania, Saint Petersburg 194100 Russian Federation
[2] North-Western State Medical University named after I.I. Mechnikov. 41 Kirochnaya str., Saint Petersburg, 191015 Russian Federation

Contact information:
Zara G. Tadtayeva — Doctor of Medical Sciences, Professor of the Department of Pharmacology with a course in Clinical Pharmacology and Pharmaco-Economics. E-mail: Tadtaeva2003@mail.ru ORCID: https://orcid.org/0000-0002-5809-1457 SPIN: 6086-0169

For citation: Tadtayeva ZG, Galustyan AN, Zavialov VO, Shanina ES, Iskalieva AR, Sardaryan IS. Isolated visual field impairment in repeated cerebral circulatory disorders in the basin of the posterior cerebral artery in a teenager. Children's Medicine of the North-West. 2024;12(3):138–145. DOI: https://doi.org/10.56871/CmN-W.2024.71.47.016

Received: 02.07.2024 **Revised: 20.08.2024** **Accepted: 10.09.2024**

Abstract. Ischemic stroke in the basin of the posterior cerebral artery (PCA) is a rare disease. The clinical observation of repeated episodes of ischemic stroke in a 16-year-old patient in the PCA basin, manifested by isolated visual disturbances (visual field defects) and headache, is presented. The disease debuted with visual flicker. 6 months after the disease, incomplete homonymous hemianopia developed — lower quadrant hemianopi, confirmed by computer perimetry. When performing magnetic resonance imaging (MRI) of the brain, foci of ischemic changes in the left parietal-occipital region regione were revealed. Partial restoration of visual functions was noted against the background of therapy. The presence of isolated homonymous hemianopia dictates the need for highly informative diagnostic methods including magnetic resonance imaging of the brain and perimetric examination for timely diagnosis, adequate therapy and prevention of the disease.

Keywords: *vertebrobasilar pool, posterior cerebral stroke, homonymous hemianopia, lower quadrant hemianopia, children*

ИЗОЛИРОВАННОЕ НАРУШЕНИЕ ПОЛЕЙ ЗРЕНИЯ ПРИ ПОВТОРНЫХ НАРУШЕНИЯХ МОЗГОВОГО КРОВООБРАЩЕНИЯ В БАССЕЙНЕ ЗАДНЕЙ МОЗГОВОЙ АРТЕРИИ У ПОДРОСТКА

© Зара Григорьевна Тадтаева[1], Анна Николаевна Галустян[1], Владислав Олегович Завьялов[2], Елизавета Сергеевна Шанина[1], Аделя Руслановна Искалиева[1], Иван Суренович Сардарян[1]

[1] Санкт-Петербургский государственный педиатрический медицинский университет. 194100, г. Санкт-Петербург, ул. Литовская, д. 2
[2] Северо-Западный государственный медицинский университет имени И.И. Мечникова. 191015, г. Санкт-Петербург, ул. Кирочная, д. 41

Контактная информация:
Зара Григорьевна Тадтаева — д.м.н., профессор кафедры фармакологии с курсом клинической фармакологии и фармакоэкономики. E-mail: Tadtaeva2003@mail.ru ORCID: https://orcid.org/0000-0002-5809-1457 SPIN: 6086-0169

Для цитирования: Тадтаева З.Г., Галустян А.Н., Завьялов В.О., Шанина Е.С., Искалиева А.Р., Сардарян И.С. Изолированное нарушение полей зрения при повторных нарушениях мозгового кровообращения в бассейне задней мозговой артерии у подростка // Children's Medicine of the North-West. 2024. Т. 12. № 3. С. 138–145. DOI: https://doi.org/10.56871/CmN-W.2024.71.47.016

Поступила: 02.07.2024 **Одобрена: 20.08.2024** **Принята к печати: 10.09.2024**

Резюме. Ишемический инсульт в бассейне задней мозговой артерии (ЗМА) является редким заболеванием. Приведено клиническое наблюдение повторных эпизодов ишемического инсульта у пациента 16 лет в бассейне ЗМА, проявившегося изолированными зрительными нарушениями (дефектами поля зрения) и головной болью. Заболевание дебютировало зрительным мерцанием. Через 6 месяцев после болезни

развилась неполная гомонимная гемианопсия — нижнеквадрантная гемианопсия, подтвержденная компьютерной периметрией. При выполнении магнитно-резонансной томографии (МРТ) головного мозга выявлены очаги ишемических изменений в левой теменно-затылочной области. На фоне терапии отмечено частичное восстановление зрительных функций. Наличие изолированной гомонимной гемианопсии диктует необходимость проведения высокоинформативных методов диагностики с включением МРТ головного мозга и периметрического обследования для своевременной диагностики, адекватной терапии и профилактики заболевания.

Ключевые слова*: вертебробазилярный бассейн, задний мозговой инсульт, гомонимная гемианопсия, нижнеквадрантная гемианопсия, дети*

INTRODUCTION

Strokes in children and adolescents constitute an urgent medical and social problem. The incidence of an arterial ischemic stroke in childhood ranges from 1.2 to 13 cases per 100,000 [1, 2]. Epidemiologic studies show that about 10–30% of all ischemic strokes and transient ischemic attacks occur in the vertebrobasilar basin (VBB) [3, 4]. Blood flow disorders in the posterior cerebral artery (PCA) blood supply zone occur in only 5–10% of cases. Despite secondary prevention with antiaggregants, recurrent cerebrovascular episodes in the VBB are not uncommon and occur in 23–52% of children [5] during the first three years.

Clinical diagnosis of insufficient blood supply to the VBB is difficult because of cross-supply of the vascular basins, so the clinical picture within the brain stem and posterior hemispheres of the brain may be transient and/or masked by other diseases [6].

The symptom complex of stroke in the VBB is characterized by a severe course: depression of consciousness of varying severity, ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficits, systemic dizziness, ataxia, dysarthria, headache, nausea, vomiting, and nystagmus [7–9]. 45–67% of patients develop persistent visual field impairment in the acute period of stroke, which fully recovers within the first 6 months of stroke in 7.5% of patients [10].

Ischemic stroke in the VBB occurs when blood flow is impaired in the vertebral, basilar, and posterior cerebral arteries. The vertebral arteries are divided into 4 segments (V_1–V_4), the first three of which form the extracranial portion and V_4 comes from the intracranial portion. The bilateral segments of V_4 merge to form the basilar artery. The cerebellar arteries (superior, anterior inferior, and posterior inferior) are part of the VBB and participate in the blood supply to the cerebellum. The two posterior cerebral arteries (PCA) supply blood to the cortical areas in the region of sulcus spinous, and the optic radiation in some cases receives blood from branches of the middle cerebral artery; accordingly, homonymous hemianopsia does not always imply infarction in the PCA basin [11, 12]. Most of the visual cortex is supplied with blood by branches of the PCA, additionally in the region of the occipital pole by branches of the middle cerebral artery (MCA) [13].

Ischemic stroke in the PCA basin in childhood is poorly studied. The most frequent variant of visual field changes is homonymous hemianopsia, occurring in 75% of patients. PCA occlusion is manifested by visual field defects: contralateral homonymous hemianopsia or contralateral quadrant hemianopsia (lower quadrant hemianopsia in the case of wedge lesions or upper quadrant hemianopsia in the case of lingual gyrus lesions) [12, 14, 15] in the absence of other manifestations of neurologic deficit. The literature describes cases of stroke in the PCA, which are manifested by hallucinations by the mechanism of "cortical release", visual and color agnosia, prosopagnosia, blindness denial syndrome (Anton's syndrome), visual attention deficit and optic-motor agnosia (Balint's syndrome) [10, 11].

We present a rare clinical observation of a patient with recurrent ischemic stroke in the PCA basin, which manifested an isolated visual field disorder in the form of incomplete homonymous hemianopsia — lower quadrant hemianopsia.

CLINICAL OBSERVATION

Patient A., 16 years old, came to the emergency room of the hospital with complaints of visual disturbance (partial loss of visual fields). From the anamnesis it is known that the disease developed acutely with visual disturbance (loss of the right visual fields) and subsequent marked diffuse headache without nausea and vomiting. He was urgently hospitalized in the neurosurgical department with the diagnosis: acute cerebral circula-

ISSN 2221-2582

tion disorder of ischemic type in the VBB. The first symptoms of the disease appeared 6 months before admission to the hospital in the form of visual flickering lasting 10–15 minutes. The frequency of visual disturbances in dynamics increased from once a month to daily. In 6 months from the onset of the disease, visual field loss on the left side developed, which regressed almost completely within 10 days. Repeated cerebral circulation disorder occurred 27 days after the first acute vascular episode in the form of loss of visual fields on the right and headache, in connection with which he was urgently hospitalized.

Anamnesis of life. Obstetric history is aggravated (acute respiratory disease and threat of early termination). He was born on time against the background of mild preeclampsia. Birth weight — 3700 g, height — 53 cm, Apgar score — 8/9 points. He was discharged on time. Psychomotor development corresponded to the age. He was observed by a neurologist at preschool and primary school age for compulsive movements, tic hyperkinesis. During his life he suffered from acute respiratory viral infections, bronchitis, infectious mononucleosis, chickenpox. Suffers from vitiligo. He denies traumas, convulsions, loss of consciousness. Heredity: paternal grandmother had a stroke at the age of 50, peptic ulcer disease. Paternal hypertension in the grandfather. Somatic status at admission: condition of average severity. Height 168 cm, weight 58 kg. Skin and visible mucous membranes with no rush. Turgor and elasticity of tissues are preserved. Nasal breathing is free. Breathing is conducted in all sections, vesicular, no rales. Frequency of respiratory movements — 18 per minute. Heart tones are rhythmic. Blood pressure (BP) 125/70 mm Hg, heart rate (HR) — 104 per minute, respiratory rate — 20 per minute. The abdomen is soft, painless. Stool, urination is normal.

Neurologic status. Consciousness is clear. Active, available for contact, adequately responds to examination. Olfaction is not disturbed. Eye slits D=S. Pupils D=S. Photoreaction is alive. Eye movements are not limited, there is no nystagmus. Convergence, accommodation are not disturbed. No diplopia. The trigeminal nerve exit points are painless. Sensitivity on the face is preserved. Nasolabial folds are equal. Hearing is not disturbed. Tongue on the center line. Swallowing and phonation are intact. Speech is clear. Movements are complete in the extremities. No paresis. Muscle tone is not disturbed. Hand reflexes are alive, D=S, knee reflexes are alive, Achilles reflexes are alive. Abdominal reflexes are alive, symmetrical. There are no pathologic reflexes. Coordination tests is performed satisfactorily. There are no meningeal symptoms.

Laboratory admission tests

Clinical blood analysis: increase in hemoglobin — 169 g/L, leukocytes — 12.3×10^9/L, monocytes — 0.88×10^9/L, average concentration of hemoglobin in erythrocytes — 363 g/L, increase in the average volume of platelets — 11.1 fL, increase in the distribution width of erythrocytes — 14.7%; biochemical analysis of blood (including lipoprotein levels), urinalysis within normal limits.

Enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR) of blood to viruses of *Herpes viridae* did not reveal the presence of viruses.

Coagulation tests (activated partial thromboplastin time (APTT), prothrombin index (PTI), prothrombin time (PTT), fibrinogen) within normal limits.

Blood homocysteine — 8.3 mmol/L.

Blood: antiphospholipid syndrome — negative result.

Examination of ophthalmologist: optic disc is pale pink, borders are clear, scleral myopic cones on the temporal side, vessels of normal course and caliber, there is no pathology in the macular area and in the periphery in the visible area. Myopia of medium degree: OD: 0.5; sph –4.50 = 1.0;

OS: 0.4; sph –4.50 = 1.0. Computerized perimetry: homonymous lower quadrant hemianopsia (Fig. 1).

Electroencephalography (EEG): no focal changes, no paroxysmal forms of activity were detected.

MRI examination of the brain revealed foci of ischemia in the occipital region on both sides (Figs. 2, 3).

MR angiography: hypoplasia of the V_5 segment of the right vertebral artery.

Multispiral computed tomography (MSCT) of the brain and neck with contrast: CT picture of thrombosis of the left vertebral artery (V_1–V_2 segments).

Against the background of antiaggregant and anticoagulant therapy under the control of coagulation tests, the patient's condition stabilized. Positive dynamics in the form of partial recovery of visual functions and laboratory parameters were noted.

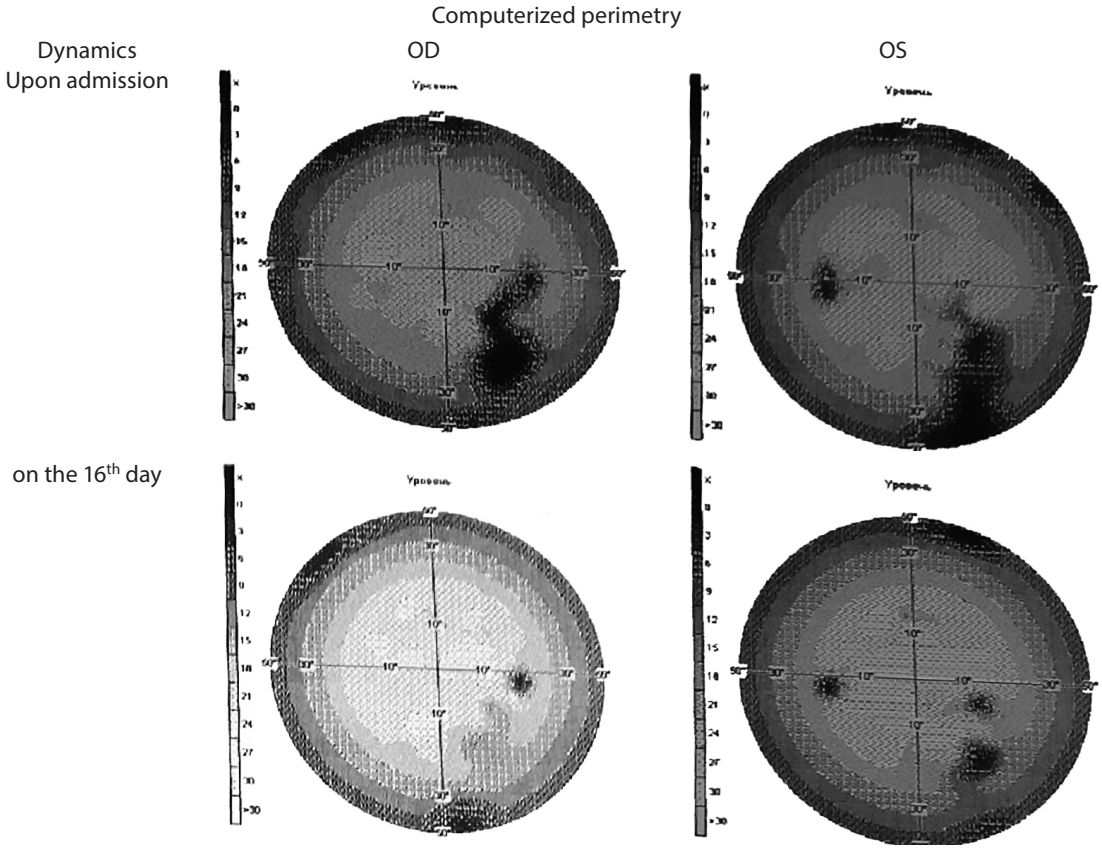

Fig. 1. Right-sided lower-quadrant homonymous hemianopia on the computer perimeter in dynamics

Рис. 1. Правосторонняя нижнеквадрантная гомонимная гемианопсия на компьютерном периметре в динамике

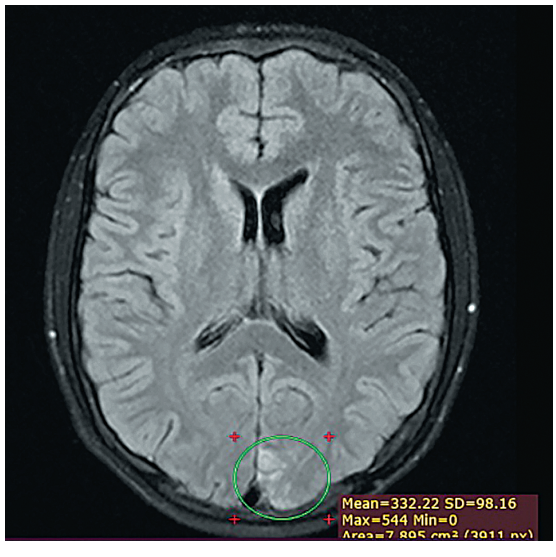

Fig. 2. MRI of the brain of a 16-year-old patient with foci of ischemic changes in the left occipital lobe

Рис. 2. МРТ головного мозга пациента 16 лет с очагами ишемических изменений в левой затылочной доле

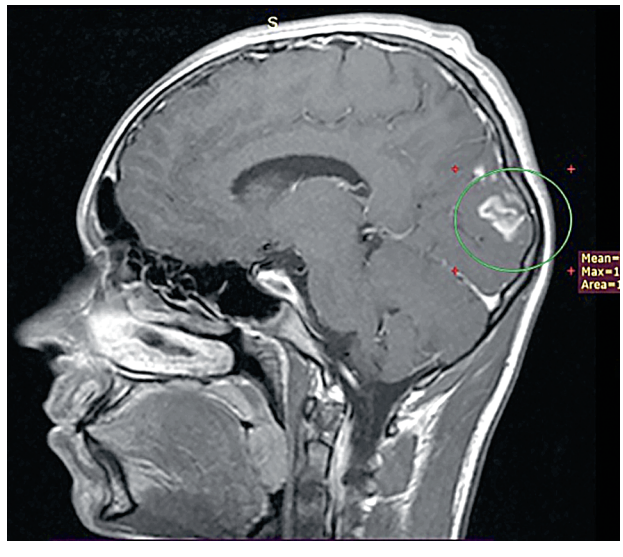

Fig. 3. MRI of the brain of a 16-year-old patient with foci of ischemic changes in the left parietal region

Рис. 3. МРТ головного мозга пациента 16 лет с очагами ишемических изменений в левой теменной области

 ISSN 2221-2582

A genetic panel of thrombophilia was performed in outpatient conditions. Allelic polymorphism of genes was revealed:

- *MTR 2756* methionine synthase gene (*Asp 919 Glu*), heterozygote A/G;
- methionine synthase reductase gene *MTRR 66 A/G,* heterozygote A/G ;
- methylenetetrahydrofolate reductase gene *MTHFR 677 C/T (Ala 222 Val)*, heterozygote C/T;
- platelet integrin subunit IIIA gene (platelet fibrinogen receptor) *ITGBZ 1565 T/C*, heterozygote T/C;
- platelet receptor gene for collagen (integrin alpha-2) *ITGA 807 C/T,* heterozygote C/T .

DISCUSSION

The diagnosis of ischemic stroke in the PCA basin is difficult because of the peculiarities of the circulation and lack of symptoms specificity [16]. The most frequent manifestations of the disease are homonymous visual field disturbances and headache. The nonspecific nature of symptoms necessitates differential diagnosis with migraine, acute sinusitis, and multiple sclerosis. The disease debuted with flickering vision without headache, which was ignored by the patient for quite a long time, leading to late diagnosis and treatment. The flickering vision was not accompanied by headache and indicated hemodynamic disorders of the visual system on one side above the chiasma. The patient was admitted to the hospital with partial loss of the same visual fields, which is a sign of blood flow disturbance in the area of the striatal cortex, optic radiation or lateral patellar body [17, 18]. Ischemic changes detected on neuroimaging in the parieto-occipital region confirm the level of visual pathway damage.

CT angiography of the spine diagnosed thrombosis of the left vertebral artery (V_1–V_2 segments). It is known that occlusion of a single vertebral artery does not lead to ischemic infarction; however, if the patient has cerebral infarction in the basin of PCA branches and vertebral artery thrombosis, the causal relationship is more likely in combination with pathology in the cervical spine, craniovertebral region, presence of cervical rib, vascular atherosclerosis, and Willis Circle anomalies. Cervical carotid and vertebral artery dissection is a well-known cause of stroke in young adults, especially in patients with migraine, fibromuscular dysplasia, hypertension, and trauma [19, 20].

Stroke in childhood and adolescence is rarely associated with adult obligatory risk factors such as vascular atherosclerosis, arterial hypertension, dyslipidemia, diabetes mellitus, etc. The main etiologic and predisposing factors are recognized as acquired and congenital prothrombotic disorders involving heart diseases (including congenital malformations and cardiomyopathies), (arteriopathies craniocervical arterial dissection, fibromuscular dysplasia, primary angiitis); congenital metabolic disorders; infections; head and neck trauma; sickle cell anemia; prothrombotic disorders; autoimmune diseases and malignancies [21, 22]. The most significant factors are heart defects, arteriopathies, hereditary thrombophilia, and prothrombotic disorders [23]. The factors determining the risk of stroke recurrence are not fully understood. They include heart disease, arteriopathies, prothrombotic factors, monogenic forms of stroke, and hereditary thrombophilia due to polymorphisms of genes of blood coagulation factors, fibrinolysis, thrombocyte hemostasis, and homocysteine metabolism [24].

In our observation, the patient had a combination of five polymorphisms of genes related to the hemostasis system: heterozygous mutations of *MTHFR* and platelet glycoprotein genes, i.e. there is a polygenic hereditary predisposition to thrombotic lesions of cerebral vessels [25].

Mutation in the methylenetetrahydrofolate reductase (MTHFR) gene leads to a thermolabile enzyme with reduced enzymatic activity and is one of the reasons for the development of hyperhomocysteinemia (HHC) [26]. Studies have shown that genetically determined HHC is associated with an increased risk of ischemic stroke due to its effects on the endothelium of the vascular wall not only in adults but also in children [27]. The patient's plasma homocysteine level was 8.3 μmol/L (with a normal range of 5 μmol/L in children) [28].

Disturbance in the system of vascular-platelet hemostasis is one of the significant pathophysiologic causes of stroke development regardless of etiology. Polymorphisms of platelet glycoprotein receptors, called human platelet antigens (HPA), modulate receptor density, altering platelet function and thrombus formation [29]. Particular attention has been paid to the fibrinogen receptor, GpIIb/IIIa complex, which mediates aggregation of active platelet forms. The pathogenetic role of GpIIIa 1565 T/C glycoprotein polymorphism is due to the increased receptor function of platelets and aggregation of these blood platelets, and fibrinogen is the main plasma cofactor of this process. An almost threefold increased risk in HPA carri-

ers for venous sinus thrombosis in children has been found [30]. Carriage of several polymorphic thrombophilia factors in a patient may indicate a cumulative prothrombotic effect on the risk of ischemic stroke [31, 32]. Thus, one of the risk factors for stroke development in a patient is hereditary multigenic thrombophilia.

DISCUSSION

The sudden onset of homonymous visual field defects is a consequence of cerebral vascular lesions in the occipital lobe supplied by branches of the posterior cerebral artery. Thorough clinical examination involving neuroimaging and perimetry makes it possible to diagnose a stroke in the basin of the posterior cerebral artery branches, and to provide timely adequate therapy and secondary prevention to avoid recurrence [33].

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

Funding source. This study was not supported by any external sources of funding.

Consent for publication. Written consent was obtained from legal representatives of the patient for publication of relevant medical information within the manuscript.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили письменное согласие законных представителей пациента на публикацию медицинских данных.

REFERENCES

1. Lee K.L., Tseng Y.C., Yang S.T., Kuo Y.T. Uncommon pediatric stroke caused by MCA dissection presenting as initial loss of consciousness. Pediatr. Neonatol. 2018;59:634–635. DOI: 10.1016/j.pedneo.2018.04.012.
2. deVeber G.A., Kirton A., Booth F.A., Yager J.Y., Wirrell E.C., Wood E. et al. Epidemiology and outcomes of arterial ischemic stroke in children: the Canadian Pediatric Ischemic Stroke Registry. Pediatr Neurol. 2017;69:58–70. DOI: 10.1016/j.pediatrneurol.2017.01.016.
3. Carey S., Wrogemann J., Booth F.A., Rafay M.F. Epidemiology, clinical presentation, and prognosis of posterior circulation ischemic stroke in children. Pediatr Neurol. 2017;74:41–50. DOI: 10.1016/j.pediatneurol.2017.05.007.
4. Bogousslavsky J., Van Melle G., Regli F. The lausanne stroke registry: analysis of 1,000 consecutive patients with first stroke. Stroke. 1988;19:1083–1092.
5. Sträter R, Becker S, von Eckardstein A. et al. Prospective assessment of risk factors for recurrent stroke during childhood — a 5-year follow-up study. Lancet. 2002;360(9345):1540–1545.
6. Ganesan V., Chong W.K., Cox T.C., Chawda S.J., Prengler M., Kirkham F.J.. Posterior circulation stroke in childhood: risk factors and recurrence. Neurology. 2002;59(10):1552–6. DOI: 10.1212/01.wnl.0000033092.87560.1a. PMID: 12451196.
7. Searls D.E., Pazdera L., Korbel E. et al. Symptoms and signs of posterior circulation ischemia in the New England Medical Center Posterior Circulation registry. Arch Neurol. 2012;69:346–351.
8. Edlow J.A., Newman-Toker D.E., Savitz S.I. Diagnosis and initial management of cerebellar infarction. Lancet Neurol. 2008;7:951–964. DOI: 10.1016/S1474-4422(08)70216-3.
9. Carvalho V., Cruz V.T. Clinical presentation of vertebrobasilar stroke. Porto Biomed J. 2020;5(6):e096. Published 2020 Nov 24. DOI: 10.1097/j.pbj.0000000000000096.
10. Rowe F.J., Wright D., Brand D. et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation and outcome. BiomedResInt. 2013;2013:719096. DOI: 10.1155/2013/719096.
11. Gusev E.I. Neurology. National leadership in 2 volumes. Ed. E.I. Guseva, A.N. Konovalova, V.I. Skvortsova. 2nd ed., revised. and additional. Moscow: GEOTAR-Media. 2019;2. (in Russian).
12. Goodwin D. Homonymous hemianopsia: problems and solutions. Clinical ophthalmologist. 2014;8:1919–27.
13. De Moraes C.G. Anatomy of the visual pathways. J Glaucoma. 2013;22(5):S2–7.

14. Skoromets A.A., Skoromets A.P., Skoromets T.A. Nervous diseases: a textbook. 6th ed. M.: MED-press-inform; 2013. (In Russian).
15. Pula J.H., Yuen C.A. Eyes and stroke: the visual aspects of cerebrovascular disease. Stroke Vasc Neurol. 2017;2:210–220. DOI: 10.1136/svn-2017-000079.
16. Kuybu O., Tadi P., Dossani R.H. Posterior cerebral artery stroke. StatPearls Publishing, Treasure Island, FL; 2021.
17. Pula J.H., Yuen C.A. Eyes and stroke: The visual aspects of cerebrovascular disease. Stroke Vasc Neurol. 2017;2:210–20.
18. Du H., Lei H., Ambler G., Fang S., He R., Yuan Q. et al. Intravenous thrombolysis before mechanical thrombectomy for acute ischemic stroke: A meta-analysis. J Am Heart Assoc. 2021;10:e022303.
19. Zhang W.N., Pan Y.H., Wang X.Y., Zhao Y. A prospective study of the incidence and correlated factors of post-stroke depression in China. PLoS One. 2013;8(11):e78981. DOI: 10.1371/journal.pone.0078981.
20. Blount S.B., Galton C. Cervical carotid or vertebral artery dissection. Underdiagnosed cause of stroke in young people. BMJ. 1997;314:243.
21. Felling R.J., Sun L.R., Maxwell E.C., Goldenberg N., Bernard T. Pediatric arterial ischemic stroke: Epidemiology, risk factors, and management. Blood Cells Mol. Dis. 2017;67:23–33. DOI: 10.1016/j.bcmd.2017.03.003.
22. Numis, Alabama; Fox K.K. Arterial ischemic stroke in children: risk factors and etiology. Well Neurol Neurologists. 2014;14:1–9.
23. Sareka-Hajar B., Hooves I. Risk factors for recurrent arterial ischemic stroke in children and adolescents. Brain science. 2020;10:24.
24. Sareka-Hajar; Lopez M.V., Castro PDC., Valderrama E.B., Herrero MCM., Gol Villanueva N., Romero AJA., Pascual S.I. Outcome of arterial ischemic stroke in children with heart disease. Euro. J. Pediatrician. Neuro. 2017;21:730–737.
25. Kenet G., Lütkhoff L.K., Albisetti M., Bernard T., Bonduel M., Brandao L., Chabrier S., Chan A., Deveber G., Fiedler B. Impact of thrombophilia on risk of arterial ischemic stroke or cerebral sinovenous thrombosis in neonates and children: A sytematic review and meta-analysis of observational studies. Circulation. 2010;121:1838–1847. DOI: 10.1161/CIRCULATIONAHA.109.913673.
26. Suslina Z.A., Tanashyan M.M., Ionova V.G. Ischemic stroke: blood, vascular wall, antithrombotic therapy. Moscow: Med. Kniga; 2004. (In Russian).
27. Tadtaeva Z.G., Katsadze Yu.L. Combination of defective genes: fibrinogen, plasminogen activator inhibitor, methylenetetrahydrofolate reductase (MTHFR) and hyperhomocysteinemia (HHC) in moyamoya disease (clinical observation). Nevrologicheskiy zhurnal. 2007;3:56–57. (in Russian).
28. Shevchenko O.P., Olifrienko G.A. Hyperhomocysteinemia and its clinical significance. Laboratoriya. 2002;1:3–7. (in Russian).
29. Wen Y., Chen D-P. Human platelet antigens in disease. Clin Chim Acta. 2018;484:87–90. DOI: 10.1016/j.cca.2018.05.009.
30. Čeri A., LeničekKrleža J., Coen Herak D. et al. Role of platelet gene polymorphisms in ischemic pediatric stroke subtypes: a case-control study. Croat Med J. 2020;61(1):18–27. DOI: 10.3325/cmj.2020.61.18.
31. Skoromets A.A., Tadtaeva Z.G., Pak T.E., Skoromets A.P. Genetic signs of thrombophilia in children and adolescents with stroke. Vestnik Sankt-Peterburgskogo universiteta. 2011;11(4):62–68. (In Russian).
32. Gerak D.C., Krleza J.L., Antolic M.R., Horvath I., Juranovich V., Theme R.Z., Zadro R. Association of gene polymorphisms of coagulation factors and enzymes of homocysteine metabolism with arterial ischemic stroke in children. wedge. dec. thrombus. 2016;23:1042–1051.
33. Rowe F.J., Wright D., Brand D. et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation, and outcome. Biomed Res Int. 2013;2013:719096.

ЛИТЕРАТУРА

1. Lee K.L., Tseng Y.C., Yang S.T., Kuo Y.T. Uncommon pediatric stroke caused by MCA dissection presenting as initial loss of consciousness. Pediatr. Neonatol. 2018;59:634–635. DOI: 10.1016/j.pedneo.2018.04.012.
2. deVeber G.A., Kirton A., Booth F.A., Yager J.Y., Wirrell E.C., Wood E. et al. Epidemiology and outcomes of arterial ischemic stroke in children: the Canadian Pediatric Ischemic Stroke Registry. Pediatr Neurol. 2017;69:58–70. DOI: 10.1016/j.pediatrneurol.2017.01.016.
3. Carey S., Wrogemann J., Booth F.A., Rafay M.F. Epidemiology, clinical presentation, and prognosis of posterior circulation ischemic stroke in children. Pediatr Neurol. 2017;74:41–50. DOI: 10.1016/j.pediatneurol.2017.05.007.
4. Bogousslavsky J., Van Melle G., Regli F. The lausanne stroke registry: analysis of 1,000 consecutive patients with first stroke. Stroke. 1988;19:1083–1092.
5. Sträter R, Becker S, von Eckardstein A. et al. Prospective assessment of risk factors for recurrent stroke during childhood – a 5-year follow-up study. Lancet. 2002;360(9345):1540–1545.
6. Ganesan V., Chong W.K., Cox T.C., Chawda S.J., Prengler M., Kirkham F.J.. Posterior circulation

stroke in childhood: risk factors and recurrence. Neurology. 2002;59(10):1552–6. DOI: 10.1212/01.wnl.0000033092.87560.1a. PMID: 12451196.
7. Searls D.E., Pazdera L., Korbel E. et al. Symptoms and signs of posterior circulation ischemia in the New England Medical Center Posterior Circulation registry. Arch Neurol. 2012;69:346–351.
8. Edlow J.A., Newman-Toker D.E., Savitz S.I. Diagnosis and initial management of cerebellar infarction. Lancet Neurol. 2008;7:951–964. DOI: 10.1016/S1474-4422(08)70216-3.
9. Carvalho V., Cruz V.T. Clinical presentation of vertebrobasilar stroke. Porto Biomed J. 2020;5(6):e096. Published 2020 Nov 24. DOI: 10.1097/j.pbj.0000000000000096.
10. Rowe F.J., Wright D., Brand D. et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation and outcome. BiomedResInt. 2013;2013:719096. DOI: 10.1155/2013/719096.
11. Гусев Е.И. Неврология. Национальное руководство в 2 т. Под ред. Е.И. Гусева, А.Н. Коновалова, В.И. Скворцовой. 2-е изд., перераб. и доп. М.: ГЭОТАР-Медиа; 2019;2.
12. Goodwin D. Homonymous hemianopsia: problems and solutions. Clinical ophthalmologist. 2014;8:1919–27.
13. De Moraes C.G. Anatomy of the visual pathways. J Glaucoma. 2013;22(5):S2–7.
14. Скоромец А.А., Скоромец А.П., Скоромец Т.А., Нервные болезни: учебное пособие. 6-е изд. М.: МЕДпресс-информ; 2013.
15. Pula J.H., Yuen C.A. Eyes and stroke: the visual aspects of cerebrovascular disease. Stroke Vasc Neurol. 2017;2:210–220. DOI: 10.1136/svn-2017-000079.
16. Kuybu O., Tadi P., Dossani R.H. Posterior cerebral artery stroke. StatPearls Publishing, Treasure Island, FL; 2021.
17. Pula J.H., Yuen C.A. Eyes and stroke: The visual aspects of cerebrovascular disease. Stroke Vasc Neurol. 2017;2:210–20.
18. Du H., Lei H., Ambler G., Fang S., He R., Yuan Q. et al. Intravenous thrombolysis before mechanical thrombectomy for acute ischemic stroke: A meta-analysis. J Am Heart Assoc. 2021;10:e022303.
19. Zhang W.N., Pan Y.H., Wang X.Y., Zhao Y. A prospective study of the incidence and correlated factors of post-stroke depression in China. PLoS One. 2013;8(11):e78981. DOI: 10.1371/journal.pone.0078981.
20. Blount S.B., Galton C. Cervical carotid or vertebral artery dissection. Underdiagnosed cause of stroke in young people. BMJ. 1997;314:243.
21. Felling R.J., Sun L.R., Maxwell E.C., Goldenberg N., Bernard T. Pediatric arterial ischemic stroke: Epidemiology, risk factors, and management. Blood Cells Mol. Dis. 2017;67:23–33. DOI: 10.1016/j.bcmd.2017.03.003.
22. Numis, Alabama; Fox K.K. Arterial ischemic stroke in children: risk factors and etiology. Well Neurol Neurologists. 2014;14:1–9.
23. Sareka-Hajar B., Hooves I. Risk factors for recurrent arterial ischemic stroke in children and adolescents. Brain science. 2020;10:24.
24. Sareka-Hajar; Lopez M.V., Castro PDC., Valderrama E.B., Herrero MCM., Gol Villanueva N., Romero AJA., Pascual S.I. Outcome of arterial ischemic stroke in children with heart disease. Euro. J. Pediatrician. Neuro. 2017;21:730–737.
25. Kenet G., Lütkhoff L.K., Albisetti M., Bernard T., Bonduel M., Brandao L., Chabrier S., Chan A., Deveber G., Fiedler B. Impact of thrombophilia on risk of arterial ischemic stroke or cerebral sinovenous thrombosis in neonates and children: A sytematic review and meta-analysis of observational studies. Circulation. 2010;121:1838–1847. DOI: 10.1161/CIRCULATIONAHA.109.913673.
26. Суслина З.А., Танашян М.М., Ионова В.Г. Ишемический инсульт: кровь, сосудистая стенка, антитромботическая терапия. М.: Мед. книга; 2004.
27. Тадтаева З.Г., Кацадзе Ю.Л. Комбинация дефектных генов: фибриногена, ингибитора активатора плазминогена, метилентетрагидрофолатредуктазы (MTHFR) и гипергомоцистеинемии (ГГЦ) при болезни мойя-мойя (клиническое наблюдение). Неврологический журнал. 2007;3:56–57.
28. Шевченко О.П., Олифриенко Г.А. Гипергомоцистеинемия и ее клиническое значение. Лаборатория. 2002;1:3–7.
29. Wen Y., Chen D-P. Human platelet antigens in disease. Clin Chim Acta. 2018;484:87–90. DOI: 10.1016/j.cca.2018.05.009.
30. Čeri A., LeničekKrleža J., Coen Herak D. et al. Role of platelet gene polymorphisms in ischemic pediatric stroke subtypes: a case-control study. Croat Med J. 2020;61(1):18–27. DOI: 10.3325/cmj.2020.61.18.
31. Скоромец А.А, Тадтаева З.Г., Пак Т.Е., Скоромец А.П. Генетические признаки тромбофилии у детей и подростков при инсульте. Вестник Санкт-Петербургского университета. 2011;11(4):62–68.
32. Gerak D.C., Krleza J.L., Antolic M.R., Horvath I., Juranovich V., Theme R.Z., Zadro R. Association of gene polymorphisms of coagulation factors and enzymes of homocysteine metabolism with arterial ischemic stroke in children. wedge. dec. thrombus. 2016;23:1042–1051.
33. Rowe F.J., Wright D., Brand D. et al. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation, and outcome. Biomed Res Int. 2013;2013:719096.