Impact Factor: 4.9

ISSN: 2181-0664
DOI: 10.26739/2181-0664
tadqiqot.uz/uzbek-medikal-journal

UZBEK MEDICAL JOURNAL

Volume 2, Issue 1

2021

ЎЗБЕК ТИББИЁТ ЖУРНАЛИ
УЗБЕКСКИЙ МЕДИЦИНСКИЙ ЖУРНАЛ | UZBEK MEDICAL JOURNAL
№1 (2021) DOI http://dx.doi.org/10.26739/2181-0664-2021-1

Page Maker | Верстка | Саҳифаловчи: Хуршид Мирзахмедов

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Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC the city of Tashkent,
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МУНДАРИЖА / СОДЕРЖАНИЕ / CONTENT

ЎЗБЕК ТИББИЁТ ЖУРНАЛИ
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Munis Dilshod qizi Fayzieva
Department of Neurology, Pediatric Neurology and Medical Genetics, Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan,
e-mail: munis.fayziyeva@gmail.com
Durdona Djurabaevna Usmanova
doctor of medical sciences, docent, Department of Neurology, Pediatric Neurology and Medical Genetic, Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan
durdona.usmanova@mail.ru

FEATURES OF COGNITIVE IMPAIRMENT DEPENDING ON THE STAGE OF CHRONIC CEREBRAL ISCHEMIA

http://dx.doi.org/10.26739/2181-0664-2021-1-10

ABSTRACT
The article presents the results of the analysis of literature sources on chronic cerebral ischemia, etiology and pathogenetic mechanisms of the development of cognitive impairment. In the pathogenesis of chronic cerebral ischemia, systemic and local factors are important, leading to disorders of cerebral hemodynamics, the most adverse effect is exerted by their combination. The most common cause of local disorders of cerebral blood flow is atherosclerotic stenosis and occlusion of intracerebral and extracranial vessels that perform transport and distribution functions.
Keywords: chronic cerebral ischemia, cognitive disorders, neurological disorders

Мунис Дилшод қизи Файзиева
Кафедра Неврологии, детской неврологии и медицинской генетики, Ташкентский Педиатрический Медицинский Институт, Ташкент, Узбекистан
Эл.почта: munis.fayziyeva@gmail.com
Дурдона Джурабаевна Усманова
доктор медицинских наук, доцент
Кафедра Неврологии, детской неврологии и медицинской генетики, Ташкентский Педиатрический Медицинский Институт, Ташкент, Узбекистан
Эл.почта: durdona.usmanova@mail.ru

ОСОБЕННОСТИ КОГНИТИВНЫХ НАРУШЕНИЙ В ЗАВИСИМОСТИ ОТ СТАДИИ ХРОНИЧЕСКОЙ ИШЕМИИ МОЗГА

АННОТАЦИЯ

В статье приведены результаты анализа источников литературы по хронической ишемии мозга, этиологии и патогенетическим механизмам развития когнитивных нарушений. В патогенезе хронической церебральной ишемии важное значение имеют системные и локальные факторы, приводящие к нарушениям гемодинамики головного мозга, наиболее неблагоприятное воздействие оказывает их сочетание. Наиболее распространенной причиной местных нарушений мозгового кровотока является атеросклеротический стеноз и закупорка внутримозговых и экстракраниальных сосудов, выполняющих функции транспорта и распределения.

Ключевые слова: хроническая ишемия мозга, когнитивные расстройства, неврологические нарушения

Мунис Дилшод қизи Файзиева
Неврология, болалар неврологияси ва тиббий генетика кафедраси,
Тошкент Педиатрия Тиббиёт Институти, Тошкент, Ўзбекистон
Эл.манзил: munis.fayziyeva@gmail.com
Дурдона Джурабаевна Усманова
Неврология, болалар неврологияси ва тиббий генетика кафедраси,
Тошкент Педиатрия Тиббиёт Институти, Тошкент, Ўзбекистон
Эл.манзил: durdona.usmanova@mail.ru

СУРУНКАЛИ БОШ МИЯ ИШЕМИЯСИ БОСҚИЧИДАН КЕЛИБ ЧИҚҚКАН ҲОЛДА КОГНИТИВ БУЗИЛИШЛАРНИНГ ХУСУСИЯТЛАРИ

АННОТАЦИЯ

Мақолада сурункали бош мия ишемияси, когнитив бузилишларнинг этиологияси ва патогенетик механизмлари бўйича адабиётлар тахлили натижалари келтириб ўтилган. Сурункали серебрал ишемия патогенезида бош мия гемодинамикаси бузилишларига олиб келадиган тизимли ва маҳаллий омиллар муҳим аҳамиятга эга, уларнинг уйғунлашуви энг ноқулай таъсир кўрсатади. Маҳаллий мия қон оқими бузилишларининг энг кенг тарқалган сабаби атеросклеротик стеноз ва транспорт ва тақсимлаш вазифасини бажарувчи қон томирларининг қопланишидир.

Калит сўзлар: сурункали бош мия ишемияси, когнитив бузилишлар, неврологик бузилишлар.

Chronic cerebral ischemia is a progressive form of cerebrovascular insufficiency of the brain, accompanied by small focal or diffuse brain damage and is manifested by neurological and cognitive disorders (13,15). With vascular pathology in the body, parallel lesions of various bloodstream parts most often develop, caused by such pathological processes as atherosclerosis, arterial hypertension, diabetes mellitus. These diseases, the summation of which in one patient is called vascular comorbidity, can hurt the course of cerebrovascular diseases, the results of its treatment and prognosis. The first pathophysiological stage of all pathological processes in vascular comorbidity in most cases is endothelial dysfunction, progressing against the background of energy instability caused by metabolic imbalances (6, 14). In chronic cerebral ischemia, as a result of cerebral ischemia, oxidative processes are intensified, while the lack of antioxidant protection of the brain leads to the formation of a state of oxidative stress (11,16). Oxidative stress is one of the systemic stress response components to any damaging effect on the body. With a decrease in cerebral blood flow, mitochondrial oxidative phosphorylation processes are disrupted, endothelial dysfunction develops due to disturbances in cellular energy metabolism (2,3). As a result, micro- and macroangiopathies develop, which is clinically manifested by the formation, depending on the severity of chronic cerebral ischemia, asthenic, cognitive, neuropsychiatric and focal manifestations. The earliest indicators of vascular brain damage are cognitive impairments

combined with emotional impairments that tend to progress. Accordingly, the dynamics of the severity of disorders in the cognitive sphere can indicate the degree of vascular lesion and the effectiveness of the therapy (2, 11, 13).

In the pathogenesis of chronic cerebral ischemia, systemic and local factors are important, leading to cerebral hemodynamics disorders, the most adverse effect is exerted by their combination (1,4,17).

Violations of systemic hemodynamics can be caused by a pathology leading to a decrease in the myocardium's pumping function, an increase or decrease in systemic arterial pressure, disruption of cerebral blood flow autoregulation, a violation of the coagulation and rheological properties of blood (6,8,12,14). The most common cause of local cerebral blood flow disorders is atherosclerotic stenosis and occlusion of intracerebral and extracranial vessels that perform transport and distribution functions. A decrease in blood flow in atherosclerosis of the main arteries leads to a reduction (obliteration and fibrosis) of the microvascular bed with the development of hypoxia and a cascade of pathobiochemical disorders mediated by ischemia. An important independent risk factor for developing chronic cerebral ischemia is type 2 diabetes mellitus (DM2), the prevalence of which in the population of people over 60 years old is up to 20% and increases with age (8,9).

According to the literature, the main causes of chronic cerebral ischemia (15):

- atherosclerosis;
- arterial hypertension.

Additional causes of chronic cerebral ischemia:

- cardiovascular diseases;
- violation of the heart rhythm;
- vascular anomalies, hereditary angiopathies;
- venous pathology;
- vascular compression;
- arterial hypotension;
- cerebral amyloidosis;
- vasculitis, diabetes mellitus;
- blood diseases.

The clinical picture of chronic cerebral ischemia has a progressive development and, according to the severity of symptoms, is divided into three stages: initial manifestations, subcompensation and decompensation (2,6).

In the stage, I (initial), headaches, dizziness, heaviness and noise in the head, sleep disturbances, increased fatigue, and irritability prevail. In the neurological status, "microorganic" symptoms are determined in the form of a revival of deep reflexes with their possible slight asymmetry, the presence of subcortical reflexes, impaired convergence, moderate cognitive fronto-subcortical disorders in the form of impaired attention, cognitive activity, memory loss for current events without impairing professional and social adaptation (10,13,14).

Stage II (subcompensation) is characterized by focal symptoms with the formation of a clinical syndrome or syndromes (vestibuloatactic, pyramidal, akinetic-rigid, etc.). Cognitive disorders are aggravated, which is expressed in a decrease in memory, including professional memory, due to impaired active search in memory and reproduction of material with sufficient preservation, as well as impaired attention, slowing down of mental processes (bradifrenia), limiting the ability to plan and control. Emotional and personality disorders are manifested in emotional lability, depression, and a decrease in criticism. The professional and social adaptation of the patient is impaired, however, the possibility of self-service remains (15).

In stage III (decompensation), in addition to the syndrome or a combination of syndromes characteristic of stage II, pseudobulbar disorders are often determined, persistent residual effects of acute cerebral circulation disorders in the form of pyramidal and extrapyramidal symptoms. Cognitive impairments are characterized by decreased criticism, subcortical or subcortical-cortical dementia, and the subcortical-frontal systems' predominant dysfunction. Emotional and personal

disorders are manifested by apathic-abulic syndrome, disinhibition. Patients lose the ability to self-care and need outside care (12,15,16).

In chronic cerebral ischemia, there is a clear correlation between the severity of neurological symptoms and patients' age. This must be borne in mind when assessing the significance of certain neurological signs that are considered normal for elderly and senile people. This dependence reflects the age-related manifestations of dysfunctions of the cardiovascular and other visceral systems that affect the state and function of the brain. To a lesser extent, this relationship can be traced in hypertensive encephalopathy. In this case, the severity of the clinical picture is largely due to the underlying disease's course and its duration (7).

Along with the progression of neurological symptoms, as the pathological process develops in the brain's neurons, there is an increase in cognitive disorders. This applies to memory and intelligence, impaired in the 3rd stage to the level of dementia, and such neuropsychological syndromes as praxis and gnosis (7,10). Initial, essentially subclinical disorders of these functions are observed already in the 1st stage, then they intensify, change, become distinct. The 2nd and especially the 3rd stages of the disease are characterized by striking violations of higher cerebral functions, which sharply reduces the quality of life and social adaptation of patients (3,15).

A feature of the cephalgic syndrome is its polymorphism, inconsistency, the absence in most communication cases with specific vascular and hemodynamic factors (excluding headache in hypertensive crises with high blood pressure numbers), a decrease in the frequency of occurrence as the disease progresses. The second most common is a vestibulo-ataxic syndrome (3,5). The main complaints of patients are dizziness, unsteadiness when walking, coordination disorders. Sometimes, especially in the initial stages, patients, complaining of dizziness, do not notice coordination disorders. The results of an otoneurological study are also insufficiently indicative. In the later stages of the disease, subjective and objective discoordination disorders are interrelated (1,5,6). Dizziness, instability when walking may be partially associated with age-related changes in the vestibular apparatus, motor system and ischemic neuropathy of the vestibular cochlear nerve. Therefore, to assess subjective vestibulo-ataxic disorders' significance, their qualitative analysis is important when interviewing a patient, neurological and otoneurological examination.

In most cases, these disorders are caused by a chronic circulatory failure in the vertebrobasilar arterial system's blood supply basin. Therefore, it is necessary to rely not on patients' subjective feelings but to look for signs of diffuse lesions of the brain regions, the blood supply of which is carried out from this vascular basin (1,8,9). In some cases, in patients with chronic cerebral ischemia of stages 2–3, ataxic disorders are caused not by cerebellar-stem dysfunction as by lesions of the frontal-stem tracts. There is a phenomenon of frontal ataxia, or gait apraxia, reminiscent of hypokinesia in Parkinsonism patients. A CT scan reveals significant hydrocephalus (along with cortical atrophy), i.e. a condition close to normotensive hydrocephalus occurs. In general, the syndrome of circulatory failure in the vertebrobasilar basin is diagnosed in chronic cerebral ischemia more often than the carotid system (8,10,15).

The pyramidal syndrome feature is its moderate clinical manifestation (anisoreflexia, mimic asymmetry, minimal paresis, revitalization of oral automatism reflexes, hand symptoms) (1,5,8). A distinct asymmetry of reflexes indicates either a previous cerebral stroke or another disease running under the guise of chronic cerebral ischemia (for example, volumetric intracranial processes, the consequences of traumatic brain injury) (1,12,16). The diffuse and fairly symmetrical revival of deep reflexes, as well as pathological pyramidal reflexes, often combined with a significant revival of oral automatism reflexes and the development of pseudobulbar syndrome, especially in old and senile age, indicates multifocal vascular brain damage (with the exclusion of other possible causes) (15, 17.18). Mental disorders are quite characteristic and varied in the form at different stages of chronic cerebral ischemia. If in the initial stages they are like asthenic, asthenodepressive and anxiety-depressive disorders, then in the 2nd and especially in the 3rd stage they are joined by pronounced dysmnestic and intellectual disorders that form the vascular dementia syndrome, which often comes first in the clinical picture. (2.4). Mental disorders, especially cognitive and emotionally volitional disorders, can be considered one of the most frequent manifestations of the

brain's organic pathology. It is known that the overwhelming majority of the cause of brain tissue damage is cerebrovascular insufficiency (1,9,10). Chronic progressive forms of cerebral insufficiency of vascular etiology are traditionally referred to in the domestic literature as discirculatory encephalopathy (1,2,3).

The leading role in the pathogenesis of cognitive impairment in chronic cerebral ischemia is played by damage to the deep sections of the white matter of the brain and basal ganglia, which leads to the disconnection of the connection between the frontal brain and subcortical structures (the phenomenon of disconnection). The mechanism of the formation of uncoupling is associated primarily with arterial hypertension and appears to be as follows (1,2,3).

Chronic uncontrolled arterial hypertension leads to secondary changes in the vascular wall of lipogyalinosis, which develops mainly in the microvasculature vessels (7, 12,15). The resulting arteriolosclerosis leads to a change in vascular reactivity or a violation of the cerebral vascular tone's autoregulation. Under these conditions, a decrease in blood pressure due to the addition of excessive antihypertensive therapy, or as a result of physiological circadian changes in blood pressure, leads to hypoperfusion in the zones of terminal blood circulation. The latter include deep cerebral structures (9,13,14). Acute ischemic episodes in the pool of deep penetrating arteries lead to the appearance of small-diameter lacunar infarctions in the deep regions of the brain. With an unfavorable course of arterial hypertension, repeated acute episodes lead to the emergence of a lacunar state, one of the variants of multi-infarction vascular dementia (1, 5,6,9). In addition to repeated acute disorders, chronic ischemia in the zones of terminal circulation is also assumed. The latter's marker reduces the periventricular or subcortical white matter leukoaraiosis, which pathomorphologically represents a zone of demyelination, gliosis, and expansion of the perivascular spaces (3,4,5). In some cases of an unfavorable course of arterial hypertension, subacute development of diffuse lesions of the white matter of the brain with a rapidly progressing dementia and other manifestations of disconnection is possible, which is sometimes denoted in the literature by the term Binswanger's disease (3,7,9).

Conclusions: Neurological and neuropsychological manifestations of cognitive vascular disorders developing due to CCI are well recognized and depend on the severity and localization of the lesion of one or another "vascular basin". The most often found violation of executive functions. In case of damage to the brain's subcortical structures, an increase in muscle tone, pyramidal signs, and frontal lobe syndrome are observed. With CVD, any cognitive function can be impaired; therefore, in many cases, it is possible to finally confirm the vascular cause of cognitive disorders only by imaging on MRI (16, 18, 19).

Reference:

1. Antipenko EA, Derugina AV, AV Gustov Systemic stresslimiting effect of mexidol in chronic cerebral ischemia // Journal of Neurology and Psychiatry. S.S. Korsakov, No. 4, 2016, pp. 28-31
2. Voronina T.A. Pioneer antioxidant neuroprotection. 20 years in clinical practice // Russian Medical Journal. Neurology, no. 1, 2016.
3. Gavrilova S. I. Ceraxon (cytokilin) in treatment of mild cognitive decline syndrome : a scientific publication / S. I. Gavrilova, Y. B. Fedorova (et al.) // Journal of Neurology and Psychiatry. - M., 2011. - №12. - C. 16-20. - Bibliography: 36 titles.
4. Gafurov B.G. Problems of teaching neurocognitive disorders in postgraduate training of a neurologist: a scientific publication / B.G. Gafurov, Sh. 5. Gafurov B.G. // Neurology. - Tashkent, 2011. - N4. - p. 7
5. Zakharov V.V. et al. Chronic insufficiency of cerebral circulation: description of a clinical case// Therapeutic Archives, No.4, 2016, pp.93-98
6. Ivanova I. Cognitive disorders in general therapeutic practice: a scientific publication / I. Ivanova, N. Shodieva, L. Pimenov // Phys. - Moscow, 2011. - №9. - pp. 72-74. - Bibliogr: 10 titles.

7. Majidova Y. N. Study of the effectiveness of a stepwise scheme of cytoflavin as an antioxidant, metabolic neuroprotector in patients with chronic cerebral ischemia: a scientific publication / Yo. N. 9. Majidova, D. D. Usmanova, J. M. Baitursunova, O. D. Mirzamukhamedov // Neurology. - Tashkent, 2011. - N4. - pp. 101-102
8. Majidova Y. N. Clinical and neurophysiological (evoked brain potentials) assess moderate cognitive disorders in patients with chronic brain ischemia: a scientific publication // Neurology. - Tashkent, 2013. - N2. - pp. 5-8
9. Rumyantseva SA, et al.Problems and prospects of correction of intermediate metabolism in patients with vascular comorbidity // Journal Neuronews №1-2 2014.
10. Saidvaliev F.S. Dynamic characteristic of cognitive impairment of different discirculatory encephalopathy stages: a scientific publication / F.S. Saidvaliev // Neurology. - Tashkent, 2012. - N3-4. - p. 161 (Cipher N9/2012/3-4)
11. Chukanova Ye.I., Chukanova A.S., Mamaev H.I. Results of the study of the effectiveness and safety of mexidol in patients with chronic brain ischemia // Journal of Neurology and Psychiatry. S.S. Korsakov, No. 2, 2015, pp. 71-74
12. Chukanova E.I., Chukanova A.S. Efficacy and safety of Mexidol FORTE 250 as part of sequential therapy in patients with chronic brain ischemia // Journal of Neurology and Psychiatry. S.S. Korsakov, 2019, vol. 119, no. 9, pp. 39-45
13. Yakhno N.N. Cognitive disorders in the neurological clinic. //Neurological Journal 2006 ,11(appendix 1);pp. 4-12
14. Achterberg S, Kappelle LJ, de Bakker PI, Traylor M, Algra A No Additional Prognostic Value of Genetic Information in the Prediction of Vascular Events after Cerebral Ischemia of Arterial Origin: The PROMISe Study. SMART Study Group and the METASTROKE Consortium. PLoS One. 2015 Apr 23;10(4):e0119203.
15. Arikan F, Vilalta J, Torne R, Noguer M, Lorenzo-Bosquet C, Sahuquillo J. Rapid resolution of ischemic brain hypoxia after cerebral revascularization in moyamoya disease. Neurosurgery. 2015 Mar;76(3):302-12
16. Igarashi K, Sakurai T, Kamiyoshi A, Ichikawa-Shindo Y, Kawate H, Yamauchi A, Toriyama Y, Tanaka M, Liu T, Xian X, Imai A, Zhai L, Owa S, Koyama T, Uetake R, Ihara M, Shindo T.Pathophysiological roles of the adrenomedullin-RAMP2 system in acute and chronic cerebral ischemia. Peptides. 2014 Dec;62:21-31

ISSN 2181-0664

Doi Journal 10.26739/2181-0664

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UZBEK MEDICAL JOURNAL

№1 (2021)

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