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Features of Cerebral Hemodynamics and Cognitive Status in Patients with Chronic Ischemia of the Brain on the Background of Stenosing Atherosclerosis of the Brachiocephalic Arteries

Abstract

Aim. To study the state of cerebral circulation and cognitive status (CS) in patients with chronic cerebral ischemia (CCI) associated with atherosclerotic lesions of the brachiocephalic arteries (BCA).

Materials and Methods. A total of 128 individuals aged 42 to 60 years were examined, including patients with CCI in the compensated (Group I, n=24) and subcompensated (Group II, n=38) stages, as well as 46 patients (Group III) with decompensated CCI, who had a history of an ischemic stroke (IS). The state of the carotid (CA), and vertebral arteries (VA) was assessed, as well as the time-averaged maximum blood flow velocity (TAMX) in the cerebral arteries, as well as in the vertebral (VA, segment V4) arteries, and basilar (BA) arteries, the functioning of the anterior (AComA) arteries and posterior (PComA) communicating arteries. The assessment of CS was performed using the Mini-Mental State Examination (MMSE), Frontal Assessment Battery (FAB), and a 10-word recall test.

Results. In Group I, isolated lesions of the CA or VA were observed, while in Group II and III multiple lesions dominated. The severity of CCI corresponded to the decrease in TAMX, most significantly in MCA in Group II ($p<0.05$) and Group III ($p<0.01$). Such vascular ischemia was clinically accompanied by an increase in ataxic, pseudobulbar, dysmnestic, and pyramidal syndromes. In patients with a non-functioning AComA and hemodynamically significant lesions of the BCA, a decrease in memory and intelligence, behavioural changes, were observed. In cases of non-functioning PComA, a decrease in TAMX in the posterior cerebral arteries, BA, and VA (V4) led to greater severity of vestibular and ataxic symptoms. According to the MMSE scale, the average score corresponded to mild cognitive reduction (CR) in Group I (25.5 ± 0.6 points) and mild dementia in Groups II and III (23.6 ± 0.2 and 20.9 ± 0.4 points, respectively). In Group III, these values bordered on moderate dementia. In the FAB test, moderate cognitive reduction CR with impaired praxis and spatial activity was noted in Group I, severe CR with impaired praxis and object function in Group II, and dementia in Group III.

Conclusions. In patients with decompensated CCI, hemodynamically significant stenoses of CA, often in combination with VA damage. The functional state of the AComA and PComA plays an important role in the development of clinical manifestations of CCI. Patients with compensated CCI showed a moderate cognitive deficit; those with subcompensated CCI demonstrated a severe cognitive deficit, while patients with decompensation and the consequences of IS exhibited signs of vascular dementia.

Keywords*: stenosing atherosclerosis, main arteries of the head, chronic cerebral ischemia, cerebral hemodynamics, collateral circulation, cognitive disorders.*

Introduction. Cerebrovascular pathology (CVP) remains one of the leading causes of mortality, as well as a major cause of disability and reduced quality of life [1]. Over the past 10-15 years, Ukraine has shown a steady increase in both the prevalence and incidence of CVP, particularly its chronic progressive forms, which is associated with the growing influence of various extreme factors. Many manifestations of CVP form clinical syndromes at an early stage, leading to social and everyday maladaptation and disability [2].

It is known that cognitive deficit accompanies chronic cerebral ischemia (CCI), represents a leading syndrome – especially during its long-term course – and has a significant negative impact on the rehabilitation process of patients with CVP [3].

These circumstances dictate the need for a rapid response to disease progression, timely diagnosis of vascular pathology of the central nervous system (CNS), and the development and implementation of modern, adequate – including surgical–treatment strategies [4,5].

Carotid artery disease is an important cause of ischemic stroke (IS). Extracranial disorders include atherosclerosis, pathological deformations, arteritis, fibromuscular dysplasia, arterial dissection, aneurysms, trauma, and post-traumatic changes of the carotid arteries (CA) [6]. The main areas of current research in carotid pathology are focused primarily on the development of new instrumental diagnostic methods that make it possible to detect CCI at early stages and to study its clinical course [7].

A significant number of studies are devoted to surgical treatment, including the determination of indications, techniques, and postoperative rehabilitation [8]. Most research addresses atherosclerotic lesions of the carotid arteries, while non-atherosclerotic forms remain largely neglected. This situation is explained by the predominant proportion of ischemic strokes of atherosclerotic origin and the much lower incidence of non-atherosclerotic diseases [9]. The management of atherosclerotic carotid artery lesions remains one of the most debated issues in vascular surgery. Ischemic stroke as a cause of mortality ranks third after cardiovascular and oncological diseases [10].

To determine treatment strategies for patients with atherosclerotic lesions of the carotid arteries, several randomized studies were conducted in the USA and Europe (NASCET, ACAS, ESCT), which demonstrated the advantages of surgical treatment of extracranial artery stenosis over conservative therapy [7,11]. The proven high efficacy of carotid endarterectomy (CEA) in preventing ischemic strokes has led to its widespread use in developed countries [11,12].

Alongside surgical interventions, endovascular procedures on the carotid arteries are being increasingly applied each year [13]. Atherosclerosis is characterised by its multifocal nature, most patients have lesions in three or more cerebral vascular basins [14]. The subclavian–vertebral steal syndrome plays an important role in patients with polysegmental lesions of the extracranial arteries [15]. Patients with combined lesions of the carotid and vertebral arteries (VA) show the most pronounced clinical manifestations of atherosclerotic lesions of the main arteries of the head (MAH) [16-18].

The assessment of cerebral hemodynamic compensation serves as an informative tool for evaluating the functional state of the vascular system and may help predict the course of this pathology, particularly in acute and chronic forms of cerebrovascular disease. It can also indicate the necessity for surgical intervention.

Aim. To study of the state of cerebral circulation and cognitive status in patients with cerebral ischemia on the background of atherosclerotic lesions of the brachiocephalic arteries.

Materials and Methods. 128 people aged 42 to 60 years (mean age 53.6 ± 1.6 years) were examined. including with CCI in compensated (Group I n=24) and subcompensated (Group II n=38) stages, as well as 46 patients (Group III) patients with decompensated CCI, who had a history of stroke.

The duration of ischemic stroke varied within the early recovery period. All patients had preserved speech and writing abilities. Informed consent was obtained from all participants for inclusion in the clinical observations.

The study of cerebral arteries was performed in triplex mode using an Ultima-PA ultrasound scanner (RADMYR, Ukraine). The structural and functional state of the common (CCA), external (ECA), internal (ICA), and VA (segment V2) was examined. The time-averaged maximum blood flow velocity (TAMX) was also measured in the siphons of the ICA, and in the anterior (ACA), middle (MCA), and posterior (PCA) cerebral arteries, as well as in the VA (segment V4) and basilar (BA) arteries.

The functional integrity of the anterior (AComA) and posterior (PComA) communicating arteries was assessed using the carotid compression test. The main hemodynamic parameters were compared among the three clinical groups.

All patients with identified stenotic processes of the MAH underwent carotid angiography in the neurosurgical department, followed by a decision regarding surgical intervention.

Neuropsychological testing was conducted using the Mini-Mental State Examination (MMSE) [19], a set of tests for assessing frontal lobe dysfunction – the Frontal Assessment Battery (FAB) [20], and a 10-word memorization test based on the method of A.R. Luria [21].

The control group (CG) included 20 clinically healthy volunteers of both sexes, matched for age.

For statistical analysis, the program «Statistica 8.0» was used, with evaluation performed using the Wilcoxon test and the Mann–Whitney U test. Differences were considered statistically significant at $p<0.05$.

Results. The study of hemodynamics in the extracranial segments of the CA and the VA revealed the following patterns: in Group I, lesions of the MAH

were noted in 41.7 % of patients, including isolated CA lesions – 59.6 %, isolated VA lesions – 27.1 %, and multiple lesions – 13.3 %; no hemodynamically significant stenoses were observed.

In patients of Group II, stenosing lesions were found in 84.2 % of cases, including isolated CA lesions – 18.7 %, isolated VA lesions – 12.5 %, multiple lesions – 53.1 %, and hemodynamically significant – 28.1 %.

In patients of Group III, stenosing lesions were noted in 95.6 % of cases, including isolated CA lesions – 13.6 %, isolated VA lesions – 6.8 %, multiple lesions – 79.5 %, and hemodynamically significant – 72.7 %.

In Group I, isolated lesions of the carotid or vertebral arteries were observed; in Group II, multiple lesions were much more common; and in Group III, these lesions reached their maximum extent with decompensation of blood flow. The differences obtained were statistically significant ($p<0.05$) (Figure 1).

The study of blood flow in the intracranial arteries showed that the severity of cerebrovascular insufficiency corresponded to a decrease in TAMX. Symptoms of organic damage to the nervous system increased from compensation to subcompensation, and were most pronounced in decompensation, with a decrease in TAMX values in the main intracranial arteries. The most reliable and significant parameter was the TAMX value in the MCA in Groups II and III ($p<0.05$). The severity of cerebrovascular insufficiency in the intracranial arteries correlated with a decrease in TAMX, with the most significant reduction observed in the MCA in Group II ($p<0.05$) and Group III ($p<0.01$). Such vascular ischemia was clinically accompanied by an increase in ataxic, pseudobulbar, dysmnestic, and pyramidal syndromes (Table 1).

We obtained data indicating a relationship between the decrease in TAMX in the artery supplying a given area and the severity of neurological symptoms. The prevalence of cognitive impairments, including decreased memory, intelligence, and behavioral changes, corresponded more closely to the reduction in TAMX in both anterior cerebral arteries when the AComA was non-functioning, with TAMX values of 34.6 ± 3.5 cm/s on the left and 37.2±3.2cm/sontheright.WhentheAComAwasfunctional, the degree of dysmnestic disorders was less pronounced, and TAMX values were 48.8 ± 4.2 cm/s on the left and 46.3 ± 5.1 cm/s on the right; these differences were statistically significant ($p<0.05$).

In cases of a non-functioning PComA, a decrease in TAMX in the PCA, BA, and intracranial segment of the VA led to more severe vestibular and ataxic symptoms. When the PComA was functional, TAMX in the PCA was 46.2 ± 3.6 cm/s; in its absence, TAMX decreased to 35.4 ± 5.1 cm/s. These differences were statistically significant ($p<0.01$).

The presence of pyramidal insufficiency correlated with a greater decrease in TAMX in the MCA on the side of the stroke (35.6 ± 7.1 cm/s) compared with patients at the same stage without a history of acute episodes ($p<0.05$).

During transcranial Doppler (TCD) assessment, the role of collateral circulation (CC) was evaluated by determining the functional status of the AComA and PComA (Table 2). The functional integrity of the AComA and PComA, the main collateral pathways, is critical in the development of clinical manifestations of ischemic brain injury (Table 2).

The prevalence of cognitive impairments, including decreased memory, intelligence, and behavioral changes,

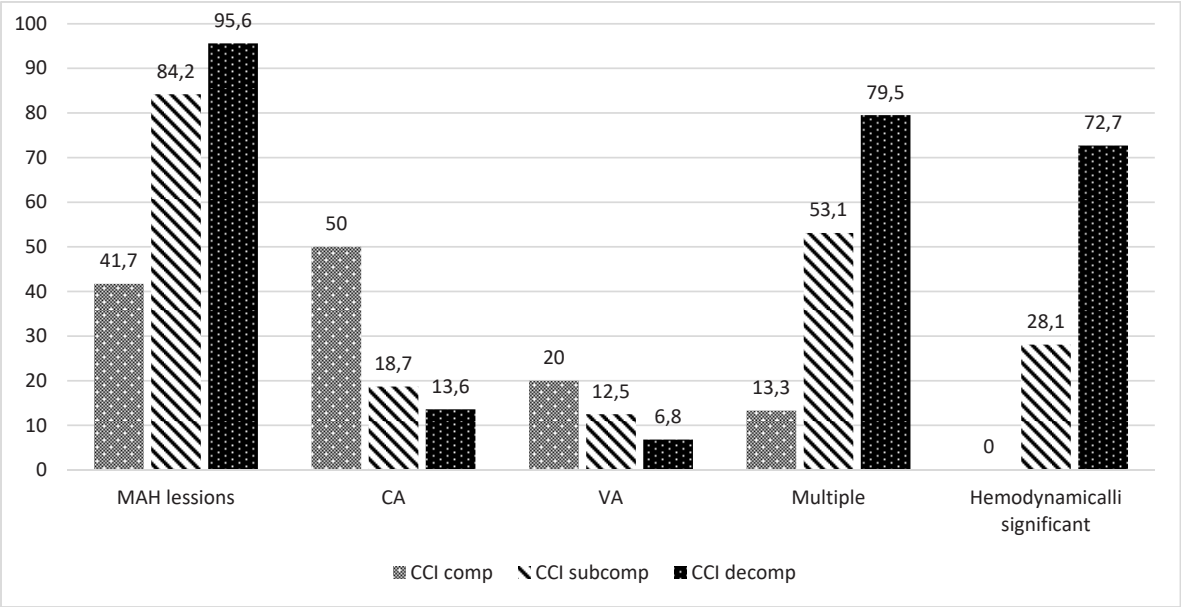

Figure 1. *Localization and degree of lesions of extracranial arteries with different compensation of cerebral circulation*

Table 1

TAMX values in cerebral arteries at different stages of CCI

	TAMX (cm/s)			
	CCI compensation	CCI subcompensation	CCI decompensation	CG
ICA (syph)	38.8 ± 2.3*	32.1 ± 2.7**	30.5 ± 2.2 **	48.1 ± 3.5
MCA	53.2 ± 3.4*	47.8 ± 2.5**	38.3 ± 3.6**	67.3 ± 5.2
ACA	44.3 ± 3.5	43.4 ± 4.3	29.8 ± 4.6 **	51.6 ± 4.8
PCA	40.4 ± 2.5	41.2 ± 3.0	31.4 ± 3.4 *	47.6 ± 4.8
VA	38.6 ± 4.2*	35.8 ± 4.2	26.2 ± 5.1 *	38.4 ± 5.5
BA	34.3 ± 2.8*	42.4 ± 4.1 *	32.1 ± 3.5	36.5 ± 4.4

Note: *p<0.05; **p<0.01

Table 2

Functional state of connecting arteries at different degrees of compensation of CCI

Connectors arteries	Functional status of the communicating arteries (%)		
	CCI compensation	CCI subcompensation	CCI decompensation
AComA 0	31.6	32.6	82.5
1	42.1	51.0	17.5
2	26.3	16.2	0
PComA S 0	68.4	74.5	90
1	31.6	25.5	10
PComA D 0	58.4	76.8	87.5
1	31.6	23.2	12.5

*Note**: not functioning – 0; functioning in one direction – 1; functioning in both directions – 2.

largely corresponded to a reduction in TAMX in both ACA when the AComA was non-functioning, with TAMX values of 34.6 ± 3.5 cm/s on the left and 37.2 ± 4.1 cm/s on the right. When the AComA was functional, the severity of dysmnestic disorders was less pronounced, and TAMX values were 48.8 ± 4.2 cm/s on the left and 46.3 ± 5.1 cm/s on the right; these differences were statistically significant (p<0.05).

In cases of a non-functioning PComA, a decrease in TAMX in the PCA, BA, and the intracranial segment of the VA led to more severe vestibular and ataxic symptoms. When the PComA was functional, TAMX in the PCA was 46.2 ± 3.6 cm/s, whereas in its absence it decreased to 35.4 ± 5.1 cm/s. These differences were statistically significant (p<0.01).

The presence of pyramidal insufficiency correlated with a more pronounced decrease in TAMX in the MCA on the side of the stroke (35.6 ± 7.1 cm/s) compared with patients at the same stage without acute episodes.

According to the results of the Luria 10-word memorization test, there was a tendency toward decreased performance in all patient groups, depending on the severity of CNS damage (p<0.05). In the control group, the average test scores at all stages of word presentation were significantly higher than those in all CCI groups. In Group I, a similar tendency was observed, with indicators significantly higher than in Groups II and III. Significant fluctuations in performance were also noted depending on the stage of ischemic brain injury, including impaired selectivity and reduced volumes of immediate and delayed recall (Figure 2).

According to the obtained data on the MMSE scale, the average scores indicated mild cognitive impairment in Group I (25.5 ± 0.6 points) and mild dementia in Groups II and III (23.6 ± 0.2 and 20.9 ± 0.4 points, respectively). In Group III, these values approached the range of moderate dementia, with decreases observed across all items of the mental status assessment. These deficits reflect vascular dementia, which predominated in some cases. In Group I, the reductions in MMSE scores were primarily due to impairments in attention, copying, and similar tasks, whereas in Groups II and III, memory, sequencing of commands, and other domains were additionally affected.

Analysis of the average scores on the FAB test revealed the following patterns: Group I demonstrated moderate cognitive impairments with deficits in praxis and spatial activity; Group II exhibited severe cognitive impairments with reduced control over activity, deficits in programming, praxis, and object function; and Group III showed signs of dementia with decreased central nervous system functioning across all categorical generalizations.

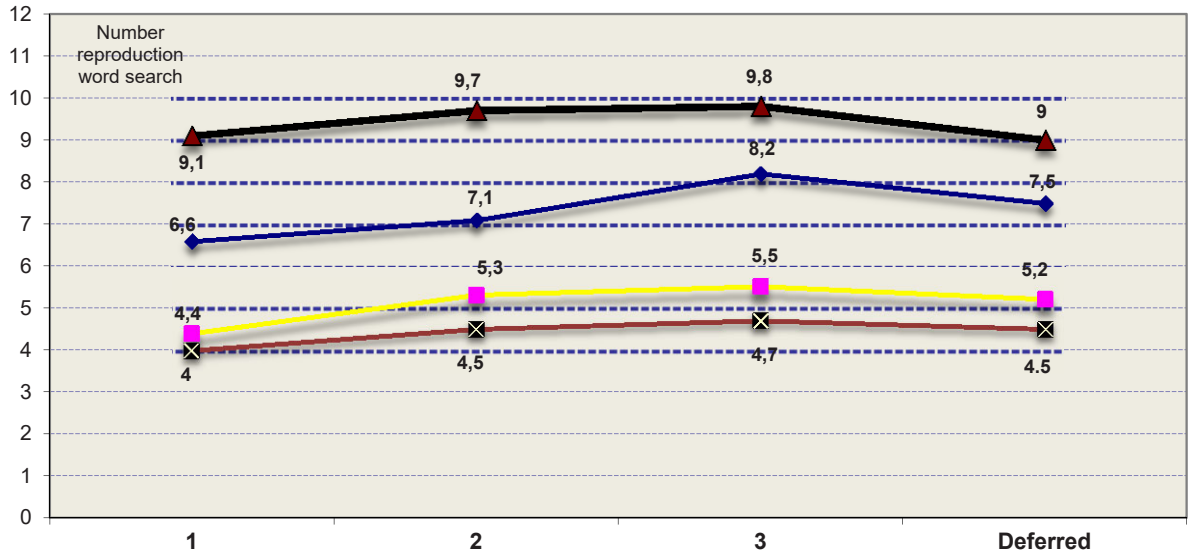

Figure 2. *Dynamics of the average number of memorizations of 10 words in three (short-term) and delayed (long-term memory) presentations in patients in three groups, depending on the compensation of ischemic processes and control. From top to bottom CG, I Group, II Group, III Group.*

Discussion. The increase in the severity of cerebrovascular insufficiency in Groups II and especially III is due not only to the degree and localization of extracranial arterial lesions, but also to a reduction in the functional capacity of collateral circulation (i.e., insufficiency of intracerebral compensation). We observed a tendency for average TAMX values along the main intracranial arteries to decrease with increasing severity of cerebrovascular insufficiency. Alongside the degree and localization of extracranial lesions in CCI, the state of CC plays a critical role. The appearance of clinical symptoms unrelated to the primary affected vascular basin can most likely be explained by the formation of CC and intracranial redistribution of blood flow.

When assessing intracranial blood circulation and the degree of extracerebral arterial stenosis, it was noted that progression of the latter does not always result in reduced blood flow in the corresponding intracerebral basin. Only pronounced, hemodynamically significant stenoses lead to a decrease in blood flow. With further progression of stenosis, there is a parallel reduction in both blood flow and intravascular pressure. These findings are consistent with the results reported by other authors [22-24].

Examination of cerebral circulation changes revealed that lesions of the MAH increased with the severity of ischemia, particularly in cases of multiple hemodynamically significant stenoses, and were accompanied by an escalation of ataxic, pseudobulbar, dysmnestic, and pyramidal syndromes. In such cases, assessment of TAMX in specific vascular basins must take into account the state of the connecting arteries, the presence of previous strokes or acute episodes, collateral circulation, and the insufficiency of cerebral vascular compensation.

In this context, memory disorders are observed, the prevalence of which during the recovery period after brain ischemia represents a key pathogenetic factor in the manifestation of residual organic and, consequently, functional lesions of the brain parenchyma. In the course of our studies, it was found that cognitive deficits take a leading role in the clinical picture, requiring careful diagnosis for timely correction and prevention of adverse effects on rehabilitation. Cognitive deficits and the development of neuropsychological syndromes are the most common manifestations of cerebral circulatory insufficiency.

Significant fluctuations in performance on the Luria 10-word memorization test were also observed, depending on the stage of ischemic CNS damage, reflecting a narrowing of the volumes of immediate and delayed word recall, both within patient groups and in comparison, with normative indicators. According to MMSE data, the formation of vascular dementia could be traced across the groups.

Analysis of cognitive function in patients with CCI of varying degrees of compensation revealed that with increasing ischemia, more severe impairments occurred in praxis and its control, object function, and short-term and long-term memory, ultimately leading to the development of vascular dementia.

Conclusions

1. In the group with compensated CCI, isolated lesions of the carotid or vertebral arteries were observed; in the group with subcompensated CCI, multiple lesions were much more common; and in patients with decompensated CCI, hemodynamically significant stenoses of the ICA and CCA predominated, often in combination with VA damage.
2. The functional state of the AComA and PComA is important in the development of clinical manifestations of cerebrovascular insufficiency.

3. Cognitive deficit comes first in the clinical picture of CCI. Neuropsychological syndromes are the most common manifestations of cerebral circulatory insufficiency.
4. The progression of cognitive deficits correlates with the stage of ischemic CNS damage.
5. Patients with compensated CCI exhibited moderate cognitive deficits affecting praxis and spatial activity; those with subcompensated CCI demonstrated severe cognitive deficits, including impaired control over activity and praxis; and patients with decompensated CCI or post-ischemic stroke sequelae showed signs of vascular dementia.

Prospects for Further Research. A promising direction for future research is the development of strategies for the pharmacotherapeutic correction of cognitive disorders, taking into account the functional state of the vascular system and the mechanisms of cerebral autoregulation.

Compliance with Ethical Standards. The study was conducted in accordance with the Declaration of Helsinki and approved by the Bioethics Commission of Odessa National Medical University.

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Primary Data and Materials: Data available upon reasonable request

The personal contribution of each author to the work.

Kalashnikov V.Y.: idea, goal, collection of research material, analysis of the results obtained; preparation of the text of the article; **Stoyanov O.M.:** idea, collection of research material, analysis of the results obtained; **Vastyanov R.S.:** analysis of the obtained results, collection of research material, analysis of the obtained results; **Andreeva T.O.:** collection of research material, translation into English; **Kugel Y.I.:** collection of research material, preparation of the work for publication.

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Особливості церебральної гемодинаміки та когнітивного стану у пацієнтів з хронічною ішемією мозку на тлі стенозуючого атеросклерозу брахіоцефальних артерій

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Резюме

Мета. Дослідити стан мозкового кровообігу та когнітивний статус (КС) у пацієнтів із хронічною ішемією мозку (ХІМ) при атеросклеротичних ураженнях брахіоцефальних артерій (БЦА).

Матеріали та методи. Обстежено 128 осіб віком від 42 до 60 років, серед яких пацієнти з ХІМ у компенсованій (Група I, n=24) та субкомпенсованій (Група II, n=38) стадіях, а також 46 пацієнтів (Група III) з декомпенсованою ХІМ, які перенесли ішемічний інсульт (ІІ). Оцінювали стан сонних (СА) та хребтових (ХА) артерій, а також усереднену за часом максимальну швидкість кровотоку (ТАМХ) у церебральних артеріях, функціонування передньої (ПСоА) та задньої (ЗСоА) сполучних артерій. Когнітивний стан оцінювали за допомогою тесту Mini-Mental State Examination (MMSE), для оцінки фронтальної дисфункції – Frontal Assessment Battery (FAB) та тесту на запам'ятовування 10 слів. Контрольну групу (КГ) склали 20 клінічно здорових добровольців.

Результати. У групі I спостерігалися ізольовані ураження СА або ХА, у групі II та III домінували множинні ураження. Вираженість ХІМ відповідала зниженню ТАМХ, найбільше в СМА у групі II ($p<0,05$) та групі III ($p<0,01$). Судинна ішемія супроводжувалася атактичним, псевдобульбарним, дисмнестичним та пірамідним синдромами. У пацієнтів із нефункціонуючою ПСоА при стенозах БЦА спостерігалося зниження пам'яті та інтелекту, поведінкові зміни. У випадках нефункціонуючої ЗСоА зниження ТАМХ у ЗМА, БА та ХА (V4) призводило до більшої вираженості вестибулярних та атактичних синдромів. За шкалою MMSE середній бал відповідав легкому когнітивному зниженню (КЗ) у групі I (25,5 ± 0,6 бала) та легкій деменції у групах II і III (23,6 ± 0,2 та 20,9 ± 0,4 бала відповідно). У групі III ці показники наближалися до помірної деменції. За

тестом FAB у групі I відзначалося помірне КЗ з порушенням праксису та просторової активності, у групі II – важке КЗ з порушенням праксису та функції об'єктів, а у групі III – деменція.

Висновки. У пацієнтів із декомпенсованою ХІМ переважали гемодинамічно значущі стенози КА, часто у поєднанні з ураженням ХА. Функціональний стан ПСоА та ЗСоА відіграє важливу роль у розвитку клінічних проявів ХІМ. Пацієнти з компенсованою ХІМ мали помірний когнітивний дефіцит, пацієнти з субкомпенсованою ХІМ демонстрували тяжкий дефіцит, пацієнти з декомпенсацією та наслідками II мали ознаки судинної деменції.

Ключові слова: *стенозуючий атеросклероз, магістральні артерії голови, хронічна церебральна ішемія, мозкова гемодинаміка, колатеральний кровообіг, когнітивні порушення*

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