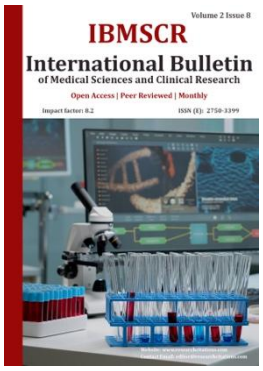

DYNAMICS OF CLINICAL AND LABORATORY PARAMETERS IN THE POST-STROKE PERIOD OF OUTPATIENT MONITORING

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Abstract. Ischemic stroke remains one of the leading causes of disability and mortality. In a study of 287 patients, it was shown that the recovery of functional abilities depends not only on clinical factors but also on social conditions. In the early period, pronounced neurological deficits and multiple cognitive impairments were observed. Changes in hemostasis, elevated levels of inflammatory markers (sICAM-1, MPO, sE-selectin), and increased platelet adhesion were identified, which increased the risk of recurrence. The use of a combination of acetylsalicylic acid and clopidogrel reduced the risk of recurrent stroke, while the addition of TMS and choline alphoscerate increased rehabilitation effectiveness by 1.5 times. Secondary prevention includes correction of risk factors and lifestyle modification. **Objective:** Optimization of outpatient rehabilitation after ischemic stroke through the assessment of clinical-biochemical changes and blood parameters at different stages of recovery. **Materials and Methods.** The study was conducted in 216 patients during the recovery period after atherothrombotic and lacunar stroke of mild and moderate severity. To assess inflammatory processes associated with endothelial dysfunction, levels of MPO, sICAM-1, and sE-selectin were measured. Rehabilitation included individualized application of TMS and alfoscerate to correct cognitive impairments and prevent recurrence. **Results.** Neurological deficits were assessed using the NIHSS scale, and adaptation was evaluated using the Barthel Index, where the 3rd subgroup showed 1.5-fold higher scores ($p<0.01$). Cognitive impairments predominated: in young patients, ACE-R scores increased (81.3→90.9), in middle-aged patients scores decreased (79.6→72.3), and in elderly patients the decline was more pronounced (70.7→67.1). A unilateral increase in MCA blood flow was observed. In the 3rd subgroup, sICAM-1, MPO, and sE-selectin levels initially decreased, but in the decompensation phase increased: in the 1st group by 1.5 times, in the 3rd group by 2.3 times ($p<0.001$). The highest sICAM-1 values were recorded in the comparative group. **Conclusions.** In both early and late recovery periods, functional recovery was 1.5 times higher ($p<0.05$), although no association with hemostasis was found. The main risk factors for recurrent stroke were type 2 diabetes mellitus, grade 3 arterial hypertension, metabolic syndrome, endothelial dysfunction, and increased platelet adhesion. In 32.1% of patients, platelet activity persisted despite ASA, while combination with clopidogrel reduced the risk by 1.5 times ($p<0.05$). Platelet adhesion was elevated in 66% in the early phase, 56% in the late phase, and 71% in recurrent stroke, requiring individualized antiplatelet therapy. TMS and choline alfoscerate increased rehabilitation effectiveness by 1.5 times, while prevention included risk factor correction and lifestyle modification.

Keywords. MPO, sICAM-1, sE-selectin, TMS, stroke.

Резюме: Ишемический инсульт продолжает оставаться одной из ведущих причин инвалидизации и смертности. В исследовании 287 пациентов показано, что

восстановление функциональных способностей зависит не только от клинических факторов, но и от социальных условий. В ранние сроки наблюдался выраженный неврологический дефицит и множественные когнитивные нарушения. Были выявлены изменения гемостаза, повышение уровня воспалительных маркёров (sICAM-1, MPO, sE-selectin) и адгезии тромбоцитов, что увеличивало риск рецидива. Применение комбинации ацетилсалициловой кислоты и клопидогреля снижало вероятность повторного инсульта, а добавление ТМС и холин-альфостерата увеличивало эффективность реабилитации в 1,5 раза. Вторичная профилактика предполагает коррекцию факторов риска и изменение образа жизни. **Цел;** Оптимизация амбулаторной реабилитации после ишемического инсульта с использованием оценки клинико-биохимических изменений и показателей крови на разных этапах восстановления **Материалы и методы;** Исследование проведено у 216 пациентов в восстановительный период после атеротромботического и лакунарного инсульта лёгкой и средней тяжести. Для оценки воспалительных процессов, связанных с эндотелиальной дисфункцией, определялись уровни MPO, sICAM-1 и sE-селектина. Реабилитация включала индивидуализированное применение ТМС и альфаксолинэстерата для коррекции когнитивных нарушений и профилактики рецидива. **Результаты.** По шкале NIHSS оценивалась выраженность дефицита, по Бартел — уровень адаптации, который у 3-й подгруппы был в 1,5 раза выше ($p<0,01$). При инсульте преобладали когнитивные нарушения: у молодых по ACE-R отмечался рост баллов (81,3→90,9), у среднего возраста — снижение (79,6→72,3), у пожилых — ещё более выраженное (70,7→67,1). Выявлено одностороннее повышение ВКА. У 3-й подгруппы уровни sICAM-1, MPO и sE-selectin первоначально снижались, но в фазе декомпенсации возрастали: в 1-й группе — в 1,5 раза, в 3-й — в 2,3 раза ($p<0,001$). Наибольшие значения sICAM-1 отмечены в сравнительной группе. **Заключения.** Анализ показал, что в раннем и позднем восстановительных периодах уровень функционального восстановления был выше в 1,5 раза ($p<0,05$), однако связи с гемостазом не выявлено. Основные факторы риска повторного инсульта: СД 2 типа, АГ 3 степени, метаболический синдром, эндотелиальная дисфункция и повышенная адгезия тромбоцитов. У 32,1% сохранялась тромбоцитарная активность на фоне АСК, тогда как комбинация с клопидогрелом снижала риск в 1,5 раза ($p<0,05$). Повышение адгезии тромбоцитов отмечено у 66% в раннем, 56% — в позднем и 71% — при повторном инсульте, что требует индивидуальной антиагрегантной терапии. Применение ТМС и холин-альфостерата повышало эффективность реабилитации в 1,5 раза, а профилактика включала коррекцию факторов риска и образа жизни.

Ключевые слова. MPO, sICAM-1 и sE-селектина, ТМС, инсульт.

Резюме. Ишемик инсульт ҳали ҳам ногиронлик ва ўлим сабабларидан бири сифатида устувор аҳамиятга эга. 287 нафар беморни қамраб олган тадқиқотда кўрсатилишича, функционал қобилиятларнинг тикланиши фақат клиник омилларга эмас, балки ижтимоий шарт-шароитларга ҳам боғлиқ. Эрта даврда қонли неврологик дефицит ва кўп функцияли когнитив бузилишлар кузатилди. Гемостазда ўзгаришлар, яллиғланиш маркерлари (sICAM-1, MPO, sE-selectin) ва тромбоцитларнинг адгезияси даражасининг ошиши аниқланди, бу эса рецидив хавфини кўпайтирди. Ацетилсалицил кислота ва клопидогрел комбинациясини қўллаш такрорий инсульт хавфини

камайтирди, ТМС ва холин-альфостерат қўшилиши эса реабилитация самарадорлигини 1,5 бараварга оширди. Иккинчи даражали профилактика омилларини тўғрилаш ва турмуш тарзини ўзгартиришни ўз ичига олади**. Мақсад:** Ишемик инсультдан кейинги амбулатор реабилитацияни турли даврларда клинико-биохимик ўзгаришлар ва қон кўрсаткичларини баҳолаш орқали оптимизация қилиш. **Материал ва методлар**. Тадқиқот атеротромботик ва лакунар инсультдан кейинги тикланиш даврида бўлган енгил ва ўрта оғирликдаги 216 беморда ўтказилди. Эндотелий дисфункцияси билан боғлиқ яллиғланиш жараёнларини баҳолаш учун MPO, sICAM-1 ва sE-selectin даражалари аниқланди. Реабилитация индивидуал равишда ТМС ва альфаксолинэстерат қўллаш орқали когнитив бузилишларни тузатиш ва рецидивнинг олдини олишга қаратилди. **Натижалар.** NIHSS шкаласи бўйича неврологик дефицит, Бартел шкаласи бўйича эса адаптация баҳоланди; 3-подгруппа беморларида адаптация даражаси 1,5 марта юқори бўлди ($p<0,01$). Инсультда кўп функцияли когнитив бузилишлар устун келди: ёшларда ACE-R баллари ўсди (81,3→90,9), ўрта ёшларда пасайиш кузатилди (79,6→72,3), кексаларда эса янада кучли пасайиш қайд этилди (70,7→67,1). Бир томонлама ВКА ўсиши аниқланди. 3-подгруппада sICAM-1, MPO ва sE-selectin бошланғичда пасайди, аммо декомпенсация фазасида: 1-подгруппада 1,5 мартага, 3-подгруппада эса 2,3 мартага кўтарилди ($p<0,001$). Энг юқори sICAM-1 даражалари қиёсий гуруҳда кузатилди. **Хулосалар.** Илк ва кеч тикланиш даврларида функционал тикланиш 1,5 марта юқори бўлди ($p<0,05$), лекин гемостаз кўрсаткичлари билан боғлиқлик топилмади. Қайталанувчи инсульт хавфининг асосий омиллари: 2-тур қандли диабет, 3-босқич артериал гипертония, метаболик синдром, эндотелий дисфункцияси ва тромбоцит адгезиясининг ошиши. 32,1% беморда АСК фонида тромбоцит фаоллиги сақланиб қолди, АСК + клопидогрел комбинацияси эса хавфни 1,5 марта камайтирди ($p<0,05$). Тромбоцит адгезияси 66% — илк, 56% — кеч даврда ва 71% — қайталанувчи инсультда юқори бўлиб, индивидуал антиагрегант терапия зарурлигини кўрсатди. ТМС ва холин-альфостерат реабилитация самарадорлигини 1,5 марта оширди, профилактика эса хавф омиллари ва турмуш тарзини тузатишни ўз ичига олди.

Калит сўзлар: MPO, sICAM-1, sE-selectin, ТМС, инсульт.

Introduction: Acute cerebral circulatory disorders remain one of the leading causes of death and disability, second only to cardiovascular diseases. According to WHO, about 15 million people experience a stroke every year.: 11% of them die in the first year, 18% experience a second stroke, and 12-14% develop cognitive impairments leading to disability. Between 2000 and 2016, mortality from chronic cerebral circulatory disorders increased by 42%, and from stroke — by 19%.

Stroke is accompanied by severe neurological and cognitive disorders that significantly impair the quality of life. The effectiveness of rehabilitation is determined not only by the clinical condition, but also by the level of psychosocial adaptation of the patient. At the same time, standard rehabilitation programs solve this problem only partially. The consequences of a stroke affect not only the patient himself, but also his family, requiring significant physical and emotional efforts to care for patients with impaired motor function.

The aim of the study: was to optimize outpatient rehabilitation of patients after ischemic stroke, taking into account clinical and biochemical changes and the dynamics of laboratory blood parameters at various stages of recovery.

Materials and methods: The study included 287 patients with ischemic stroke who were admitted to the Department of Emergency Neurology of the Bukhara branch of the Russian National Research Center and were observed on an outpatient basis. The main group consisted of 216 patients (average age 61.5 ± 2.8 years; men — 52.7%, women — 47.3%), who were divided into three subgroups depending on the time elapsed after the stroke: 1-3 months (n = 72), 3-6 months (n = 81) and 6-12 months (n = 63The comparison group consisted of 71 patients with recurrent stroke, and the control group consisted of 48 healthy volunteers.

According to the WHO classification (2021), participants were divided into age categories: young (18-44 years old), middle-aged (45-59 years old) and elderly (60-74 years old). The main group was dominated by elderly patients (48.6%), while in the comparison group 12.9% were young, 50% were middle—aged and 37.1% were elderly. Elderly patients were not included in the study.

Results: During clinical and biochemical blood analysis in patients with ischemic stroke on an outpatient basis, risk factors for neurological disorders, as well as features of the clinical, neurological and psycho-emotional state, were studied. Hemiparesis was the most common subjective symptom in all groups. Speech disorders (aphasia, dysarthria), cognitive disorders, hemianopia, nystagmus, hypesthesia of half of the face, nasopharyngeal asymmetry, dysphagia, tongue deviation, hemihypesthesia, anisoreflexia, pathological and oral automatic reflexes, static and dynamic ataxia, headache, dizziness, tinnitus and sleep were significantly more common in the main group ($p < 0.001$).

In the post-ischemic period, the dynamics of neurological symptoms according to the NIHSS scale was observed. In the 1st subgroup, moderate disorders were 1.5 times more common than mild or pronounced changes ($p < 0.01$). In the 2nd subgroup, the average violations prevailed with an excess of 3.5 times over the expressed ones ($p < 0.001$). In the 3rd subgroup, the degree of neurological deficit was the greatest, on average exceeding the pronounced changes by 3.8 times ($p < 0.001$) (Fig. 1). In the main group, the average NIHSS deficit was 11.09 ± 0.1 points.

According to the NIHSS scale, the severity of the deficiency depended on the duration of the stroke, according to Barthel, the level of adaptation. In the 3rd subgroup, adaptation was 1.5 times higher than in the 1st and 2nd ($p<0.01$).

In ischemic stroke, multifunctional cognitive disorders prevailed. According to the ACE-R scale, young patients showed an increase in scores: 81.3 in the comparison group, 83.3 in the 1st n/A group, 86.8 in the 2nd, and 90.9 in the 3rd. In the middle age, the indicators were lower: 79.6; 75.1; 79.0 and 72.3, respectively. The most pronounced decrease was found in elderly patients: 70.7; 78.8; 70.4 and 67.1 (табл. 1).

Table 1

Assessment of cognitive functions on the modified Addenbrooke's scale (in points)

Young age (18-44)					
Cognitive domains	Stan dard	Comparative group (n=71)	The main group		
			The main group	group (n=71)	The main group
Attention	18	15,2±0,2	14,2±0,2	15,5±0,2	15,9±0,3

Memory	26	21,1±0,3	21,3±0,3	22,2±0,3	23,2±0,4*
The speed of verbal associations	14	11,5±0,16	12,6±0,17	12,9±0,2	13,1±0,2*
Speech	26	21,4±0,3	21,8±0,3	22,3±0,3	24,6±0,4*
Visual-space functions	16	12,1±0,17	13,4±0,18	13,9±0,2	14,1±0,2*
Total	100	81,3±1,14	83,3±1,16	86,8±1,07	90,9±1,4*
Average age (45-59)					
Cognitive domains	Stan dard	Comparative group (n=71)	The main group		
			1-n/group (n=72)	2- /group (n=81)	3-n/group (n=63)
Attention	18	13,1±0,2	13,2±0,2	13,5±0,17	13,9±0,22
Memory	26	20,3±0,3	21,6±0,3	22,6±0,3	22,9±0,36
The speed of verbal associations	14	12,9±0.17	11,4±0,15	12,5±0,15	13,1±0,2
Speech	26	17,9±0,24	18,3±0,25	19,1±0,2	19,2±0,3
Visual-space functions	16	12,4±0,17	10,6±0,14	11,3±0,13	11,8±0,2
Total	100	79,6±1,1	75,1±1,0	79±0,9	72,3±1,14*
The elderly (60-74)					
Cognitive domains	Stan dard	Comparative group (n=71)	The main group		
			1-n/group (n=72)	2- /group (n=81)	3-n/group (n=63)
Attention	18	13,2±0,2	13,1±0,2	14,7±0,18	11,2±0,2*
Memory	26	19,1±0,3	21,2±0,3	18,2±0,22	17,1±0,3*
The speed of verbal associations	14	9,5±0,13	11,1±0.15	9,8±0,12	9,5±0,1
Speech	26	17,3±0,24	21,1±0,3	18,9±0,23	19,0±0,3
Visual-space functions	16	11,3±0,15	12,3±0,2	11,1±0,13	10,3±0,2
Total	100	70,7±1,0	78,8±1.09	70,4±0.8	67,1±1.06*
Note:	significant relative to the comparative group (*- p<0.05)				

According to Table 1, cognitive deficits in the main group of patients significantly differed from the comparison group, as well as differed between age categories. There was a tendency to improve cognitive functions with increasing age and duration of the post-stroke period. Motor aphasia in patients of the 1st subgroup was 1.5 times more common than alexia in other groups ($p<0.01$). In the 3rd subgroup, agraphy was combined with acalculia, the frequency of which was 2 times higher ($p<0.05$). Speech disorders varied significantly: in the 1st subgroup, up to 60% of patients had aphasia and dysarthria, in the 2nd - from 30 to 50%, in the 3rd - 15-30%. Patients in the 3rd subgroup showed significant improvement in NIHSS, Barthel index, and ACE-R scores compared with the 1st and 2nd subgroups ($p<0.05$).

Significant correlations with concomitant pathology were found in the main and comparative groups: the presence of comorbid diseases increased the risk of recurrent stroke and coronary heart disease. The state of the hemostasis system in stroke depended on the volume of brain damage, premorbid coagulopathies, and concomitant pathology. Coagulogram analysis showed significant differences in antithrombin III activity: increased activity of the hemostatic system was observed in the 1st subgroup. The predominance of hypercoagulation was considered as a marker of ongoing ischemia and an increased risk of its recurrence. In the comparison group, signs of hypercoagulation were 2.5 times more common than in the main group ($p<0.05$), which is explained by repeated stroke and the need for inpatient treatment. The most pronounced changes in hemostasis towards hypercoagulation were observed in atherothrombotic stroke — 1.5 times more often than in lacunar stroke ($p<0.05$).

In lacunar stroke, milder and more localized coagulation changes were observed, which were quickly compensated by the activation of compensatory mechanisms. Statistically significant differences between lacunar and atherothrombotic strokes were revealed by the prothrombin index and blood clotting time. The analysis of inflammatory parameters showed an increase in the level of C-reactive protein and fibrinogen, which confirms the active involvement of inflammatory processes in the pathogenesis of ischemic stroke (Table 2).

Table 2

Average rates of inflammatory symptoms in patients depending on the course of ischemic stroke

Markers of inflammation	The main group			Comparative group (n=71)
	1-n/group (n=72)	2- /group (n=81)	3-n/group (n=63)	
White blood cells 4-9 × 10^9/l.				
Monocytes, 3-10%				
Lymphocytes, %				
Rod-shaped neutrophils, 1-4%				
Segmented neutrophils, 40-70%	73,0±1,01*	52,0±0,6**	61,75±0,9	65,92±0,92
C-reactive protein, 5 mg/L. gacha	10,88±0,1	10,3 ± 0,1	8,09± 0,1*	10,88±0,15
Fibrinogen, 2-4 g/l	4,67±0,17	4.1 ± 0,4	3,74±0,06*	4,67±0,06

Note: significant in relation to the comparative group (*- $p<0,05$, **-$p<0,01$)

The main group had lower levels of total cholesterol, LDL and atherogenicity coefficient compared to the comparison group. Patients in the comparison group had elevated LDL (3.73±0.05 mmol/l), hypercholesterolemia (5.34±0.11 mmol/l) and atherogenicity coefficient (3.69±0.05), which indicates a more pronounced dyslipidemia compared with the main group (Table 3).

Table 3

Average values of lipidogram parameters in patients with ischemic stroke

Lipidogram indicators	The main group	Comparative

	1-n/group (n=72)	2- /group (n=81)	3-n/group (n=63)	group (n=71)
Total cholesterol, mmol/l 5.2 mmol/l	6,4±0.08	5,2±0.06*	4,92±0.07	6,7±0.09
LDL, 3.0 mmol/l gacha	3,53±0,04	3,33±0,11	2,86±0,21*	3,73±0,05
HDL, 1.0-1.5 mmol/l	1,14±0,35	1,19±0,35	1,33±0,36*	1,04±0,01
Total triglyceridyl, 0.4-1.7 mmol/L	1,61±0,72	1,13±0,72	0,98±1,10*	1,72±0,02
Coefficient of atherogenicity	3,69±0,04	3,08±0,14	2,93±0,21*	3,9±0,0.5

Premicalization:- significant in relation to the comparative group (*- p<0,05)

When assessing vascular inflammation, an increase in the level of myeloperoxidase was noted in all groups, which indicates the persistence of oxidative stress. In the comparison group, the concentrations of sICAM-1, MPO, and sE-selectin were 1.5 times higher than in the 3rd subgroup (p<0.05), (Table 4).

Table 4

Indications for the use of intracellular adhesion markers in patients who have undergone AI

Prescription of AI	The main group			Comparative group (n=71)
	1-n/group (n=72)	2- /group (n=81)	3-n/group (n=63)	
sISAM-1, 150-450 ng/ml	557,0 ±7,7	531,0 ±6.5*	498 ±7,9**	587,0 ±8,2
MPO, 0-469 ng/ml	547,3 ±7,6	531,7 ±6,5*	487,8±6,01**	577,2 ± 8,1
sE-selectin, 58-70 ng/ml	152,8±2,1*	131,0±1,6*	114,0±1,8**	167,3±2,4
Decrease:	significant relative to the comparative group (*- <0.05, **- p<0.01)			

Elevated levels of myeloperoxidase (MPO) reflect neutrophil activation, increased endothelial dysfunction, hypercoagulation, and progression of atherothrombosis. In the 1st subgroup, during the compensation period, a decrease in the concentrations of sICAM-1, MPO and sE-selectin was observed, whereas in the decompensation phase, their growth acquired a pathological character. The ACA analysis revealed a unilateral increase: in patients of the 3rd subgroup, the values decreased in the early stages, but in the decompensation phase, the levels of sICAM-1 increased 1.5-2.3 times (p<0.001). In the comparison group, the concentration of sICAM-1 was significantly higher.

High levels of sICAM-1, MPO, and sE-selectin correlated with the severity of neurological deficits on the NIHSS scale and decreased functionality on the Barthel index, indicating a pronounced activity of inflammation and brain damage. Vascular blood flow analysis most often revealed stenosis of the right internal carotid artery, less often — stenosis of the right and left common carotid arteries.

The effectiveness of rehabilitation was assessed using TMS and choline alfosterate (OGr-1) and standard therapy (OGr-2). With comparable baseline values (2-3 points), significant positive dynamics was noted at the outpatient stage. According to the Barthel index, mobility increased in all groups, while the increase in OGp-1 (+3.3%; $p<0.01$) was more significant than in OGp-2 (+2.7%; $p>0.05$).

In patients with different duration of stroke, rehabilitation using TMS and choline-alphaesterate showed a more pronounced positive effect than standard treatment (OGr-2, $p<0.05$). In the OGp-1 group, there was a significant improvement in cognitive functions on the ACE-R scale compared to the control indicators.

In patients who had suffered a stroke, according to the Barthel scale, the indicators increased from 69.9±0.86 to 89.3±0.7 ($p<0.001$). In OGr-1, sICAM-1 levels decreased from 557.9±0.1 to 479.7±0.09 ng/ml, sE-selectin from 152.8±0.05 to 114.1±0.03 ng/ml, MPO from 547.3±0.7 to 472.3 ng/ml. In OGr-2: sICAM-1 from 531.9±0.1 to 479.1±0.09 ng/ml, sE-selectin from 131±0.04 to 111±0.03 ng/ml, MPO from 531.7±0.5 to 472.3 ng/ml. In the 3rd subgroup: sICAM-1 from 498.7±0.1 to 467.9±0.1 ng/ml, sE-selectin from 114±0.03 to 90±0.02 ng/ml, MPO from 505.3±0.8 to 470.3±0.9 ng/ml. A decrease in biomarkers indicated the effectiveness of therapy and a decrease in platelet activity and hemostasis. In the comparison group, the effect was 1.5 times lower, reflecting the presence of chronic inflammation.

In OGr-2, sICAM-1 levels decreased from 567.2±0.2 to 538.1±0.1 ng/ml, sE-selectin from 151.9±0.05 to 139.5±0.05 ng/ml, MPO from 558.8±0.7 to 521.7±0.7 ng/ml. This reflected the hyperactivity of platelets and hemostasis before treatment and the lower effectiveness of rehabilitation compared to OGp-1.

Elevated levels of myeloperoxidase (MPO) reflect neutrophil activation, increased endothelial dysfunction, hypercoagulation, and progression of atherothrombosis. In the 1st subgroup, during the compensation period, a decrease in the concentrations of sICAM-1, MPO and sE-selectin was observed, whereas in the decompensation phase their growth assumed a pathological character. In patients of the 3rd subgroup, the values of sICAM-1 decreased in the early stages, but during decompensation they increased 1.5-2.3 times ($p<0.001$), while in the comparison group the concentrations of sICAM-1 were significantly higher.

High levels of sICAM-1, MPO, and sE-selectin correlated with the severity of neurological deficits on the NIHSS scale and decreased functional activity on the Barthel index, indicating a pronounced inflammatory process and brain damage. Most often, vascular blood flow analysis revealed stenosis of the right internal carotid artery, less often — stenosis of the right and left common carotid arteries.

The effectiveness of rehabilitation was assessed using TMS and choline alfosterate (OGr-1) and standard therapy (OGr-2). With comparable baseline values (2-3 points), significant positive dynamics was observed at the outpatient stage. Mobility according to the Barthel index increased in all groups, while the increase in OGp-1 (+3.3%; $p<0.01$) was more significant than in OGp-2 (+2.7%; $p>0.05$).

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