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Mechanisms of cognitive impairment in children after coronavirus infection

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ABSTRACT. ***Introduction.*** Identification of the causes of cognitive impairment, early diagnosis, prediction, and rehabilitation remain important tasks in pediatric practice. ***The aim*** — to investigate the mechanisms underlying the development of cognitive impairment and to identify risk factors in children who have had mild to moderate COVID-19. ***Materials and methods.*** The study included 147 children aged 7–18 years who had SARS-CoV-2 infection. Group 1 comprised 22 children with cognitive impairment, while Group 2 included 125 children without impairment. The groups were comparable in terms of age and sex. Cognitive impairment was assessed using the syndrome-based factor analysis of higher cortical functions proposed by A.R. Luria. The frequency and severity of asthenic syndrome were evaluated using the Malkova–Chertova scale (modified for adolescents). Transcranial duplex ultrasound of cerebral vessels was performed. Statistical analysis was conducted using IBM SPSS Statistics 23. Differences were considered statistically significant at $p < 0.05$. ***Results.*** Cognitive impairment was detected in 13.6% of children from the early stages of SARS-CoV-2 infection and persisted for up to three years. Moderate and severe asthenia were associated with an increased risk of cognitive deficit. The likelihood of cognitive impairment was also influenced by the severity of infection symptoms, the presence of neurological manifestations, as well as the child's sex and age.

KEYWORDS: *cognitive impairment, children, COVID-19, SARS-CoV-2, asthenic syndrome, risk factors*

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Механизмы формирования когнитивных нарушений у детей, перенесших коронавирусную инфекцию

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РЕЗЮМЕ. ***Введение.*** Выявление причин формирования когнитивных нарушений, их ранняя диагностика, прогнозирование и реабилитация пациентов остаются актуальными задачами педиатрической практики. ***Цель исследования*** — изучить механизмы формирования и определить факторы риска развития когнитивных нарушений у детей, перенесших легкую и среднетяжелую формы COVID-19. ***Материалы и методы.*** Под наблюдением находились 147 детей в возрасте от 7 до 18 лет, перенесших инфекцию SARS-CoV-2. В 1-ю группу вошли 22 ребенка с когнитивными нарушениями, во 2-ю — 125 детей без нарушений. Группы были сопоставимы по полу и возрасту. Когнитивные нарушения оценивали с помощью синдромного факторного анализа нарушений высших корковых функций человека, предложенного А.Р. Лурией. Частоту и тяжесть астенического синдрома определяли по шкале Л.Д. Малковой и Т.Г. Чертовой в модификации для подростков (Шкала астенического состояния). Проводилось транскраниальное дуплексное сканирование сосудов головного мозга. Статистическая обработка осуществлялась с использованием IBM SPSS Statistics 23. Значимость принимали при $p < 0,05$. ***Результаты.*** Когнитивные нарушения выявлены у 13,6% детей с первых дней заболевания SARS-CoV-2 и сохранялись до трех лет. Установлено, что умеренная и тяжелая астения повышает риск когнитивного дефицита. На вероятность развития нарушений также влияют выраженность симптомов инфекции, наличие неврологических проявлений, а также пол и возраст ребенка.

КЛЮЧЕВЫЕ СЛОВА: *когнитивные нарушения, дети, COVID-19, SARS-CoV-2, астенический синдром, факторы риска*

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INTRODUCTION

Children's health is one of the priority issues of medicine in the Russian Federation. At the same time, the state of physical and mental health largely depends on the body's ability to adapt to changing environmental conditions [1, 2].

Adaptation to environmental factors is ensured by the autonomic nervous system (ANS) through the coordinated activity of the sympathetic and parasympathetic divisions [3]. Exposure to adverse factors exceeding the body's reserve capacities leads to tension of adaptive mechanisms. Researchers attribute the novel coronavirus infection (SARS-CoV-2), COVID-19, to the causes of reduced quality of life and deterioration of physical and mental health in children and adolescents [4–7].

It is known that since the onset of the COVID-19 pandemic, an increase in neurological disorders has been observed worldwide, the genesis of which may be associated with damage to nervous system cells following the passage of the SARS-CoV-2 virus through the blood-brain barrier. Children of this age are particularly vulnerable in this regard, since it is during this period of life that active development of environmental perception, memory, speech, and psychomotor coordination occurs [4, 8]. Disruption of adaptive mechanisms may affect the intellectual development of the child, cause a decline in cognitive abilities, and, as a consequence, lead to difficulties in learning educational material, among other effects [2, 9].

For a comprehensive assessment of higher mental functions (HMF) and the emotional-personal sphere, it is recommended to study cerebral hemodynamics with determination of the state of adaptive and compensatory mechanisms of cerebral blood flow autoregulation [10]. For this purpose, transcranial duplex scanning (TCDS) of cerebral vessels is performed, which makes it possible to study baseline blood flow characteristics and cerebrovascular reactivity (the ability of cerebral vessels to change their diameter in response to various specific stimuli) in real time [11].

In this regard, the issues of early detection of cognitive disorders, identification of risk factors for their development, and development of prognostic models allowing timely implementation of preventive and rehabilitation measures are relevant [12, 13]. All of the above determined the purpose of the present study.

AIM

The aim is to investigate the mechanisms underlying the development of cognitive impairments and identify risk factors for their onset in children who have had mild or moderate COVID-19.

MATERIALS AND METHODS

The present cohort study was conducted from 2020 to 2024 at Children's Polyclinic No. 1 of the Krasnogorsk City Hospital. A total of 147 patients who had experienced outpatient SARS-CoV-2 infection were observed. There were 54 boys and 93 girls. The age of the participants ranged from 7 to 18 years, with a median age of 13 (10; 15) years. The children were divided into two groups: Group 1 included 22 children (6 boys and 16 girls) with identified cognitive impairment, and Group 2 included 125 children (48 boys and 77 girls) without cognitive impairment. Both groups were comparable in terms of sex and age (Table 1).

Inclusion criteria: children aged 7 to 18 years, belonging to health groups I–II, with laboratory-confirmed mild to moderate coronavirus infection.

Exclusion criteria: patients younger than 7 years or older than 18 years; children belonging to health groups III–IV (chronic somatic and neurological diseases, including autism spectrum disorders, intellectual disability of any etiology, and organic psychosomatic disorders); children who had or had not experienced coronavirus infection.

Higher mental function (HMF) impairments (memory, thinking, speech, perception, and attention) were identified using the syndrome-based factor analysis of HMF disorders proposed by Alexander Romanovich Luria, which enables assessment of working memory and attention processes, including memorization, retention, and recall of information. Intellectual activity and mnemonic performance of the child were analyzed; the speed of auditory-verbal memorization of a list of 10 words was calculated; visual memory and voluntary attention were also assessed, and the capacities of memorization and delayed text recall were determined [14].

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Table 1. Distribution of study participants by sex and age, n/%*

Таблица 1. Распределение наблюдаемых детей по полу и возрасту, n/%*

Возраст Age	1-я группа 1st group n=22		2-я группа 2nd group n=125		Итого Total n=147	
	пол sex		пол sex		пол sex	
	мальчики boys	девочки girls	мальчики boys	девочки girls	мальчики boys	д девочки girls
7–11 лет 7–11 years	3/13,6	8/36,4	18/14,4	20/16,0	21/14,3	28/19,0
12–15 лет 12–15 years	2/9,1	8/36,4	17/13,6	41/32,8	19/12,9	49/33,3
16–18 лет 16–18 years	1/4,5	–	13/10,4	16/12,8	14/9,5	16/10,9
Итого Total	6/27,3	16/72,7	48/38,4	77/61,6	54/36,7	93/63,3

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

* p >0,05.

To assess the frequency and severity of asthenic syndrome, an express test for asthenic manifestations was performed using the Asthenic State Scale (developed by L.D. Malkova and T.G. Chertova in a modified version for adolescents) [15]. Manifestations of asthenic syndrome included general weakness, rapid fatigability (reduced tolerance to physical and academic activities), decreased appetite, and emotional lability (EL).

In the framework of a comprehensive assessment of higher mental functions (HMF) and the emotional-personal sphere, transcranial duplex scanning (TCDS) of cerebral vessels with functional tests was performed. The key measured parameter was the reactivity index (RI), calculated using the formula:

$$RI = V_{sys} / V_{base},$$

where V_{sys} is the linear blood flow velocity after the test; V_{base} is the baseline velocity (before the test).

An RI value ranging from 1.1 to 1.4 was interpreted as a positive response, corresponding to minimal activity of autoregulatory mechanisms (normal); an RI value exceeding 1.4 was considered an enhanced positive response (in cases of pre-existing vascular tone disturbances); RI values of 0.9–1.1 were interpreted as a negative response to hypercapnia (exhaustion of the regulatory system due to structural changes in the vascular wall caused by endothelial damage); RI values below 0.9 were regarded as a paradoxical response, indicating failure of autoregulatory mechanisms [16].

A comprehensive examination was performed in all children at baseline (within the first 48 hours of illness) and dynamically (at 1, 12, 24, and 36 months).

For statistical processing of the results, IBM SPSS Statistics 23 software was used. The Shapiro–Wilk test was applied to assess data normality. Data with non-normal distribution were presented as median and first and third quartiles Me (Q_1–Q_3); comparisons were performed using the Mann–Whitney U test. Categorical data were presented as absolute values (n) and percentages (%), and compared using Fisher's exact test.

The strength of association between binary dependent variables and binary or quantitative predictors was assessed using odds ratios (OR) obtained from univariate logistic regression models. Results were considered statistically significant at $p < 0.05$. Predictive modeling of cognitive disorder risk was performed using logistic regression with the Akaike information criterion. The informativeness of the models was evaluated using Nagelkerke's pseudo-R^2, ROC curve analysis, and AUC (area under the curve). For all models, sensitivity, specificity, predictive accuracy, and 95% confidence intervals (CI) were calculated.

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RESULTS

At the early stage of coronavirus infection in children, the following complaints were reported (n=147): elevated body temperature (117 – 79.6%), myalgia (23 – 15.6%), arthralgia (13 – 8.8%), abdominal pain (12 – 8.2%), nausea (11 – 7.5%), general weakness (142 – 96.6%), headache (125 – 85.0%), dizziness (56 – 38.1%), sweating (117 – 79.6%), and tachycardia (68 – 46.3%).

Intergroup analysis showed that in the acute phase of the disease these symptoms were more frequent in patients with cognitive impairment than in children without it: emotional lability (EL) – 19/20 (95%) and 73/127 (57.5%); dizziness – 13/20 (65%) and 43/127 (33.9%); sleep disturbances – 13/20 (65%) and 21/127 (16.5%), respectively (p <0.05). At the same time, most clinical symptoms in Group 1 persisted throughout the entire follow-up period (Table 2).

Conversely, in the group of patients without cognitive impairment, by the end of the 36th month a statistically significant (p <0.05) decrease in the frequency of all studied symptoms was recorded compared with the beginning of the disease. A positive trend toward a reduction in the frequency of several symptoms was already observed from the first month (Table 3).

Table 2. Neurological symptoms and findings in children of Group 1 during follow-up, n/%*

Таблица 2. Симптомы и показатели неврологического статуса детей 1-й группы в динамике, n/%*

Симптомы и неврологический статус Symptoms and neurological status	Исходно Initially n=20	Через 1 месяц At 1 month n=18	Через 12 месяцев At 12 months n=16	Через 24 месяца At 24 months n=13	Через 36 месяцев At 36 months n=12
Слабость Weakness	20/100,0	17/94,4	16/100,0	11/84,6	11/91,7
Утомляемость Fatigue	20/100,0	16/88,9	15/93,8	11/84,6	11/91,7
Эмоциональная лабильность Emotional lability	19/95,0	14/77,8	13/81,3	11/84,6	10/83,3
Нарушение аппетита Loss of appetite	16/80,0	12/66,7	11/68,8	9/69,2	8/66,7
Нарушение вкуса Taste impairment	14/70,0	12/66,7	11/68,8	9/69,2	8/66,7
Головная боль Headache	18/90,0	15/83,3	14/87,5	11/84,6	10/83,3
Нарушения обоняния Olfactory impairment	14/70,0	12/66,7	11/68,8	9/69,2	8/66,7
Потливость Hyperhidrosis	18/90,0	14/77,8	14/87,5	11/84,6	9/75,0
Тахикардия Tachycardia	11/55,0	9/50,0	8/50,0	7/53,8	6/50,0
Головокружение Dizziness	13/65,0	11/61,1	12/75	9/69,2	8/66,7
Лабильность артериального давления Blood pressure lability	9/45,0	7/38,9	6/37,5	5/35,5	4/33,3
Нарушения сна Sleep disorders	13/65,0	7/38,9	12/75	9/69,2	8/66,7
Дизосмия Dysosmia	14/70,0	10/55,6	12/75	10/76,9	8/66,7
Дисгевзия Dysgeusia	15/75,0	11/61,1	13/81,3	11/84,6	9/75,0

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

* p >0,05.

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Table 3. Neurological symptoms and findings in children of Group 2 during follow-up, n/%

Таблица 3. Симптомы и показатели неврологического статуса детей 2-й группы в динамике, n/%

Симптомы и неврологический статус Symptoms and neurological status	Исходно Initially n=127	Через 1 месяц At 1 month n=118	Через 12 месяцев At 12 months n=85	Через 24 месяца At 24 months n=74	Через 36 месяцев At 36 months n=79
Слабость Weakness	122/96,1	108/91,5	80/94,1	63/85,1*	44/55,7*
Утомляемость Fatigue	113/89	105/89,0	76/89,4	60/81,1	44/55,7*
Эмоциональная лабильность Emotional lability	73/57,5	63/53,4	43/50,6	37/50,0	32/40,5*
Нарушение аппетита Loss of appetite	94/74,0	63/53,4*	40/47,1*	30/40,5*	29/36,7*
Нарушение вкуса Taste impairment	70/55,1	45/38,1*	28/32,9*	24/32,4*	19/24,1*
Головная боль Headache	107/84,3	80/67,8*	61/71,8*	53/71,6*	52/65,8*
Нарушения обоняния Olfactory impairment	64/50,4	47/39,8	29/34,1*	30/40,5*	20/25,3*
Потливость Sweating	99/78,0	93/78,8	51/60*	40/54,1*	37/46,8*
Тахикардия Tachycardia	57/44,9	44/37,3	22/25,9*	21/28,4*	15/19,0*
Головокружение Dizziness	43/33,9	39/33,1	27/31,8	25/33,8	16/20,3*
Лабильность артериального давления Arterial pressure lability	45/35,4	36/30,5	22/25,9	22/29,7	14/17,7*
Нарушения сна Sleep disorders	21/16,5	16/13,6	12/14,1	10/13,5	5/6,3*
Дизосмия Dysosmia	70/55,1	34/28,8*	34/40*	29/39,2*	26/32,9*
Дисгевзия Dysgeusia	67/52,8	31/26,2*	30/35,3*	27/36,5*	23/29,1*

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

* Values are statistically significantly different from baseline (p <0.05).

* Значения статистически значимо отличаются от исходных (p <0,05).

As shown in Table 3, appetite disturbances decreased from 94 (74%) to 29 (36.7%), headache from 107 (84.3%) to 52 (65.8%). By the 36th month, dysosmia and dysgeusia were also less frequent than at baseline, decreasing from 70 (55.1%) to 26 (32.9%) and from 67 (52.8%) to 23 (29.1%) of children, respectively. At the same time, throughout the entire follow-up period, children with cognitive impairment had significantly higher rates of almost all post-COVID symptoms (emotional lability, appetite disturbances, smell and taste disorders, sleep disturbances, dizziness, sweating, tachycardia, as well as dysosmia and dysgeusia) compared with Group 2 (p <0.05).

Interesting data were obtained when analyzing the dynamics of clinical manifestations of cognitive impairment in children of Group 1. Thus, 30 days after COVID-19, two children showed no problems with memory, attention, or thinking, whereas 18 children with cognitive impairment continued to report reduced academic performance. Examination of schoolchildren revealed working memory deficits (17/94.4%) as well as impaired distribution and switching of attention (16/88.9%). By the end of the first year, cognitive

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disorders had resolved in four children, attention concentration had improved, and academic performance had normalized. Thus, one year after COVID-19, cognitive deficits persisted in 16 of 20 children, accounting for 88.9%.

Twenty-four months after COVID-19, transient post-COVID cognitive impairments resolved in another three children from Group 1; however, they persisted in 13 children in the form of impaired attention concentration and cognitive flexibility, short-term memory deficits, impaired encoding, and reduced verbal fluency.

By the end of the third year of follow-up, cognitive functions had normalized in one more

Table 4. Transcranial duplex ultrasound findings of cerebral vessels in children of Group 1 during follow-up, n/%

Таблица 4. Результаты транскраниального дуплексного сканирования сосудов головного мозга у детей 1-й группы в динамике, n/%

Параметры цереброваскулярной реактивности **Cerebrovascular reactivity parameters**	**Исходно** **Initially** **n=20**	**Через 1 месяц** **At 1 month** **n=18**	**Через 12 месяцев** **At 12 months** **n=16**	**Через 24 месяца** **At 24 months** **n=13**	**Через 36 месяцев** **At 36 months** **n=12**
ИР усиленно + RI enhanced + n/%	0/0	0/0	0/0,0	0/0	0/0
ИР+ RI+ n/%	9/45	9/50	8/50	7/53,8	10/83,3
ИР (–) RI (–) n/%	10/50	8/44,4	8/50	6/46,1	4/33,3
ИР <1,1 RI <1,1 n/%	11 / 55	9 / 50	8 / 50	6 / 46,1	4 / 33,3
ИР RI Me (Q_1; Q_3)	1,08 (1; 1,12)	1,09 (1; 1,12)	1,10 (1,05; 1,12)	1,10 (1,09; 1,13)	1,18* (1,1; 1,2)
ИР парадоксальный RI paradoxical n/%	1/5	1/5,6	0/0,0	0/0	0/0
Повышение линейной скорости кровотока в артериях каротидного бассейна Increased linear blood flow velocity in the carotid circulation n/%	12/60	11/61,1	11/68,8%	8/61,5	8/66,7
Повышение линейной скорости кровотока в артериях вертебробазилярного бассейна Increased linear blood flow velocity in the arteries of the vertebrobasilar basin n/%	11/55	10/55,6	10/62,6%	7/53,8	8/66,7
Повышение линейной скорости кровотока в глубоких венах мозга Increased linear blood flow velocity in the deep cerebral veins n/%	15/75	13/72,2	14/87,5	9/69,2	8/66,7

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

Note: * — values are statistically significantly different from baseline (p <0.05). RI+ — positive reactivity index; RI(–) — negative reactivity index.

Примечание: * — значения статистически значимо отличаются от исходных (p <0,05); ИР+ — положительный индекс реактивности; ИР (–) — отрицательный индекс реактивности.

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child from Group 1. At the same time, 12 (60.0%) patients still exhibited impairments in memory, attention, cognitive switching skills, visual gnosia, and visuospatial function. Such abnormalities significantly reduced the cognitive and adaptive capacities of the children, diminished their ability to acquire new experience, caused distortions in behavioral and psycho-emotional domains, and often led to certain changes in personality development and socialization difficulties.

Analysis of TCDS data of cerebral vessels during COVID-19 and in the follow-up period confirmed the possibility of the infection affecting the

Table 5. Transcranial duplex ultrasound findings of cerebral vessels in children of Group 2 during follow-up, n/%

Таблица 5. Результаты транскраниального дуплексного сканирования сосудов головного мозга у детей 2-й группы в динамике, n/%

Параметры цереброваскулярной **реактивности** **Cerebrovascular reactivity parameters**	**Исходно** **Baseline** **n=127**	**Через 1 месяц** **At 1 month** **n=118**	**Через 12 месяцев** **At 12 months** **n=85**	**Через 24 месяца** **At 24 months** **n=74**	**Через 36 месяцев** **At 36 months** **n=79**
ИР усиленно + RI enhanced + n/%	1/0,8	2/1,7	0/0,0	3/4,1	8/10,1*
ИР+ RI+ n/%	48/37,8	56/47,5	44/51,8*	50/67,6*	60/75,9*
ИР (–) RI (–) n/%	73/57,5	56/47,5	37/43,5	13/17,6*	6/7,6*
ИР <1,1 RI <1,1 n/%	75/59,1	59/50	37/43,5*	16/21,6*	8/10,1*
ИР RI Me (Q_1; Q_3)	1,07 (1; 1,14)	1,09 (1,02;1,18)	1,1* (1,07; 1;16)	1,16* (1,1; 1,23)	1,2* (1,15; 1,3)
ИР парадоксальный RI paradoxical n/%	3/2,4	4/3,4	0/0,0	2/2,7	1/1,3
Повышение линейной скорости кровотока в артериях каротидного бассейна Increased blood flow velocity in the carotid circulation n/%	94/74	89/75,4	55/64,7	45/60,8	49/62,0
Повышение линейной скорости кровотока в артериях вертебро-базилярного бассейна Increased linear blood flow velocity in the arteries of the vertebrobasilar basin n/%	77/60,6	74/62,7	46/54,1	40/54,1	40/0,6
Повышение линейной скорости кровотока в глубоких венах мозга Increased blood flow velocity in the deep cerebral veins n/%	103/81,1	95/80,5	63/74,1	55/74,3	59/74,7

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

Note: * — values are statistically significantly different from baseline (p <0.05). RI+ — positive reactivity index; RI(–) — negative reactivity index..

Примечание: * — значения статистически значимо отличаются от исходных (p <0,05); ИР+ — положительный индекс реактивности; ИР (–) — отрицательный индекс реактивности.

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regulatory system of cerebral blood flow. The results showed that disturbances of cerebral blood flow regulation were initially observed with equal frequency in both groups of children (p >0.05). Thus, a positive RI, indicating minimal activity of autoregulatory mechanisms, was detected in 9 (45%) children of Group 1 and in 48 (37.8%) patients of Group 2; a negative RI, indicating impaired autoregulation, was found in 10 (50%) and 73 (57.5%), respectively. In most patients in the first days of infection, regardless of the presence or absence of cognitive impairment, a similar increase (p > 0.05) in linear blood flow velocity was observed in the carotid artery system: 12 (60%) and 94 (74%), in the vertebrobasilar system: 11 (55%) and 77 (60.6%), as well as in the cerebral venous system: 15 (75%) and 103 (81.1%), respectively.

However, TCDS data obtained during follow-up showed that persistent cognitive deficit may also be associated with disturbances in cerebrovascular reactivity, which, after arising in the acute phase of infection, remained in patients of Group 1 throughout the entire observation period (Table 4), whereas in children of Group 2 cerebrovascular reactivity parameters returned to normal by the end of the third year (p <0.05). At the same time, most children in Group 2 demonstrated persistently increased blood flow velocity in the carotid and vertebrobasilar arteries (VBA), as well as in cerebral veins, throughout the observation period (Table 5).

A reduced reactivity index was significantly more frequently recorded in Group 1 than in Group 2 across all follow-up periods, being observed by the end of the second year in 6 (46.1%) and

Table 6. Predictive models for cognitive impairment in children after COVID-19 at different follow-up time points (odds ratio, 95% CI)

Таблица 6. Модели прогнозирования развития когнитивных нарушений у детей после COVID-19 в различные периоды наблюдения (отношение шансов (доверительный интервал 95%))

Предиктор **Predictor**	**Для 1-го месяца** **At 1 month**	**Для 12-го месяца** **At 12 months**	**Для 24-го месяца** **At 24 months**	**Для 36-го месяца** **At 36 months**
Возраст Age	0,63 (0,43–0,95) p=0,028	0,62 (0,43–0,89) p=0,011	0,47 (0,25–0,86) p=0,015	–
Женский пол Female sex	13,65 (2,12–88,39) p=0,006	–	–	–
Головная боль Headache	11,50 (1,5-88,13) p=0,019	–	–	–
Нарушения обоняния Olfactory impairment	–	–	–	4,49 (1,03–19,52) p=0,045
Нарушение сна Sleep disturbance	–	7,67 (1,43–41,09) p=0,017	27,07 (1,87–392,57) p=0,016	27,24 (5,01–148,18) p <0,001
Выраженность астенического состояния, баллы Severity of asthenic condition, points	–	1,10 (1,02–1,20) p=0,012	1,33 (1,11–1,59) p=0,002	1,11 (1,03–1,19) p=0,004
Повышение линейной скорости кровотока в артериях каротидного бассейна Increased blood flow velocity in the carotid circulation	11,33 (1,47–87,20) p=0,020	0,16 (0,03–0,96) p=0,046	–	–
Индекс реактивности <1,1 Reactivity index <1.1	16,13 (1,21–215,49) p=0,035	–	–	–

The table is based on the authors' original data

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Table 7. Informativeness of forecasting models

Таблица 7. Информативность моделей прогнозирования

Информативность **Informativeness**	**1-й месяц** **At 1 month**	**12-й месяц** **At 12 months**	**24-й месяц** **At 24 months**	**36-й месяц** **At 36 months**
Чувствительность Sensitivity	99,1%	66,7%	88,2%	57,9%
Специфичность Specificity	63,6%	93,8%	94,4%	96,6%
Точность Accuracy	95,9%	86,4%	92,5%	87,2%
AUC	0,929	0,904	0,980	0,876
Псевдо-R^2 Найджелкерке Nagelkerke's pseudo-R^2	0,585	0,584	0,672	0,521

The table is based on the authors' original data

Таблица составлена по данным авторского исследования

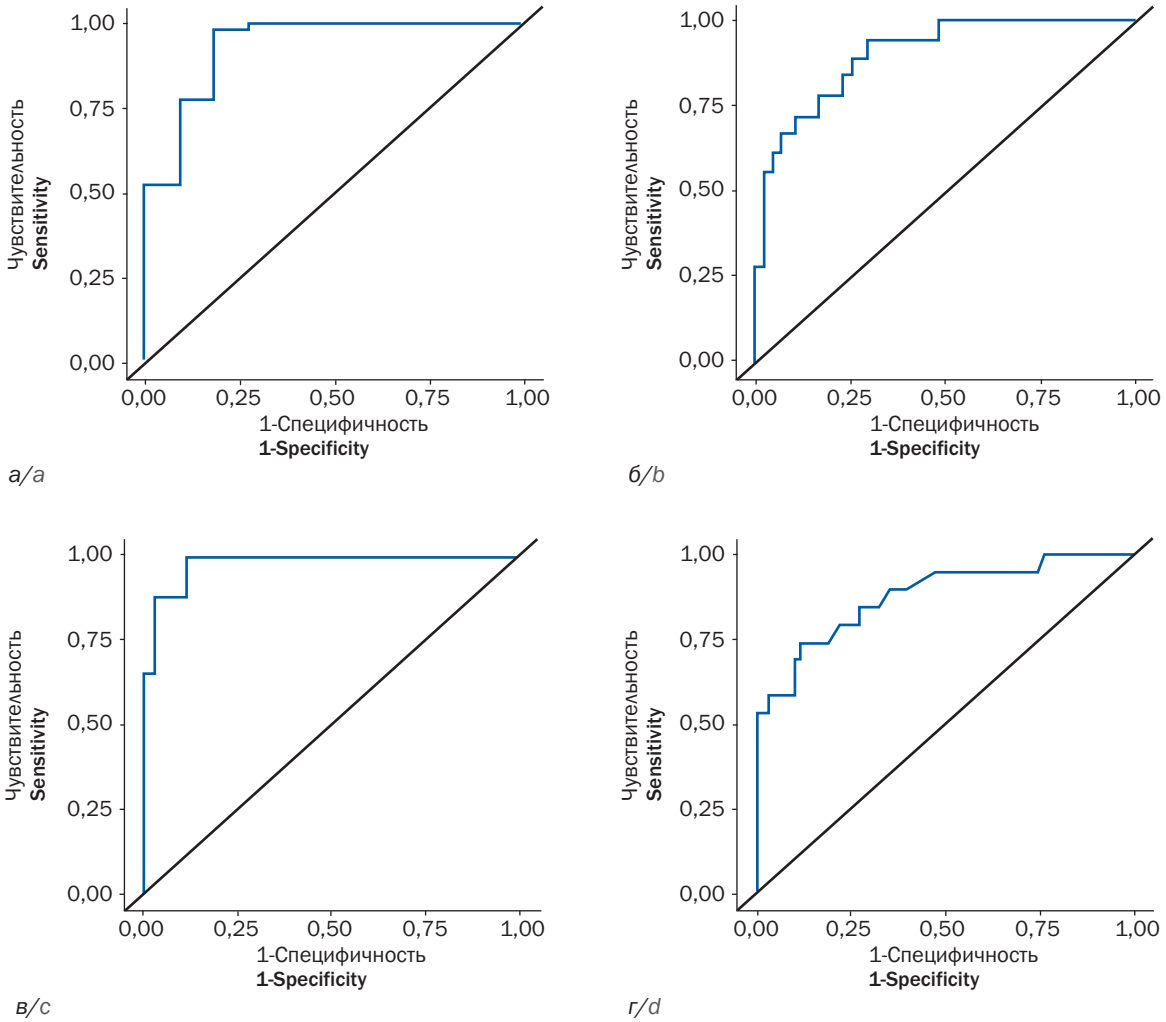

Fig. 1. ROC curves for predicting cognitive impairment at 1 (*a*), 12 (*b*), 24 (*c*), and 36 (*d*) months after COVID-19 using the developed model (the figure is prepared by the authors using their own data)

Рис. 1. ROC-кривые для прогноза развития когнитивных нарушений через 1 (*а*), 12 (*б*), 24 (*в*), 36 (*г*) месяцев после COVID-19 с использованием разработанной модели (рисунок подготовлен авторами по собственным данным)

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13 (17.6%) children, respectively (p=0.031); and by the end of the third year in 4 (33.3%) and 6 (7.6%) children, respectively (p=0.024). Quantitative RI values also differed: at 24 months of follow-up, RI values in patients with cognitive impairment were lower than in children without cognitive impairment: 1.10 (1.09; 1.13) versus 1.16 (1.1; 1.23), respectively (p=0.011). By 36 months, RI values <1.1 were more frequently observed in Group 1 than in Group 2: 4 (33.3%) and 13 (10.1%), respectively (p=0.049). These findings indicate an association between persistent cerebrovascular reactivity disturbances and the persistence of cognitive deficits over three years after COVID-19 infection.

Using logistic regression, the most effective predictors of cognitive impairment development were identified for each follow-up period. Cerebral vessel TCDS parameters and the child's age played the most significant role in predicting cognitive deficits (Table 6).

All developed predictive models for cognitive impairment demonstrated high informativeness (Table 7).

ROC curves for the prediction of cognitive impairment in children who had undergone coronavirus infection at different follow-up periods, using the developed models, are presented in Figure 1.

We also demonstrated a correlation between the probability of developing cognitive impairment and the severity of asthenia. Thus, univariate analysis showed that the presence of grade I asthenic syndrome reduced the likelihood of developing cognitive impairment by 21% at the end of the first month after COVID-19 (95% CI 0.064–0.67; p=0.009). At the same time, grade II–III asthenia, conversely, increased the risk of cognitive impairment by 3.9-fold in children one year after coronavirus infection (95% CI 1.17–13.02; p=0.026), by 23.5-fold after two years (95% CI 2.55–217.12; p=0.005), and by 13-fold at the end of the third year (95% CI 1.28–134.75; p=0.030). This relationship was confirmed by analysis of asthenic syndrome scores using the Asthenic State Scale (ASS) in the two patient groups across different follow-up periods (Table 8).

As shown in Table 8, despite initially similar asthenic manifestations in both groups of children (84 (79; 98) and 78.5 (64; 90) points, respectively), in the subsequent period the severity of asthenia scores was significantly higher in Group 1 than in Group 2. The severity of asthenia in points was also included in the predictive models for cognitive impairment for the 1st, 2nd, and 3rd years of follow-up (p <0.05).

DISCUSSION

Studies by a number of authors have shown that post-COVID syndrome in 11–53% of children may be accompanied by cognitive impairment

Table 8. Cognitive impairment severity in two patient groups at different follow-up time points (L.D. Malkova scale, Me (Q_1–Q_3))

Таблица 8. Выраженность когнитивных нарушений у пациентов двух групп в различные периоды наблюдения в баллах по шкале Л.Д. Малковой, Me (Q_1–Q_3)

Период наблюдения Observation period	Количество пациентов Number of patients, n	1-я группа (с когнитивными нарушениями) Group 1 (with cognitive impairments)	2-я группа (без когнитивных нарушений) Group 2 (without cognitive impairment)	p-value
Начало болезни Disease onset	147	84 (79–98)	78,5 (64–90)	0,034
1-й месяц At 1 month	136	79 (69–96)	69,5 (59–81)	0,043
12-й месяц At 12 months	101	69 (64–81)	61 (53–69)	0,003
24-й месяц At 24 months	87	68 (62–77)	55 (51–61)	<0,001
36-й месяц At 36 months	91	61 (52–67)	51 (46–60)	0,007

The table is based on the authors' original data

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[17–19]. In the present study, a decline in cognitive functions after SARS-CoV-2 infection was detected both at disease onset (13.6%) and subsequently, persisting after 36 months in the majority of children (12 (60.0%)) with initially identified cognitive impairment, which is likely due to two factors:

1) direct damage to neurons and glial cells of the brain through interaction of the coronavirus with angiotensin-converting enzyme type 2 on the surface of neurons;
2) degradation of endothelial cell protein by the viral enzyme Mpro [20, 21].

The present study confirms the previously described in the literature [22, 23] stable association between cognitive impairment and asthenic syndrome in children who have had SARS-CoV-2 infection. Unlike previous publications, we have shown and scientifically substantiated that a statistically significant relationship between cognitive impairment and asthenic syndrome not only persists over a three-year period but also depends on the severity of asthenization. Thus, grade II–III asthenia increased the risk of cognitive impairment by 3.9-fold one year after COVID-19, by 23.5-fold after two years, and by 13-fold at the end of the third year ($p < 0.05$). These findings were obtained for the first time.

The study clearly demonstrates that disturbances in cerebral hemodynamics may contribute to reduced cognitive functions and the development of asthenic syndrome [11]. This assumption was confirmed by our TCDS findings, which allowed an objective assessment of the degree of intracranial blood flow disturbances in children throughout the entire clinical follow-up period (from the first month to 36 months after COVID-19). These results were also obtained for the first time.

CONCLUSION

Cognitive impairment is detected in 13.6% of children from the first days of coronavirus infection, even in cases of mild and moderate disease, and persists for up to three years.

Children who have had coronavirus infection should be followed up by a pediatrician, a neurologist, and a psychotherapist for at least three years to ensure timely correction of cognitive impairment.

During follow-up of a child with or after COVID-19, it is important to take into account age, the severity of asthenic syndrome and TCDS parameters, which are prognostically significant early markers of cognitive dysfunction.

ADDITIONAL INFORMATION

Authors' contribution. L.A. Kharitonova contributed to the study concept and design, formulation of research objectives, literature review, interpretation of the data, and drafting of the manuscript; S.N. Novoselova was responsible for data collection, instrumental examinations (transcranial duplex ultrasound of cerebral vessels), statistical analysis, development of predictive models, and critical revision of the manuscript.

Both authors made substantial contributions to the study, approved the final version of the manuscript, and take responsibility for all aspects of the work.

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

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. Л.А. Харитонова — разработка концепции и дизайна исследования, формулировка целей и задач, анализ научной литературы, интерпретация полученных данных, подготовка и написание рукописи; С.Н. Новоселова — сбор клинического материала, проведение инструментальных исследований (транскраниального дуплексного сканирования сосудов головного мозга), статистическая обработка данных, участие в построении прогностических моделей, редактирование и критический пересмотр содержания статьи.

Авторы внесли существенный вклад в проведение исследования, согласовали окончательный вариант рукописи и несут ответственность за все аспекты работы.

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

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

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