DOI: https://doi.org/10.17816/PAVLOVJ611176 EDN: BKLSOX

Clinical and Metabolic Profile and Cognitive Functions in Children and Adolescents with Carbohydrate Metabolism Disorders Depending on Body Weight

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ABSTRACT

INTRODUCTION: Hyperglycemia causes glucotoxicity of neurons via different mechanisms, and, in combination with obesity, is a strong predictor of cognitive dysfunction. Free fatty acids and circulating cytokines cross the blood-brain barrier leading to neuroinflammation and proliferation of microglia. These alterations can be detected using neuroimaging methods. Thus, evaluation of cognitive functions and glycemic profile seems relevant in children with disorders of carbohydrate metabolism and different weights.

AIM: To analyze clinical and metabolic profile and cognitive functions in children and adolescents with carbohydrate metabolism disorders depending on body weight.

MATERIALS AND METHODS: A prospective, open, controlled study was conducted in 2022–2023. The study included 53 children aged from 7 to 18 years with carbohydrate metabolism disorders with duration of the disease of 1 to 7 years: group 1 — children with excessive body weight or obesity (n=33) and group 2 (n=20) — children with normal body weight. The work included evaluation of anthropometric parameters, carbohydrate metabolism disorders (glycemia and its variability, determination of glycated hemoglobin, immunoreactive insulin, and C-peptide), lipid spectrum, verification of non-alcoholic fatty liver disease, and testing using children's version of Wechsler questionnaire.

RESULTS: Children with carbohydrate metabolism disorders and overweight or obesity more often had relatives with overweight (p=0.04), or diabetes mellitus (p=0.03) and were more often diagnosed with lipidemia (p=0.048) and fatty hepatosis (p=0.031). Children with carbohydrate metabolism disorders, both normal and overweight, showed statistically significant differences in the immunoreactive insulin index: among boys (p=0.030, p=0.001) and girls (p=0.020, p=0.002). Glycemia before bedtime and the time of glycemia above the target range were higher in overweight children (p=0.029, p=0.002). In Wechsler test, children with overweight or obesity and normal body weight children differed in the following parameters: vocabulary (speech function), letter-digit test, Kohs Block Design Test (constructional-spatial praxis; p=0.043, p=0.008 and p=0.005 respectively).

CONCLUSIONS: Children with carbohydrate metabolism disorders in combination with excessive body weight and obesity are characterized by impairment of some cognitive functions associated with asymptomatic glycemic variability.

Keywords: obesity; hyperglycemia; cognitive functions; children and adolescents.

To cite this article:
Samoylova YuG, Matveeva MV, Filippova TA, Podchinenova DV, Yun VE, Galyukova DE, Koshmeleva MV. Clinical and Metabolic Profile and Cognitive Functions in Children and Adolescents with Carbohydrate Metabolism Disorders Depending on Body Weight. *I.P. Pavlov Russian Medical Biological Herald.* 2025;33(2):221–230. DOI: 10.17816/PAVLOVJ611176 EDN: BKLSOX

Received: 24.10.2023 **Accepted:** 19.12.2023 **Published online:** 30.06.2025

DOI: https://doi.org/10.17816/PAVLOVJ611176 EDN: BKLSOX

Клинико-метаболический профиль и когнитивные функции у детей и подростков с нарушением углеводного обмена в зависимости от массы тела

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АННОТАЦИЯ

Введение. Гипергликемия через различные механизмы вызывает глюкозотоксичность нейронов и в сочетании с ожирением является сильным предиктором когнитивной дисфункции. Свободные жирные кислоты и циркулирующие цитокины через гематоэнцефалический барьер приводят к нейроинфламации и пролиферации микроглии. Эти изменения могут быть обнаружены с помощью методов нейровизуализации. Таким образом, представляется актуальным оценка когнитивных функций и гликемического профиля у детей с нарушением углеводного обмена и различной массой.

Цель. Проанализировать клинико-метаболический профиль и когнитивные функции у детей и подростков с нарушением углеводного обмена в зависимости от массы тела.

Материалы и методы. Проспективное, открытое, контролируемое исследование выполнено в период 2022–2023 гг. В исследование включили 53 ребенка в возрасте от 7 до 18 лет с нарушением углеводного обмена и длительностью заболевания от 1 до 7 лет: 1 группа — с избыточной массой тела или ожирением (n=33) и 2 группа (n=20) — с нормальной массой тела. В ходе работы проводили оценку антропометрических параметров, нарушений углеводного обмена (гликемия и ее вариабельность, определение гликированного гемоглобина, иммунореактивного инсулина и С-пептида), липидного спектра, верификацию неалкогольной жировой болезни печени, тестирование с использованием детского варианта опросника Векслера.

Результаты. Дети с нарушением углеводного обмена и избыточной массой тела или ожирением чаще имели родственников с избытком массы тела (p=0,04) или сахарным диабетом (p=0,03), также чаще у них диагностировали дислипидемию (p=0,048) и жировой гепатоз (p=0,031). Дети с нарушением углеводного обмена как с нормальным, так и с избыточным весом статистически значимо отличались по показателю иммунореактивного инсулина: среди мальчиков (p=0,030, p=0,001) и девочек (p=0,020, p=0,002). Гликемия перед сном и время нахождения гликемии выше целевого диапазона были выше у детей с избыточным весом (p=0,029, p=0,002). По тесту Векслера дети с избыточной массой тела или ожирением и нормальной массой тела различались по следующим параметрам: словарный (функция речи), шифровка, кубики Косса (конструктивно-пространственный праксис; p=0,043, p=0,008 и p=0,005 соответственно).

Выводы. Для детей с нарушением углеводного обмена в сочетании с избыточной массой тела и ожирением характерно снижение некоторых когнитивных функций, ассоциированное с бессимптомной вариабельностью гликемии.

Ключевые слова: ожирение; гипергликемия; когнитивные функции; дети и подростки.

Как цитировать:
Самойлова Ю.Г., Матвеева М.В., Филиппова Т.А., Подчиненова Д.В., Юн В.Э., Галюкова Д.Е., Кошмелева М.В. Клинико-метаболический профиль и когнитивные функции у детей и подростков с нарушением углеводного обмена в зависимости от массы тела // Российский медико-биологический вестник имени академика И.П. Павлова. 2025. Т. 33, № 2. С. 221–230. DOI: 10.17816/PAVLOVJ611176 EDN: BKLSOX

Рукопись получена: 24.10.2023 **Рукопись одобрена:** 19.12.2023 **Опубликована online:** 30.06.2025

ЭКО•ВЕКТОР

INTRODUCTION

Advances in molecular biology and neurobiology over the past 30 years opened the way for a deeper understanding of how hyperglycemia affects the brain a non-classic insulin-sensitive tissue. Having crossed the blood-brain barrier, insulin binds to its receptor and initiates a series of phosphorylation events [1, 2]. The signaling pathway mediates a wide range of cellular functions, including synaptic plasticity, cholesterol synthesis, viability of neurons and transport of neurotransmitters [3]. That is, hyperglycemia induces glucotoxicity of neurons by different mechanisms, such as mitochondrial dysfunction, oxidative stress, polyol and hexosamine pathways, and also accumulation of glycated agents [4, 5]. Dysglycemia can combine with obesity, which together are strong predictors of cognitive dysfunction [6].

Overweight is a growing problem worldwide, including children's population. The penetration of free fatty acids and circulating cytokines across the blood-brain barrier leads to neuroinflammation and microglial proliferation [7]. Obesity is associated with a number of adverse alterations in the brain function and structure that can be detected using neuroimaging methods [8].

Thus, it seems relevant to evaluate cognitive functions and glycemic profile in children with carbohydrate metabolism disorders and different weights. The hypothesis of the conducted study was that children with excessive body weight and impaired carbohydrate metabolism are characterized by more pronounced cognitive impairment compared to children with normal body weight.

The **aim** of this study to analyze clinical and metabolic profile and cognitive functions in children and adolescents with carbohydrate metabolism disorders depending on body weight.

MATERIALS AND METHODS

The study was conducted in 2022–2023 at the Siberian State Medical University (SibSMU) at the clinical site of endocrinology department of Children's Hospital No. 1 (Tomsk) and Children's Clinic of SibSMU. The study was approved by Ethics Committee of SibSMU (Protocol No. 6905/1 of November 26, 2018). All study participants and their legal representatives signed a voluntary informed consent.

Study design: prospective, open, controlled, conducted in parallel groups. The sample size was not pre-calculated and was limited by the time interval for inclusion of patients (outpatient, inpatient). The study included 53 children aged 7 to 18 years with impaired fasting glycemia and impaired glucose tolerance and a disease duration of up to 7 years, who were divided into two groups: ***group 1*** — 33 children with overweight and obesity and ***group 2*** (comparison group) — 20 children with normal weight. The groups formed in this way had no statistically significant differences in gender and age (Table 1).

Table 1. Demographic characteristics of study groups

Parameters	Group 1	Group 2	*p*
n	33	20	–
Age, Me [Q1; Q3], years	12.7 [7.1; 16.9]	13.1 [7.4; 16.8]	*0.411*
Male gender, n	16	10	*0.345*
Female gender, n	17	10	*0.395*

Примечания: Me — медиана, Q1 — нижний квартиль, Q3 — верхний квартиль

Non-inclusion criteria: the known presence of autoimmune diseases, clinically significant concomitant pathology of the cardiovascular, nervous, genitourinary systems, gastrointestinal tract, blood diseases, systemic diseases, exacerbations of chronic diseases, oncological diseases, no signed informed consent.

Carbohydrate metabolism disorders were verified according to the clinical guidelines 'Algorithms for specialized medical care for patients with diabetes mellitus', 10th edition (supplemented), 2021 [9]. The duration of carbohydrate metabolism disorders with the underlying obesity was 2 years or more in 9 patients (27.3%), 1 year in 15 (45.4%) children and adolescents, and in 9 people (27.3%) they were identified before inclusion in the study. The group with normal weight included 13 (65.0%) people with duration of these disorders for 1 year, and 7 (35.0%) people with the newly diagnosed condition. All children with impaired carbohydrate metabolism were on diet therapy. Patients with impaired carbohydrate metabolism took metformin 500 mg daily.

The body mass index (BMI) was calculated in the Anthroplus WHO program (WHO, 2007). The results are presented as standard deviation scores (SDS). Sexual development was assessed using the Tanner classification [10].

Glycated hemoglobin (HbA1C; reference value 4.27–6.07%) and average daily fluctuations in the glycemic profile were determined using liquid chromatography. The content of immunoreactive insulin (IRI; reference value 1.9–23.0 μIU/ml) was also assessed using enzyme-linked immunosorbent assay (ELISA). C-peptide (reference value 1.10–4.40 ng/ml) was determined using enhanced chemiluminescence on an ELISA analyzer. The lipid spectrum of blood plasma [11–13] was determined using an enzymatic colorimetric test on an automatic clinical chemistry analyzer Hitachi-911 (Hitachi, Germany) using a Roche reagent kit. Continuous monitoring of blood glycemia was performed using iPro™2 Professional Continuous Glucose Monitoring (Medtronic, USA), CGMS Guardian REAL-Time (Medtronic, USA), FreeStyle Libre (Abbot, USA) devices. Ultrasound examination of the abdominal organs to verify non-alcoholic fatty liver disease [14, 15] was performed on an M7 ultrasound device (Shenzhen Mindray Bio-Medical Electronics, China).

The neuropsychological testing was conducted using children's version of the Wechsler questionnaire, which consists of 12 subtests. The Wechsler Intelligence Scale for Children is intended for children from 6 years old and evaluates verbal (6 subtests) and non-verbal (5 tests) perception. The tests were interpreted using 'keys'. The first part includes assessment of general knowledge, general comprehension, concentration, ability to establish similarity; working memory, verbal experience. The second part analyzes visual-motor skills, visual perception, sensorimotor coordination, logical thinking, synthesis of the whole from parts. Children were tested in the morning hours in calm environment.

Statistical data processing was performed using the SPSS 23.0 software package (IBM SPSS Statistics, USA). The normality of distribution of quantitative variables was assessed using the Kolmogorov–Smirnov test. Sets of quantitative indicators whose distribution differed from normal were described using the median (Me) values and the lower and upper quartiles [Q1; Q3]. The Mann–Whitney test was used to assess the significance of differences in quantitative variables between two independent groups. Statistically significant differences in quantitative indicators in more than two independent groups were assessed using nonparametric analysis of variance. Correlation analysis between quantitative variables was performed using a nonparametric method with calculation of the Spearman rank correlation coefficient. Nominal data were described using absolute values and percentages. Comparison of nominal data was performed using the Pearson χ^2 criterion. In cases where the number of expected observations in any cell of the four-field table was < 5, Fisher's exact test was used to assess the level of significance of differences. Differences were considered statistically significant at p <0.05.

RESULTS

Based on history data, children with obesity and impaired carbohydrate metabolism statistically more often had relatives with excess body weight or impaired carbohydrate metabolism compared to children with normal weight (Table 2).

When assessing anthropometric data, all study participants did not statistically significantly differ from each other in SDS of height. In children and adolescents with overweight, the median BMI was 2.99 [2.55; 3.86] kg/m^2, with normal weight — 0.75 [0.40; 1.00] kg/m^2 (p=0.001).

When analyzing complications of carbohydrate metabolism disorders, dyslipidemia and fatty hepatosis were verified (Figure 1), which statistically significantly prevailed in the group of children with overweight and obesity (15.2% versus 10.0%, p=0.048, and 24.2% versus 10.0%, p=0.031 respectively).

When assessing laboratory parameters, children with carbohydrate metabolism disorders, both with normal and excess weight, showed statistically significant difference in immunoreactive insulin (IRI) parameter (Table 3).

Table 2. History data of children in study groups

Parameters	Group 1	Group 2	*p*
Breast feeding, n (%)	3 (9.1)	5 (25)	*0.165*
Artificial feeding, n (%)	16 (48.5)	10 (50)	*0.072*
Mixed feeding, n (%)	14 (42.4)	5 (25)	*0.077*
Vaccination by the calendar, n (%)	33 (100)	20 (100)	*0.127*
Allergic diseases, n (%)	8 (24.2)	6 (30)	*0.606*
Family history of diabetes mellitus, n (%)	18 (54.5)	6 (30)	***0.040***
Family history of obesity, n (%)	11 (33.3)	7 (35)	***0.030***

When monitoring glycemia, no statistically significant differences between fasting glycemia parameters were noted. Upon that, before bedtime glycemia was statistically significantly higher in children with overweight and obesity. Also, the time of glycemia staying within the target range statistically significantly differed in the groups (Table 4).

The next stage was evaluation of the intelligence by Wechsler test, where children with overweight or obesity and children with healthy weight differed in the following subtests: vocabulary (speech function), Kohs block design test (constructional-spatial praxis), coding (oculo-motor coordination, memory, learnability, motor activity, Table 5).

In correlation analysis, the longer time of hyperglycemia being within the target range had statically significant relationship with Kohs blocks test (r=0.625, p=0.001), which confirms the effect of carbohydrate metabolism disorders on the structure of cognitive functions.

DISCUSSION

The most common carbohydrate metabolism disorder in childhood and adolescence, according to the literature, is carbohydrate intolerance, diagnosed in 5–25% of patients with obesity [16]. In this study, the differences in the clinical-metabolic profile in children with carbohydrate metabolism disorders with and without overweight or obesity were evaluated. Thus, it was found that children with overweight or obesity more often had relatives with diabetes mellitus and/or obesity, they more often had dyslipidemia and steatohepatosis, which agrees with the literature data [17]. It is known that the risk of type 2 diabetes mellitus increases by 45% with increase in body weight [18].

In this study, despite that fact that fasting glycemia level did not differ between the groups, increased insulin level, evening glycemia and reduction of the time of glycemia being in the target range were noted in children with impaired carbohydrate metabolism and obesity.

Table 3. Comparative analysis of laboratory parameters of carbohydrate metabolism (Me [Q1; Q3]) in study groups depending on gender and age

Parameters	Group 1				Group 2				*p*
	Boys		Girls		Boys		Girls		
	≤ 10 years	> 10 years	≤ 10 years	> 10 years	≤ 10 years	> 10 years	≤ 10 years	> 10 years	
n	9	10	7	7	4	7	3	6	–
Fasting blood glucose, mmol/l	5.10 [5.35; 6.80]	5.60 [5.10; 6.30]	5.30 [4.90; 6.20]	5.13 [5.00; 5.90]	5.60 [4.10; 7.15]	5.30 [4.80; 5.90]	6.00 [4.80; 6.20]	5.90 [5.10; 6.30]	*0.678*
Glycated hemoglobin, %	5.70 [5.35; 6.00]	5.81 [5.15; 7.33]	5.40 [5.10; 7.15]	5.01 [4.90; 6.25]	5.90 [5.50; 6.30]	5.20 [4.90; 7.30]	5.70 [5.21; 6.95]	5.60 [5.15; 7.14]	*0.726*
Immunoreactive insulin, μIU/ml	23.60 [14.40; 56.15]	29.90 [14.65; 41.90]	32.10 [21.15; 54.60]	30.90 [27.60; 48.50]	18.75 [11.50; 29.60]	31.60 [24.60; 61.65]	39.20 [21.60; 58.54]	33.40 [26.80; 55.16]	***0.002***
С-пептид, нг/мл	3.20 [2.46; 4.80]	3.10 [1.40; 5.80]	3.81 [2.41; 4.90]	4.20 [3.20; 5.40]	4.02 [1.13; 5.70]	5.20 [2.10; 5.90]	5.14 [3.30; 5.85]	4.20 [1.10; 5.65]	*0.638*

Notes: Me — median, Q1 — lower quartile, Q3 — upper quartile

Table 4. Comparative analysis of glycemic variability (Me [Q1; Q3]) in study group

Parameters	Group 1	Group 2	*p*
Fasting glycemia, mmol/l	5.2 [3.2; 7.1]	4.9 [3.5; 7.2]	*0.072*
Glycemia before bedtime, mmol/l	6.8 [5.2; 8.1]	6.3 [8.3; 10.6]	***0.029***
Mean glycemia level, mmol/l	5.6 [4.3; 6.2]	5.6 [3.9; 6.1]	*0.658*
Time of glycemia above the target range, %	24.8 [33.0; 75.0]	28.9 [40.0; 82.0]	*0.127*
Time of glycemia within the target range, %	56.8 [29.0; 65.0]	60.3 [45.0; 87.0]	***0.002***
Time of glycemia below the target range, %	11.5 [1.0; 17.0]	13.2 [2.0; 20.0]	*0.805*

Notes: Me — median, Q1 — lower quartile, Q3 — upper quartile

Table 5. Comparative analysis of subtest results of Wechsler test (score) in the study groups

Scale	Group number	Meaning	*p*
General knowledge, initial assessment	1	15.5 [13.7; 27.2]	*0.090*
	2	16.0 [14.0; 18.0]	
General knowledge, scale score	1	10.5 [6.0; 18.3]	*0.109*
	2	8.0 [7.0; 12.5]	
Comprehension, initial assessment	1	20.0 [17.0; 24.5]	*0.860*
	2	24.0 [15.5; 27.5]	
Comprehension, scale score	1	15.0 [10.7; 20.0]	*0.360*
	2	20.0 [11.0; 20.0]	
Arithmetic, initial assessment	1	10.5 [8.5; 15.2]	*0.460*
	2	10.0 [9.0; 12.0]	
Arithmetic, scale score	1	11.0 [5.5; 15.7]	*0.240*
	2	10.0 [6.0; 13.0]	
Similarity, initial assessment	1	16.0 [14.0; 21.5]	*0.093*
	2	19.0 [14.5; 22.5]	
Similarity, scale score	1	14.0 [10.7; 16.25]	*0.810*
	2	13.0 [8.0; 16.5]	
Vocabulary, initial assessment	1	34.5 [14.0; 66.8]	***0.043***
	2	39.0 [31.5; 57.0]	
Vocabulary, scale score	1	11.5 [9.0; 17.0]	*0.680*
	2	10.0 [7.5; 14.5]	
Repetition of numbers, initial assessment	1	9.0 [8.5; 11.3]	*0.140*
	2	10.0 [8.5; 11.0]	
Repetition of numbers, scale score	1	8.5 [5.7; 10.5]	*0.690*
	2	11.0 [3.0; 11.0]	
Missing parts, initial assessment	1	16.5 [13.0; 20.0]	*0.570*
	2	13.0 [11.0; 17.0]	
Missing parts, scale score	1	13.5 [11.2; 20.0]	*0.920*
	2	12.0 [8.0; 16.5]	
Sequential pictures, initial assessment	1	42.0 [40.0; 51.0]	*0.670*
	2	40.0 [29.0; 42.5]	
Sequential pictures, scale score	1	14.0 [12.7; 17.7]	*0.930*
	2	13.0 [9.0; 14.5]	
Kohs block design test, initial assessment	1	38.5 [35.5; 49.7]	***0.050***
	2	39.0 [33.0; 41.0]	
Kohs block design test, scale score	1	13.0 [10.0; 15.7]	*0.150*
	2	12.0 [10.5; 13.0]	
Composing figures, initial assessment	1	18.5 [14.5; 29.7]	*0.630*
	2	27.0 [14.0; 31.5]	
Composing figures, scale score	1	6.5 [4.7; 14.5]	*0.390*
	2	12.0 [5.0; 17.0]	
Coding, initial assessment	1	60.0 [42.5; 78.7]	***0.070***
	2	75.0 [67.5; 88.5]	
Coding, scale score	1	15.0 [7.2; 12.0]	***0.008***
	2	20.0 [17.5; 20.0]	
Labyrinth, initial assessment	1	18.0 [13.7; 20.5]	*0.810*
	2	18.0 [15.0; 20.0]	
Labyrinth, scale score	1	11.0 [6.7; 14.5]	*0.740*
	2	11.0 [7.5; 14.5]	

Apparently, this is due to the peculiarities of the eating behavior of children with obesity (predominance of food intake in the evening), and not associated with cognitive impairment [19]. Evening elevation of glycemia most likely explains the identified intergroup differences in the time of glycemia being above the target range in children with overweight and obesity. In this regard, we believe that the inclusion of diagnostics of eating disorders and programs for the correction of the identified changes can bring not only a decrease in BMI, but also an improvement in glycemic parameters [20, 21].

Increase in BMI and body weight are known to be predictors of type 2 diabetes mellitus, and the use of tools that allow glycemia to be assessed at more than one point, can reduce the risk of its development [22]. It is especially important to emphasize this fact, since hyperglycemia is often asymptomatic in obesity, so it was reported in one study that almost 80.0% of overweight children have elevated glucose and insulin levels [23]. In addition, prolonged glycemic variability is associated with cognitive impairment [24]. Recent studies evidence that overweight and obesity in children and adolescents affect cognitive processes and can impair executive functions, cognitive plasticity, planning and decision making [25]. In this study, in Wechsler test, children with excess body weight or obesity and carbohydrate metabolism disorders showed reduced performance in tests associated with spatial-constructional skills, verbal fluency, motor functions and memory. Further study of the issue is required to more accurately determine which cognitive functions may be impaired, whether this is of functional character and whether cognitive training can be used for the rehabilitation of children with obesity. A review of 64 studies showed the potential of cognitive interventions as a structural unit of weight loss programs [26]. In addition, the introduction of web technologies, familiar to children and adolescents, will help to more easily assimilate the usual recommendations for changing eating behavior [27].

Limitations of this study are a small sample size and the lack of confirmation of the identified impairment of cognitive functions in the neuroimaging examination. In addition, more than half of children with carbohydrate metabolism disorders included in the study received metformin, and in this parameter the study group differs from the comparison group, taking into account the effect of this drug on both tissue sensitivity to insulin and glycemic indices. It is also necessary to observe changes in the indices in dynamics.

CONCLUSIONS

Children with overweight or obesity and impaired carbohydrate metabolism more often have burdened history of metabolic disorders and are also more often diagnosed with dyslipidemia and fatty hepatosis. Evening glycemia and the time of glycemia being above the target range were higher in overweight children.

Children with overweight or obesity and impaired carbohydrate metabolism are characterized by a decrease in cognitive functions associated with asymptomatic glycemic variability.

The obtained results confirm the importance of monitoring glycemia in children from risk groups (with burdened family history), as well as changing approaches to teaching children in the 'Obesity School' due to the fact that much knowledge will not be learned due to a deficit in cognitive domains.

ADDITIONAL INFORMATION

Author contributions. Yu.G. Samoylova — concept and design of the study, writing the text, editing; M.V. Matveeva, T.A. Filippova, D.V. Podchinenova, V.E. Yun, D.E. Galyukova, M V. Koshmeleva — collection of material, writing the text. All authors approved the manuscript (the publication version), and also agreed to be responsible for all aspects of the work, ensuring proper consideration and resolution of issues related to the accuracy and integrity of any part of it.

Ethics approval. The study was approved from the Ethics Committee of the Siberian State Medical University (Protocol No. 6905/1 of November 26, 2018).

Consent for publication. All participants of study and their representatives voluntarily signed an informed consent form before being included in the study.

Funding sources. No funding.

Disclosure of interests. The authors have no relationships, activities or interests for the last three years related with for-profit or not-for-profit third parties whose interests may be affected by the content of the article.

Statement of originality. The authors did not use previously published information (text, illustrations, data) when creating this work.

Data availability statement. The editorial policy regarding data sharing does not applicable to this work, and no new data were collected or created.

Generative AI. Generative AI technologies were not used for this article creation.

Provenance and peer-review. This work was submitted to the journal on its own initiative and reviewed according to the usual procedure. Two external reviewers, a member of the editorial board and the scientific editor of the publication participated in the review.

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