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PROTON MAGNETIC RESONANCE SPECTROSCOPY IN CHILDREN WITH DELAYED MENTAL AND SPEECH DEVELOPMENT ASSOCIATED WITH FOCAL TEMPORAL LOBE EPILEPSY

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Background. The delays in mental and speech development are caused by epilepsy, and a special place among the forms of which is focal temporal epilepsy. The study of biomarkers of the considered pathological condition using proton magnetic resonance spectroscopy as indicators amenable to objective assessment and measurement determines the practical relevance of this work.

Aim. The aim of the study was to determine the role and place of proton magnetic resonance spectroscopy in clinical practice in children with mental and speech retardation associated with temporal lobe epilepsy.

Materials and methods. 37 children aged 2 to 10 years were studied. Of these, 15 children with a diagnosis of "mental and speech development delay, structural focal temporal epilepsy" were included in the first comparison group. The second comparison group consisted of 12 children without CNS pathology undergoing 1H-MRI examination to exclude somatic diseases. The third comparison group consisted of 10 children with "structural focal temporal epilepsy", without mental and speech development delay.

Discussion. Multivoxel proton magnetic resonance spectroscopy (method PRESS) was used to determine the concentration of neurometabolites in the brain tissues of patients. In patients with mental and speech development delay associated with temporal epilepsy, a decrease in the ratio of NAA/Cr concentrations ($p < 0.05$) was revealed in the postcentral gyrus on the right, temporal lobe on the right and hippocampus and inner capsule on both sides, due to a decrease in the concentration of N-acetylaspartate; an increase in the ratio of Cho/Cr concentrations ($p < 0.05$) in the prefrontal cortex, postcentral gyrus and inner capsule on both sides, due to an increase in the concentration of choline. Two patients also showed lipid peaks on the lesion side when compared with EEG data.

Conclusions. The revealed metabolic changes in patients with delayed mental and speech development associated with temporal lobe epilepsy may be useful as an additional method of differential diagnosis with other forms of mental and speech development delay.

Keywords: mental retardation; speech delays; epilepsy; magnetic resonance imaging; proton magnetic resonance spectroscopy.

ПРОТОННАЯ МАГНИТНО-РЕЗОНАНСНАЯ СПЕКТРОСКОПИЯ У ДЕТЕЙ С ЗАДЕРЖКОЙ ПСИХОРЕЧЕВОГО РАЗВИТИЯ, АССОЦИИРОВАННОЙ С ФОКАЛЬНОЙ ВИСОЧНОЙ ЭПИЛЕПСИЕЙ

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Актуальность. Одна из причин задержки психоречевого развития — эпилепсия, среди форм которой особое место занимает фокальная височная эпилепсия. Изучение биомаркеров рассматриваемого патологического состояния с помощью протонной магнитно-резонансной спектроскопии как показателей, поддающихся объективной оценке и измерению, определяет практическую актуальность данной работы.

Цель исследования — определить роль и место протонной магнитно-резонансной спектроскопии в клинической практике у детей с задержкой психоречевого развития, ассоциированной с височной эпилепсией.

Материалы и методы. Исследовано 37 детей в возрасте от 2 до 10 лет, из них 15 детей с диагнозом «Задержка психоречевого развития, структурная фокальная височная эпилепсия» вошли в первую группу сравнения. Вторую группу сравнения составили 12 детей без патологии центральной нервной системы, проходящие обследование методом протонной магнитно-резонансной спектроскопии для исключения соматических заболеваний. Третью группу сравнения составили 10 детей со структурной фокальной височной эпилепсией, без задержки психоречевого развития.

Обсуждение. Для определения концентрации нейрометаболитов в тканях головного мозга у пациентов использовали мультивоксельную протонную магнитно-резонансную спектроскопию (методом PRESS). У пациентов с задержкой психоречевого развития, ассоциированной с височной эпилепсией, выявилось снижение соотношения концентраций NAA/Cr ($p < 0,05$) в постцентральной извилине справа, височной доле справа и гиппокампах и внутренней капсуле с обеих сторон за счет снижения концентрации N-ацетиласпартата; увеличение соотношения концентраций Cho/Cr ($p < 0,05$) в префронтальной коре, постцентральных извилинах и внутренней капсуле с обеих сторон за счет повышения концентрации холина. У двух пациентов также обнаружены пики липидов на стороне поражения при сопоставлении с данными электроэнцефалограммы.

Заключение. Протонная магнитно-резонансная спектроскопия с учетом выявленных метаболических изменений у пациентов с задержкой психоречевого развития, ассоциированной с височной эпилепсией, может быть использована как дополнительный метод дифференциальной диагностики с другими формами задержки психоречевого развития.

Ключевые слова: задержка психического развития; задержка речевого развития; эпилепсия; магнитно-резонансная томография; протонная магнитно-резонансная спектроскопия.

BACKGROUND

Psycho-speech development retardation (PSDR) is a group of diseases induced by various causes and includes a delay in mental and speech development. Impairment of the formation and development of basic mental functions in a child, such as memory, attention, thinking, emotional–volitional sphere, skills, intellectual growth, and cognition refers to mental retardation. A delay in speech development is defined as a later acquisition of oral speech by children, with subsequent possible problems in the acquisition of reading and writing skills, which causes difficulties in the acquisition of school skills and affects the overall academic performance of the child. Epilepsy is one of the causes of PSDR, with focal temporal lobe epilepsy as a special form [2, 3].

In Russia, on average, 5%–10% of children have speech problems. In other countries, this figure can reach 30% [5]. Currently, there is a growing interest in the early diagnosis of speech development disorders in children.

Most scientific papers generally focused on the identification of the classical forms of epilepsy. However, these studies have not compared the data in children with disorders of psychoverbal development. The diagnosis of patients was based only on the identification of a focus of epileptiform activity. The development of molecular neuroimaging technologies, which include proton magnetic resonance spectroscopy (^{1}H-MRS), made it possible to determine the place and role of ^{1}H-MRS in clinical practice in these patients.

The study aimed to determine the role and value of ^{1}H-MRS in clinical practice in children with mental retardation associated with temporal lobe epilepsy.

MATERIALS AND METHODS OF RESEARCH

The study included 37 children aged 2–10 years. Fifteen pediatric patients aged 2–8 years [mean age 4.6 years (*SD* 2.028); 8 boys, 7 girls] with a diagnosis of PSDR and structural focal temporal lobe epilepsy were included in group 1. Group 2 consisted of 12 pediatric patients aged 3–10 years [mean age 5 years (*SD* 2.065); 7 boys, 5 girls] without pathologies of the central nervous system (CNS) who were undergoing ^{1}H-MRS examination to rule out somatic diseases. Group 3 consisted of 10 pediatric patients aged 4–9 years [mean age 6 years (*SD* 1.699); 6 boys, 4 girls] with structural focal temporal lobe epilepsy, without PSDR.

The inclusion criteria were as follows: (1) patients who underwent a routine examination in a private neurological center, with complaints of lack of speech and mental retardation, (2) patients aged up to 10 years, and (3) patients who had no contraindications to the ^{1}H-MRS procedure, i.e., intravenous anesthesia. The exclusion criteria were as follows: (1) patients with severe organic brain

lesions, including hydrocephalus, brain developmental abnormalities, genetic syndromes (chromosomal, metabolic, etc.), etc., and (2) patients in transient remission.

All patients underwent electroencephalography (EEG) followed by the determination of a pattern typical of epilepsy. To rule out organic brain lesions, routine magnetic resonance imaging (Philips Ingenia 1.5 T MRI system) was performed using standard research protocols (T1-weighted imaging, T2-weighted imaging, fluid-attenuated inversion recovery-weighted imaging, and diffusion-weighted imaging). To determine the concentration of neurometabolites in the brain tissues of patients, multivoxel ^{1}H-MRS (PRESS method) was used in the prefrontal cortex, postcentral gyri, temporal lobes, internal capsule, and hippocampi on both sides. When calculating the ratio of the concentration of the main metabolites, we relied on the indicators of N-acetylaspartate (NAA) and creatine (Cr) with their characteristic chemical shifts of 2.0 and 3.0 ppm, respectively. Spectrograms were analyzed using the Philips IntelliSpace Portal software package for ^{1}H-MRS. Statistical processing and analysis of research data were performed using Microsoft Office Excel programs.

STUDY RESULTS

Routine magnetic resonance imaging revealed changes in patients with PSDR associated with focal temporal lobe epilepsy, namely, one patient had signs of dysmyelination in the region of the islets of the temporal lobes, eight had enlarged perivascular spaces, six had signs of incomplete myelination, and three had cysts of the pellucid septum.

Figure 1 shows the results of changes in the concentration of the main metabolites characteristic of patients with PSDR associated with focal temporal lobe epilepsy. Two patients with PSDR associated with temporal lobe epilepsy, who had a lipid peak on the side of the lesion, showed certain changes in the main metabolites (Fig. 2).

Data on the ratio of metabolite concentrations obtained using the PRESS program in pediatric patients diagnosed with PSDR and structural focal temporal epilepsy from group 1 and pediatric patients without CNS pathologies from group 2 are presented in Table 1.

Compared with a group of pediatric patients without CNS pathologies, metabolic changes were revealed in pediatric patients with PSDR associated with temporal lobe epilepsy, namely, a decrease in the ratio of NAA/Cr concentrations ($p < 0.05$) in the postcentral gyrus on the right, temporal lobe on the right, and hippocampi and inner capsule on both sides by reducing the concentration of NAA; an increase in the ratio of choline (Cho)/Cr concentrations ($p < 0.05$) in the prefrontal cortex, postcentral gyri, and internal capsule on both sides because of an increase in Cho concentrations. Two patients showed lipid peaks on the side of the lesion when

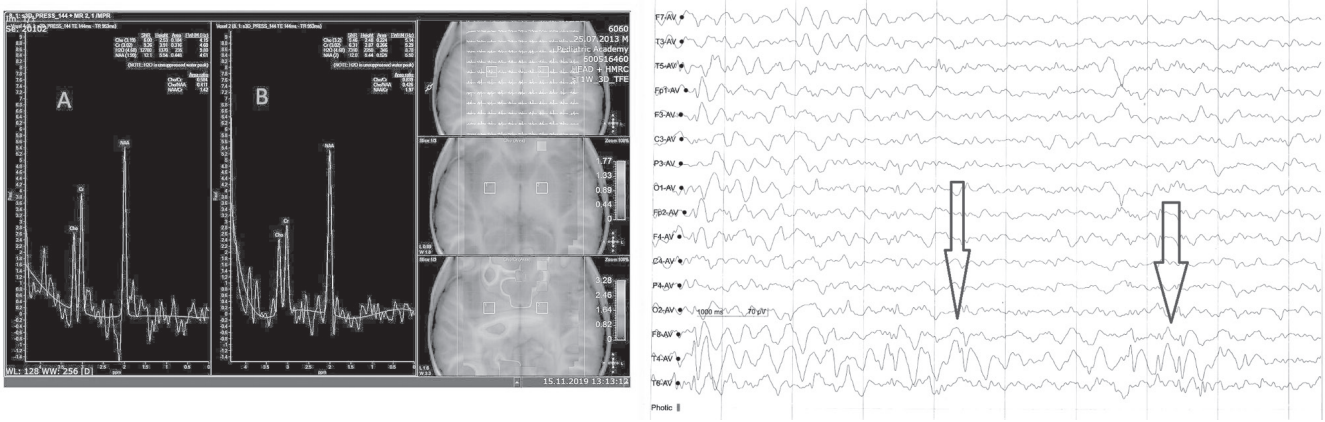

Fig. 1. Left: ^{1}H-MRC of the brain of a patient with mental and speech development delay associated with focal temporal lobe epilepsy. Field of study: poles of the temporal lobes. There is a lateralization of pathology in the right temporal lobe in the form of a decrease in the concentration of N-acetylaspartate (A). The indicators of metabolites in the left temporal lobe are within normal values (B). Right: this patient's EEG revealed epileptiform activity in the temporal region on the right (arrows), without generalization

Рис. 1. Слева: протонная магнитно-резонансная спектроскопия головного мозга пациента с задержкой психоречевого развития, ассоциированной с фокальной височной эпилепсией. Область исследования: полюсы височных долей. Отмечается латерализация патологии в правой височной доле в виде снижения концентрации N-ацетиласпартата (А). Показатели метаболитов в левой височной доле в пределах нормальных величин (В). Справа: на электроэнцефалограмме выявлена эпилептиформная активность в височной области справа (стрелки), без генерализации

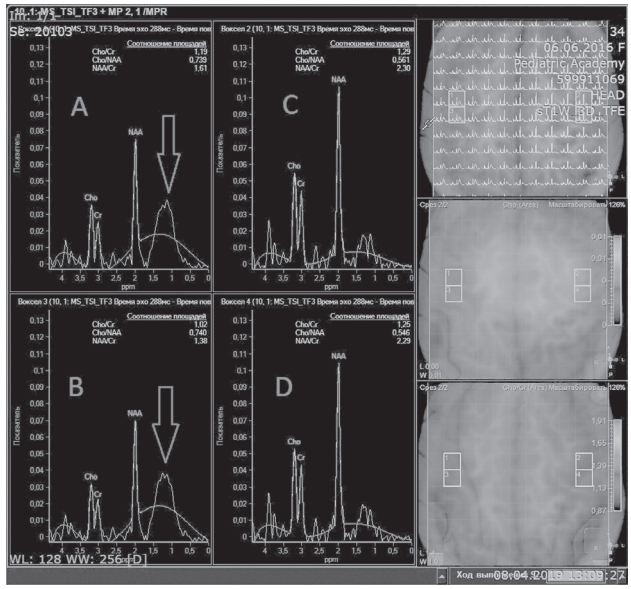

Fig. 2. ^{1}H-MRC of the brain of a patient with mental and speech development delay associated with focal temporal lobe epilepsy. Research area: central and postcentral gyri on both sides. There is a lateralization of pathology in the right frontal and parietal lobes with a decrease in the concentration of N-acetylaspartate (A, B). Lipid peaks are visualized (arrows). In the left parts of the brain, these changes are not observed (C, D). There is also an increase in the concentration of choline on both sides

Рис. 2. Протонная магнитно-резонансная спектроскопия головного мозга пациента с задержкой психоречевого развития, ассоциированной с фокальной височной эпилепсией. Область исследования: центральные и постцентральные извилины с обеих сторон. Отмечается латерализация патологии в правых лобной и теменной долях со снижением концентрации N-ацетиласпартата (A, B). Визуализируются пики липидов (стрелки). В левых отделах головного мозга данных изменений не наблюдается (C, D). С двух сторон прослеживается увеличение концентрации холина

Table 1 / Таблица 1

Average values of the ratio of metabolites in patients from the first and second comparison groups
Средние значения соотношения метаболитов у пациентов из первой и второй групп сравнения

Brain area/ Область головного мозга	1st group NAA/Cr (*SD*) / 1-я группа	2nd group NAA/Cr (*SD*) / 2-я группа	1st group Cho/NAA (*SD*) / 1-я группа	2nd group Cho/NAA (*SD*) / 2-я группа	1st group Cho/Cr (*SD*) / 1-я группа	2nd group Cho/Cr (*SD*) / 2-я группа
Prefrontal cortex (right) / Префронтальная кора (справа)	1.56 (0.732)	2 (0.162), $p = 0.183$	1.08 (0.442)	0.84 (0.231), $p = 0.152$	1.33 (0.42)	0.96 (0.166), $\mathbf{p = 0.01}$
Prefrontal cortex (left) / Префронтальная кора (слева)	1.69 (0.755)	2.12 (0.181), $p = 0.167$	1.14 (0.553)	0.8 (0.229), $p = 0.2$	1.38 (0.477)	1 (0.172), $\mathbf{p = 0.037}$
Postcentral gyrus (right) / Постцентральная извилина (справа)	1.59 (0.699)	2.09 (0.183), $\mathbf{p = 0.041}$	1.16 (0.594)	0.77 (0.226), $p = 0.236$	1.46 (0.356)	0.92 (0.175), $\mathbf{p = 0.021}$
Postcentral gyrus (left) / Постцентральная извилина (слева)	1.62 (0.676)	1.98 (0.181), $p = 0.217$	1.11 (0.545)	0.9 (0.267), $p = 0.323$	1.25 (0.375)	0.88 (0.165), $\mathbf{p = 0.01}$
Temporal lobe (right) / Височная доля (справа)	1.52 (0.463)	1.99 (0.223), $\mathbf{p = 0.05}$	1.06 (0.404)	0.85 (0.197), $p = 0.152$	1.14 (0.394)	0.95 (0.299), $p = 0.347$
Temporal lobe (left) / Височная доля (слева)	1.67 (0.474)	1.89 (0.205), $p = 0.09$	1.12 (0.457)	0.9 (0.241), $p = 0.256$	1.36 (0.517)	1.06 (0.222), $p = 0.126$
Hippocampus (right) / Гиппокамп (справа)	1.47 (0.516)	1.95 (0.214), $\mathbf{p = 0.01}$	1.03 (0.361)	0.78 (0.18), $p = 0.126$	1.08 (0.247)	0.98 (0.186), $p = 0.277$
Hippocampus (left) / Гиппокамп (слева)	1.69 (0.58)	2.17 (0.23), $\mathbf{p = 0.032}$	1.2 (0.520)	0.89 (0.246), $p = 0.152$	1.26 (0.394)	0.99 (0.19), $p = 0.067$
Internal capsule (right) / Внутренняя капсула (справа)	1.59 (0.46)	2.06 (0.226), $\mathbf{p = 0.05}$	0.95 (0.39)	0.79 (0.183), $p = 0.456$	1.28 (0.236)	1.02 (0.191), $\mathbf{p = 0.09}$
Internal capsule (left) / Внутренняя капсула (слева)	1.58 (0.334)	1.92 (0.176), $\mathbf{p = 0.03}$	0.99 (0.339)	0.75 (0.219), $p = 0.075$	1.34 (0.279)	0.97 (0.175), $\mathbf{p = 0.01}$

Note. The Mann–Whitney *U*-index was used for comparison. The selected values of the correlation coefficient (p) are statistically significant ($p < 0.5$) for these comparison areas. *Примечание.* Для сопоставления использовали *U*-индекс Манна – Уитни. Выделенные значения коэффициента корреляции (p) статистически значимые ($p < 0,5$) для данных областей сравнения.

Table 2 / Таблица 2

The average values of the ratio of metabolites in patients from the first and third comparison groups
Средние значения соотношения метаболитов у пациентов из первой и третьей групп сравнения

Brain area/ Область головного мозга	1st group NAA/ Cr (*SD*) / 1-я группа	3rd group NAA/ Cr (*SD*) / 3-я группа	1st group Cho/ NAA (*SD*) / 1-я группа	3rd group Cho/ NAA (*SD*) / 3-я группа	1st group Cho/ Cr (*SD*) / 1-я группа	3rd group Cho/ Cr (*SD*) / 3-я группа
Prefrontal cortex (right) / Префронтальная кора (справа)	1.56 (0.732)	1.97 (0.226), $p = 0.261$	1.08 (0.442)	0.88 (0.193), $p = 0.261$	1.33 (0.42)	1.05 (0.362), $p = 0.144$
Prefrontal cortex (left) / Префронтальная кора (слева)	1.69 (0.755)	1.98 (0.269), $p = 0.723$	1.14 (0.553)	0.87 (0.34), $p = 0.311$	1.38 (0.477)	1.1 (0.349), $p = 0.103$
Postcentral gyrus (right) / Постцентральная извилина (справа)	1.59 (0.699)	1.9 (0.298), $p = 0.311$	1.16 (0.594)	0.88 (0.451), $p = 0.285$	1.46 (0.356)	0.97 (0.258), $p = 0.167$
Postcentral gyrus (left) / Постцентральная извилина (слева)	1.62 (0.676)	1.93 (0.386), $p = 0.338$	1.11 (0.545)	0.93 (0.565), $p = 0.461$	1.25 (0.375)	1.02 (0.319), $p = 0.160$
Temporal lobe (right) / Височная доля (справа)	1.52 (0.463)	1.58 (0.193), $p = 0.261$	1.06 (0.404)	0.85 (0.263), $p = 0.238$	1.14 (0.394)	0.94 (0.368), $p = 0.129$
Temporal lobe (left) / Височная доля (слева)	1.67 (0.474)	1.9 (0.141), $p = 0.129$	1.12 (0.457)	0.97 (0.471), $p = 0.367$	1.36 (0.517)	1.04 (0.442), $p = 0.091$
Hippocampus (right) / Гиппокамп (справа)	1.47 (0.516)	1.87 (0.182), **$p = 0.023$**	1.03 (0.361)	0.9 (0.368), $p = 0.355$	1.08 (0.247)	0.92 (0.147), $p = 0.103$
Hippocampus (left) / Гиппокамп (слева)	1.69 (0.58)	1.95 (0.299), $p = 0.338$	1.2 (0.520)	1.06 (0.492), $p = 0.495$	1.26 (0.394)	0.98 (0.244), **$p = 0.048$**
Internal capsule (right) / Внутренняя капсула (справа)	1.59 (0.46)	1.89 (0.338), $p = 0.062$	0.95 (0.39)	0.77 (0.235), $p = 0.338$	1.28 (0.236)	1.07 (0.176), **$p = 0.031$**
Internal capsule (left) / Внутренняя капсула (слева)	1.58 (0.334)	1.85 (0.302), **$p = 0.026$**	0.99 (0.339)	0.82 (0.181), $p = 0.129$	1.34 (0.279)	1.08 (0.257), **$p = 0.026$**

Note. The Mann–Whitney *U*-index was used for comparison. The selected values of the correlation coefficient (*p*) are statistically significant ($p < 0.5$) for these comparison areas. *Примечание.* Для сопоставления использовали *U*-индекс Манна – Уитни. Выделенные значения коэффициента корреляции (*p*) статистически значимые ($p < 0,5$) для данных областей сравнения.

compared with electroencephalogram data. Other changes in the ratios of metabolites were not significant ($p > 0.05$).

Data on the ratios of metabolite concentrations obtained using the PRESS program in pediatric patients diagnosed with PSDR and structural focal temporal epilepsy from group 1 and pediatric patients with structural focal temporal epilepsy without PSDR from group 3 are presented in Table 2.

Compared with children with structural focal temporal lobe epilepsy without PSDR, pediatric patients with PSDR associated with temporal lobe epilepsy were found to have metabolism alterations in the brain, namely, NAA/Cr concentrations ($p < 0.05$) in the hippocampus on the right and in the inner capsule on the left decreased, whereas Cho/Cr concentrations ($p < 0.05$) in the hippocampus on the left and the internal capsule on both sides were increased. Other changes in the ratios of metabolites were not significant ($p > 0.05$).

The EEG showed a pathological pattern characteristic of focal temporal lobe epilepsy. Data showed a correlation between ^{1}H-MRS and EEG.

DISCUSSION AND CONCLUSIONS

Generally, studies investigating epilepsy only focused on the identification of the pathological focus of epileptiform activity. Data on the relationship of such manifestations with the course of PSDR are not presented. Thus, discussions should consider the aforementioned difficulties.

In patients with PSDR associated with focal temporal lobe epilepsy, NAA/Cr concentrations decreased due to a decrease in NAA concentrations in the postcentral gyrus on the right and the right temporal lobe, which most likely indicates neuronal dysfunction of these areas and lateralization process.

In most cases, in patients with epilepsy who underwent ^{1}H-MRS of the brain, lateralization of parameters was also noted with a decrease in NAA/Cr concentration on the side of the lesion [12]. Similar changes were obtained in a study of 100 patients with temporal lobe epilepsy. A decrease in NAA concentration was registered in the focus determined by EEG data compared with the contralateral side. The degree of asymmetry correlated with a decrease in NAA concentration and a pathological EEG

pattern [9, 10]. Another study found that a decrease in NAA concentration is not only detected in the ipsilateral hippocampus but is also consistent with the findings of positron emission tomography combined with computed tomography, indicating the limbic and subcortical nuclei involved in the pathological processes [8].

In our case, in patients with PSDR associated with focal temporal lobe epilepsy, metabolic alterations were noted in the hippocampus and in the internal capsule on both sides, where the main conductive paths of the brain are located.

Thus, the use of ^{1}H-MRS made it possible to lateralize the epileptic focus in 19 patients. However, mesiotemporal sclerosis was diagnosed in 57.5% of patients in this group [8].

In another study, 30 patients with temporal lobe epilepsy were examined using ^{1}H-MRS. The use of the ^{1}H-MRS asymmetry index made it possible to lateralize the process in 26 patients, and in 16 and 10 cases, it was lateralized on the right and left, respectively. In the remaining four patients, lateralization could not be performed [6].

With epileptiform activity, disorders most probably occur in neurotransmitter systems, followed by the depletion of the concentration of metabolites. This leads to the breakdown of neuronal bonds not only in the cortical centers of praxis and gnosis but also in the centers of speech, causing disorders characteristic of PSDR [1, 13].

In addition to a decrease in NAA concentration in the examined patients with PSDR associated with temporal lobe epilepsy, a change in Cho/Cr concentrations was noted in the prefrontal cortex and region of the postcentral gyri and internal capsule on both sides due to an increase in Cho concentration, which corresponds to the functioning of the limbic and perilimbic systems and may be associated with disorders of higher mental functions [4, 11]. In two patients, lipid peaks were also detected on the lesion side, which only confirms neuronal damage. Similar changes were detected using ^{1}H-MRS in 46 patients with temporal lobe epilepsy in the hippocampus, where a significant increase in the concentrations of lactate, myo-inositol, choline-to-creatine ratio, glutamine, and glutamate on the side of the lesion was noted [7].

CONCLUSION

Significant metabolic changes in the brain were revealed when comparing the concentrations of the main metabolites in pediatric patients with PSDR associated with focal temporal lobe epilepsy with other groups of patients. Despite this, it cannot be stated that a decrease in NAA concentration is pathognomonic for this pathology. NAA only allows lateralization of the focus of epileptiform activity in the brain, as in patients with classic focal epilepsy, which is not a specific sign of PSDR associated with focal temporal lobe epilepsy. An increase in Cho concentration in the internal capsule on both sides during a decrease in NAA concentration is a distinguishing characteristic of patients with PSDR associated with focal temporal lobe epilepsy. This may be due to an impairment of higher mental functions caused by damage to neurons responsible for the limbic and perilimbic systems. In two patients, lipid peaks were also registered on the side of the lesion, which only confirms neuronal damage.

Thus, ^{1}H-MRS, taking into account the revealed metabolic changes in patients with PSDR associated with temporal lobe epilepsy, can be used as an additional differential diagnostic method with other forms of PSDR.

ADDITIONAL INFORMATION

Author contributions. All authors confirm that their authorship complies with the ICMJE criteria. All authors have made a significant contribution to the development of the concept, research, and preparation of the article and have read and approved the final version before its publication.

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