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Paraneoplastic acute ataxia (opsoclonus-myoclonus syndrome) in children, description of two clinical cases

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ABSTRACT

The relevance of studying opsoclonus-myoclonus syndrome in children is due to the complexity of diagnosing this rare pathology, the frequent association of this condition with the neoplastic process, the peculiarities of therapy and the possibility of a recurrent course. This is one of the few paraneoplastic syndromes that occurs in both children and adults, although the mechanisms of immune dysfunction vary. Neurological dysfunction is caused by an autoimmune process and is observed in 3% of children with neuroblastoma. There is evidence of the development of the syndrome in celiac disease. In other cases, the disease may be associated with a viral infection (caused by Epstein–Barr viruses, Coxsackie B, influenza enteroviruses). There is evidence of rare cases of opsoclonus myoclonus syndrome associated with Lyme disease. The clinical picture is represented by ataxia, myoclonic paroxysms and saccading movements of the eyeballs. The article provides brief literature data on the etiology and clinical criteria of o syndrome opsoclonus-myoclonus in children, the importance of oncological search for the primary developing symptoms of ataxia, myoclonus and opsoclonus. The paper describes the authors' own observations of two young patients who were treated at the St. Petersburg State Pediatric Medical University and the Children's City Hospital No. 22 with the diagnosis of opsoclonus-myoclonus syndrome caused by poorly differentiated neuroblastoma of the paravertebral region and the retroperitoneal space. The article presents a description of the etiology (paraneoplastic process in the background of neuroblastoma), clinical picture, features of the course of the disease and therapy, data of neuroimaging, electroencephalographic studies and laboratory tests. Features of the first case are due to the rapid development of neurological symptoms, in the second observation, the syndrome of opsoclonus-myoclonus debuted in the early postoperative period. The study of opsoclonus-myoclonus syndrome presents significant difficulties, due to both the rarity of this pathology and the difficulties of diagnosis. Patients are more likely to seek medical care precisely because of the development of neurological symptoms, rather than because of the clinical manifestations of the tumor. To standardize the diagnosis and treatment, it is necessary to combine data on all diagnosed cases, which was the basis for writing this work.

Keywords: opsoklonus-myoclonus, neuroblastoma, Kinsbourne's syndrome

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Паранеопластическая острая атаксия (синдром опсоклонуса-миоклонуса) у детей, описание двух клинических случаев

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АННОТАЦИЯ

Актуальность изучения синдрома опсоклонуса-миоклонуса у детей обусловлена сложностью диагностики этой редкой патологии, частой ассоциацией данного состояния с неопластическим процессом, особенностями терапии и возможностью рецидивирующего течения. Это один из немногих паранеопластических синдромов, который встречается как у детей, так и у взрослых, хотя механизмы иммунной дисфункции у них различаются. Неврологическая дисфункция обусловлена аутоиммунным процессом и наблюдается у 3% детей с нейробластомой. Есть данные о развитии этого синдрома при целиакии. В остальных случаях заболевание может быть ассоциировано с вирусной инфекцией, вызванной вирусами Эпштейна–Барр, Коксаки В, энтеровирусами гриппа. Есть данные о редких случаях синдрома опсоклонуса-миоклонуса, связанного с болезнью Лайма. Клиническая картина представлена атаксией, миоклоническими пароксизмами и саккадирующими движениями глазных яблок. В статье приведены краткие литературные данные об этиологии и о клинических критериях синдрома опсоклонуса-миоклонуса у детей, важность онкологического поиска при первично развивающихся симптомах атаксии, миоклонуса и опсоклонуса. В работе описаны наблюдения двух пациенток раннего возраста, находившихся в клинике Санкт-Петербургского государственного педиатрического медицинского университета и СПб ГБУЗ «Детская городская больница № 22» с диагностированным синдромом опсоклонуса-миоклонуса, обусловленного низкодифференцированной нейробластомой паравертебральной области и забрюшинного пространства. Представлено описание этиологии (паранеопластического процесса на фоне нейробластомы), клинической картины, особенностей течения заболевания и терапии, данных нейровизуализации, электроэнцефалографических исследований и лабораторных тестов. Особенности первого случая обусловлены быстрым развитием неврологической симптоматики, во втором наблюдении синдром опсоклонуса-миоклонуса дебютировал в раннем послеоперационном периоде. Изучение синдрома опсоклонуса-миоклонуса представляет значительные трудности, обусловлено как редкостью данной патологии, так и сложностями диагностики. Пациенты чаще обращаются за медицинской помощью именно из-за развития неврологической симптоматики, а не из-за клинических проявлений опухоли. Для стандартизации диагностики и лечения необходимо объединять данные обо всех диагностированных случаях, что и послужило основанием для написания данной статьи.

Ключевые слова: опсоклонус-миоклонус, нейробластома, энцефалопатия Кинсбурна

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INTRODUCTION

Opsoclonus-myoclonus syndrome, or Kinsbourne encephalopathy, is a rare neurological disorder. According to statistical studies conducted in the United Kingdom, the syndrome is detected in 0.18 cases per 1 million people per year [15]. The prevalence of the disease in the Russian Federation is unknown. The disease is observed mainly in children. The average age of patients at the onset of the disease is 18 months [10], with girls predominating among affected children.

The name "opsoclonus-myoclonus syndrome" was proposed by P. Talon and C. Stoll in 1985 [14]. This disease was first described by M. Kinsbourne in 1962. He presented a report observing six children aged 9–20 months with coordination disorders, myoclonus, and chaotic eye movements, with four patients showing improvement with adrenocorticotropic hormone therapy [11]. The term "opsoclonus" (Greek *ops* — eye, *klonos* — disorderly movements) was proposed in 1913 by Polish neurologist K. Ozechowski. Other terms are also used to describe opsoclonus: "ocular myoclonus", "flutter", "eye dance", "paired ataxic eye movements", "rapid eye movements", "jelly-like tremor", "saccadomania", and "random eye excitation" [7].

The disease is polyetiological. In 50% of cases it is associated with a paraneoplastic process associated with neuroblastoma (88%) or ganglioneuroma (12%) of the abdominal and thoracic regions [3, 8]. Currently, opsoclonus-myoclonus syndrome is considered an autoimmune disease of the nervous system, which may be triggered by neuronal proteins produced by the tumor. It is believed that clinical manifestations result from an autoimmune response against cross-reactive proteins of the tumor and neurons and axons of the cerebellum [9]. Autoantibodies bind to target antigens and disrupt cell function, but no significant inflammatory cell destruction is observed [11]. In other cases, the disease develops against the background of a previous viral infection or autoimmune process [12].

The clinical picture of the disease is characterized by a basic triad of symptoms: ataxia, opsoclonus, and myoclonus. Ataxia is observed in 81.7% of patients, opsoclonus in 58.9%, and myoclonus in 56.6% of patients [6]. Limb tremors, behavioral, emotional, and speech disorders (dysphasia, dysarthria, mutism), sleep disorders, vomiting, and excessive salivation are often detected [12]. In most cases, the disease begins acutely, with symptoms of cerebellar ataxia. Then opsoclonus develops, which is considered to be hyperkinesis of the eyeballs. It manifests itself as chaotic saccades of the eyeballs in various directions with a frequency of 10–25 Hz, occurring both during their movement and when the gaze is fixed. Opsoclonus may be combined with rapid eyelid movements and persists during sleep. The clinical picture is complemented by myoclonus, which manifests itself in the form of rapid, jerky twitching of facial muscles, limbs, and trunk with a small amplitude and varying degrees of severity.

Diagnosis of the disease is based on a specific clinical picture and detection of cancer or autoimmune disease. A search for neuronal antibodies is performed as a specific diagnostic test. However, in large studies conducted against a background of broad antineuronal reactivity, no specific antibodies for this syndrome have been identified [4, 5].

Since the prevalence of OMS is low [15], it's interesting to look at clinical cases of opsoclonus-myoclonus syndrome in two girls. The syndrome was caused by poorly differentiated neuroblastoma of the paravertebral region and retroperitoneal space.

DESCRIPTION OF CLINICAL CASES

Clinical case No. 1

Patient N., 2 years old, was admitted to the psychoneurological department of St. Petersburg State Budgetary Healthcare Institution "Children's City Hospital No. 22" on an emergency basis due to complaints of acute gait disturbance and tremors in hands and legs. These complaints had been noted for two days; the girl began to stumble, tremors of extremities appeared at rest and during movement, and behavioral disorders (crankiness, tearfulness) were noted. The medical history indicates that the child was born during the fourth pregnancy (the first child is healthy, the second and third pregnancies ended in medical abortion), second full-term delivery, birth weight 3630 g, Apgar score 8/9, discharged from the maternity hospital on the 5th day of life. Mental and motor development up to one year of age was unremarkable. No neurological pathology was detected in the first two years of life, but a delay in speech development was noted. Preventive vaccination was carried out in accordance with the National Preventive Vaccination Calendar.

Upon admission, her condition was moderate, and she reacted negatively to examination: she cried, turned away, and acted capriciously. She understood spoken language, but her speech consisted of single words and onomatopoeia. The skin and visible mucous membranes are without rash, there are three pigment spots of "coffee with milk" color, a pink hemangioma in the left lateral region of the abdomen 3.0×3.0 cm. Cranial innervation was without pathology. The volume of movement in the limbs was full (passive and active movements). Gait disturbance was noted — wide base with forward tilt, unsteadiness when walking, stands without support on a wide base. There was kinesiogenic tremor in the hands of medium amplitude. The patient sat steadily and sat down independently. There was no miss. Muscle tone was physiological and symmetrical. Deep reflexes were moderately active and symmetrical. Abdominal reflexes were symmetrical

and active. There were no pathological foot signs. Preliminary diagnosis upon admission: acute ataxia, unspecified. During the week after admission, neurological disorders progressed. Missed movements appeared in both hands, along with coarse kinetic tremor in hands while using objects. There were difficulties in eating. When the girl tried to bring a spoon to her mouth, she experienced coarse intentional tremor, missed movements, and myoclonus in her hands, symmetrically on both sides. She sat steadily but stood unsteadily without support. When walking, she experienced myoclonus in her legs. She started to avoid walking on her own, walking with a wide base of support, and walking unsteadily with support along the couch. Muscle tone decreased dynamically, muscle strength was 5 points on both sides. Opsoclonus appeared when following objects with her eyes, on both sides. Subsequently, neurological symptoms progressed. Progression of opsoclonus (when fixing her gaze and during voluntary eye movements), drooling, and isolated episodes of vomiting were observed. The girl could not stand independently, sat with slight support, had pronounced myoclonus in the limbs, generalized tremor in the limbs, progression of muscle hypotonia in the limbs (symmetrical), sleep disturbance, pronounced behavioral disturbance (tearfulness, capriciousness, negativism).

Laboratory diagnostics.The following tests were performed, no pathological changes were found: complete blood count, urinalysis, general therapeutic blood chemistry, electrolytes, antistreptolysin O (upon admission and over time), laboratory assessment of thyroid function (free thyroxine (T_4) and triiodothyronine (T_3), thyroid-stimulating hormone (TSH)), prothrombin index (PTI), international normalized ratio (INR), fibrinogen protein, antithrombin (AT III), activated partial thromboplastin time (APTT). *Sars Coronavirus* polymerase chain reaction (blood) was negative. Stool test for *Salmonella sp., Shigella sp., E. coli* was negative. Antibodies (IgG, IgM in blood serum) to *B. burgdorferi* (Lyme disease), tick-borne encephalitis virus, *Herpes simplex virus* 1, 2, 3, 6 — negative. IgG, IgM, PCR DNA of Epstein–Barr virus — negative. Antibodies to *Rubella virus*: IgG — positive (associated with vaccination), IgM — negative. PCR (blood) DNA *Herpes virus* 6, cytomegalovirus — negative. Lumbar puncture performed and no signs of inflammation in the cerebrospinal fluid were observed. Bacteriological examination of cerebrospinal fluid detected no microflora growth. Serological examination of cerebrospinal fluid: *Haemophilus influenzae B Ag, Nesseria meningitidis B Ag, Nesseria meningitidis C Ag, Streptococcus pneumonia Ag, Streptococcus B Ag* were not detected. Blood tests for ganglioside antibodies and neuronal antibodies (Hu, Yo-1, Ri, PNMA2, Cv, GAD, amphiphysin) were negative. Fundus examination showed no pathology. No otorhinolaryngological pathology was detected. Electroencephalography recorded only an EEG pattern of active wakefulness, represented by diffuse non-zoned theta/beta activity, distorted by a significant number of artifacts. The main rhythm is not reliably recorded (a short episode with a frequency corresponding to the age norm cannot be ruled out). Focal and epileptiform activity has not been reliably recorded during the session.

Oncological examination included detection of blood tumor markers (alpha-fetoprotein in blood plasma within normal limits — 1.8 Me/ml), radiological diagnosis of the brain, thymus gland, abdominal organs, retroperitoneal space, small pelvis, and mediastinum.

Neuroimaging. Upon admission, a computed tomography (CT) scan of the brain was performed to rule out a volumetric process in the brain, intracranial hemorrhage, and post-traumatic changes (no pathology).

On the 8th day of the disease, magnetic resonance imaging (MRI) of the brain was performed. Residual changes in the periventricular white matter at the level of the posterior horns of the lateral ventricles were observed on the MRI. After intravenous contrast enhancement of the areas, no pathological signal amplification was detected in the brain substance or meninges.

Ultrasound examination (US) of the abdominal organs revealed no pathological changes, and US of the urinary system organs revealed no pathological changes. According to ultrasound examination of the thymus gland, its size was smaller than the age norm.

X-ray of the chest organs in direct and lateral projection: the lung tissue was swollen; no visible infiltrative changes were detected. There was thickening of the pulmonary pattern in basal sections, more pronounced on the right. The roots of the lungs were poorly structured and not enlarged. The diaphragm was outlined. The lateral sinuses were clear. The heart shadow was not enlarged. An additional intense shadow was detected paravertebrally on the left at the level of Th_8–Th_{11}.

MRI of the abdominal organs showed MR signs of moderate splenomegaly.

MRI of the retroperitoneal space showed a paravertebral mass at the level of the diaphragm on the left, measuring 44×21.1×38.6 mm, widely adjacent to the spine, originating from the intervertebral foramen, limiting diffusion (measured diffusion coefficient (MDC) not less than 0.39×10^{-3} mm^2/s), accumulating contrast agent. Conclusion: MR image of a paravertebral mass on the left at the level of the diaphragm — neuroblastoma?

As a result of the detection of a paravertebral space mass, a consultation with an oncologist was conducted. Conclusion: retroperitoneal space mass (neuroblastoma?), acute cerebellar ataxia, opsoclonus-myoclonus syndrome.

The patient was referred to the N.N. Petrov National Medical Research Center for Oncology of the Russian

Ministry of Health to continue the examination and determine further treatment tactics. MRI of the spinal cord with intravenous contrast was performed at the hospital; no data on spinal cord membrane damage or myelopathy were found. A CT scan with contrast agent was performed on the chest: the intrathoracic lymph nodes were not enlarged. The thymus measured 46×35 mm. There was a soft tissue formation with clear contours, measuring 43×23×43 mm, prevertebrally on the left, at the level of Th_9–Th_{12}. A trephine biopsy of the neoplasm was performed.

After examination at the N.N. Petrov National Medical Research Center for Oncology, a final diagnosis was made: poorly differentiated neuroblastoma of the paravertebral region on the left at the level of Th_9–Th_{12}, stage II according to INSS, stage L2 according to INRGSS. Medium risk group. Complication of the underlying disease: Kinsbourne encephalopathy (opsoclonus-myoclonus syndrome). Amplification of the *N-MYC* gene was negative. Histological assessment of the trephine biopsy in the left paravertebral region identified: columns of poorly differentiated neuroblastoma with foci of undifferentiated neuroblastoma (less than 5% of the tumor). Marker of proliferative activity is low (MKI). Adverse prognosis group. The child underwent three courses of systemic antineoplastic treatment (according to the NB2004 protocol). After the third course, the girl was discharged with significant improvement of her state (no opsoclonus, she could sit down and sit independently, walked with support, freely manipulated objects) . X-ray demonstrated 86% reduction of the paravertebral tumor on the left according to NB. The patient was discharged home under the supervision of an oncologist.

Clinical case No. 2

Parents of patient K., aged 1 year and 10 months, contacted the SPbSPMU Consultative and Diagnostic Center. The parents complained about her motor development delay (she could not walk or stand with support), abnormal eye movements, muscle twitching, unsteadiness, and hyperactivity.

Medical history and history of the disease. Perinatal history is aggravated. The pregnancy was the first, complicated by gestosis and acute respiratory viral infection at 20 weeks. The child was born at 42 weeks, with an Apgar score of 8/9. Psychomotor development was age-appropriate. She began walking independently at 1 year and 1 month and could say a few words. At 1 year and 6 months, her parents noticed a peculiar smell in her urine. An ultrasound examination of the kidneys and urinary tract revealed a mass in the left adrenal gland. Surgical treatment was performed at 1 year and 8 months. After histological examination of the mass, the diagnosis was established: low-grade neuroblastoma of the left adrenal gland. Following surgery, during the early postoperative period, while the girl was in the intensive care unit, tremors were noted in her right arm, then her head, neck, and torso. When treated, she was unable to stand without support or move independently. Her speech development was limited to pronouncing individual words. There was no family history of the condition. The child was hospitalized at the SPbSPMU clinic. *Neurological status.* Overall condition was satisfactory. Cranial innervation: the gaze was fixed and the patient could track objects. The head was turned toward the left shoulder. Eye slits: D=S. Eyeball were twitching. The large fontanelle was closed. Motor skills. The girl held her head up, rolled over, but could not sit up independently. She sat with support. Muscle tone: diffuse muscle hypotonia. Deep reflexes: symmetrical and lively. Meningeal signs: negative. Head circumference: 7 cm. Chest circumference: 47 cm. Intentional tremor. Static-motor ataxia. Brain MRI was performed and showed no pathological changes. Video-electroencephalographic monitoring with sleep recording showed no epileptiform activity and no ictal patterns. Diagnosis: opsoclonus-myoclonus of paraneoplastic origin. Postoperative condition following operative treatment of a mass lesion of the left adrenal gland. Concomitant: hyperexcitability syndrome. Courses of hormone therapy and chemotherapy were performed. After the first course of chemotherapy with cyclophosphamide, the condition worsened. The patient experienced agitation, sleep disturbances, and, after two weeks, vomiting associated with food intake and at night, increased hyperkinesis, and saccadic eye movements. Eye myoclonus appeared when falling asleep. During the next cycle of chemotherapy (cyclophosphamide + dexamethasone), the child's mother reported a reduction in myoclonus. However, the symptoms were not completely eliminated. Four courses of rituximab were administered with positive results. Myoclonus and ataxia decreased, and motor and speech skills improved. The child is being monitored by an oncologist and neurologist at St. Petersburg State Medical University, and rehabilitation is being carried out. Independent walking skills resumed at 3 years and 5 months, and myoclonic jerks were eliminated. Periodically, especially during emotional reactions, abnormal eye movements occur.

DISCUSSION

Specific features of the first case are determined by rapid development of neurological symptoms (within 2 weeks), predominance of increasing trunk and limb ataxia in the neurological status, up to the development of astasia-abasia, relatively late onset of opsoclonus, pronounced behavioral disorders, and autonomic disorders (vomiting, sleep disturbances). The second observation involved the development of opsoclonus-myoclonus syndrome in the early postoperative period in a 1-year-old child diagnosed

with neuroblastoma. It was characterized by the development of pronounced ataxia, myoclonus, and opsoclonic seizures, which persisted for a long time against the background of polychemotherapy. A positive effect was noted after repeated courses of rituximab.

Currently, the algorithm for diagnosing the disease is based on analyzing the clinical picture and ruling out other possible nosological forms with similar manifestations. After establishing the diagnosis of opsoclonus-myoclonus syndrome, it is necessary to identify its etiology, which determines further therapeutic approach.

The lack of established diagnostic criteria leads to late diagnosis of the disease. When the disease manifests as acute ataxia syndrome (especially in the absence of distinct opsoclonus or its later appearance), differential diagnosis is required.

It is recommended to first exclude acute disorders of cerebral circulation, hemorrhages, volumetric formations of the posterior cranial fossa structures, cerebrospinal fluid dynamics disorders, inflammatory diseases of the central nervous system (encephalitis caused by the herpes virus group, encephalitis of other etiologies), acute demyelinating diseases, including autoimmune and genetic diseases, debuting with acute ataxia. Diagnosis of the disease is complicated by the absence of simultaneously developing triad of main symptoms [13].

It is important to note that if the results of cancer screening are negative, repeat imaging (MRI/CT) of the chest, abdomen, and pelvis should be performed at least 6 months later, and a whole-body MRI is also recommended [1, 2, 13].

The clinical picture of the first case included a comprehensive examination of the patient, which made it possible to establish the diagnosis and etiology of the disease within 18 days of its onset and to determine the further treatment strategy.

The choice of the correct route, continuity between medical institutions, and the rapid initiation of systemic antineoplastic therapy contributed to rapid regression of neurological symptoms and the tumor. The second observation demonstrates the need for long-term treatment of opsoclonus-myoclonus syndrome of paraneoplastic origin in cases of persistent neurological disorders.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

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

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Consent for publication. Written consent was obtained from legal representatives of the patients for publication of relevant medical information within the manuscript.

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Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

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

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REFERENCES

1. Narzieva S.P., Marchenko N.V., Tashchilkin A.I., Dubickij D.L., Shevchenko E.Yu., Novokshonov D.Yu., Lopareva D.D., Deryagina A.A., Postanogov R.A., Kucenko V.P. The use and safety of contrast agents in modern imaging in pediatrics. *Visualization in Medicine*. 2025;7(1):27–35. (In Russian). DOI: 10.56871/ViM.2025.92.83.003.
2. Razinova A.A., Pozdnyakova O.F., Kostik M.M., Pozdnyakov A.V., Masalova V.V., Gajdar E.V., Snegireva L.S., Grebenyuk M.M., Drobotov K.P. The role of whole-body magnetic resonance imaging in detecting inflammatory changes in joints in children with juvenile idiopathic arthritis. *Visualization in Medicine*. 2024; 6(3): 19-28. (In Russian). DOI: 10.56871/ViM.2024.68.67.003.
3. Shnajder N.A., Kantemirova E.A., Ezhikova K.A. Opsiklonus-mioklonus. *Nervno-myshechnye bolezni*. 2013;(3):22–26. (In Russian).
4. Bataller L., Rosenfeld M.R., Graus F., Vilchez J.J., Cheung N.K., Dalmau J. Autoantigen diversity in the opsoclonus-myoclonus syndrome. *Ann Neurol*. 2003;53(3):347–353. DOI: 10.1002/ana.10462.
5. Antunes N.L., Khakoo Y., Matthay K.K., Seeger R.C., Stram D.O., Gerstner E., Abrey L.E., Dalmau J. Antineuronal antibodies in patients with neuroblastoma and paraneoplastic opsoclonus-myoclonus. *J Pediatr Hematol Oncol*. 2000;22(4):315–320. DOI: 10.1097/00043426-200007000-00007.
6. Giraldo S.J., Michaelis M., Kerr L. et al. Inaugural patient-reported registry of pediatric opsoclonus-myoclonus-ataxia syndrome: presentation, diagnosis, and treatment of 194 patients. *Pediatric Neurology*. 2024;188:128–134. DOI: 10.1016/j.pediatrneurol.2024.06.007.

7. Kinsbourne M. Myoclonic encephalopathy of infants. *J Neurol Neurosurg Psychiatry*. 1962;25(3):271–276. DOI: 10.1136/jnnp.25.3.271.
8. Koh P.S., Raffensperger J.G., Berry S., Larsen M.B., Johnstone H.S., Chou P., Luck S.R., Hammer M., Cohn S.L. Long-term outcome in children with opsoclonus-myoclonus and ataxia and coincident neuroblastoma. *J Pediatr*. 1994;125(5 Pt 1):712–716. DOI: 10.1016/s0022-3476(94)70062-1.
9. Korfei M., Fühlhuber V., Schmidt-Wöll T., Kaps M., Preissner K.T., Blaes F. Functional characterisation of autoantibodies from patients with pediatric opsoclonus-myoclonus-syndrome. *J Neuroimmunol*. 2005;170(1-2):150–157. DOI: 10.1016/j.jneuroim.2005.08.018.
10. Pang K.K., de Sousa C., Lang B., Pike M.G. A prospective study of the presentation and management of dancing eye syndrome/opsoclonus-myoclonus syndrome in the United Kingdom. *Eur J Paediatr Neurol*. 2010;14(2):156–161. DOI: 10.1016/j.ejpn.2009.03.002.
11. Panzer J.A., Anand R., Dalmau J., Lynch D.R. Antibodies to dendritic neuronal surface antigens in opsoclonus myoclonus ataxia syndrome. *J Neuroimmunol*. 2015;286:86–92. DOI: 10.1016/j.jneuroim.2015.07.007. Erratum in: *J Neuroimmunol*. 2016;300:74. DOI: 10.1016/j.jneuroim.2016.05.001.
12. Pranzatelli M.R. The neurobiology of the opsoclonus-myoclonus syndrome. *Clin Neuropharmacol*. 1992;15(3):186–228. DOI: 10.1097/00002826-199206000-00002.
13. Rossor T., Yeh E.A., Khakoo Y. et al. Diagnosis and management of opsoclonus-myoclonus-ataxia syndrome in children. *Neurol Neuroimmunol Neuroinflamm*. 2022;9(3):e1153. DOI: 10.1212/NXI.0000000000001153.
14. Rothenberg A.B., Berdon W.E., D'Angio G.J. et al. The association between neuroblastoma and opsoclonus-myoclonus syndrome: a historical review. *J Springer*. 2009;39(7):723–726. DOI: 10.1007/s00247-009-1282-x.
15. Tate E.D., Allison T.J., Pranzatelli M.R., Verhulst S.J. Neuroepidemiologic trends in 105 US cases of pediatric opsoclonus-myoclonus syndrome. *J Pediatr Oncol Nurs*. 2005;22(1):8–19. DOI: 10.1177/1043454204272560.

ЛИТЕРАТУРА

1. Нарзиева С.П., Марченко Н.В., Тащилкин А.И., Дубицкий Д.Л., Шевченко Е.Ю., Новокшонов Д.Ю., Лопарева Д.Д., Дерягина А.А., Постаногов Р.А., Куценко В.П. Использование и безопасность контрастных веществ в современной визуализации в педиатрии. *Визуализация в медицине*. 2025;7(1):27–35. DOI: 10.56871/ViM.2025.92.83.003.
2. Разинова А.А., Позднякова О.Ф., Костик М.М., Поздняков А.В., Масалова В.В., Гайдар Е.В., Снегирева Л.С., Гребенюк М.М., Дроботов К.П. Роль магнитно-резонансной томографии по протоколу whole-body в выявлении воспалительных изменений в суставах у детей с ювенильным идиопатическим артритом. *Визуализация в медицине*. 2024;6(3):19–28. DOI: 10.56871/ViM.2024.68.67.003.
3. Шнайдер Н.А., Кантемирова Е.А., Ежикова К.А. Опсиклонус-миоклонус. *Нервно-мышечные болезни*. 2013;(3):22–26.
4. Bataller L., Rosenfeld M.R., Graus F., Vilchez J.J., Cheung N.K., Dalmau J. Autoantigen diversity in the opsoclonus-myoclonus syndrome. *Ann Neurol*. 2003;53(3):347–353. DOI: 10.1002/ana.10462.
5. Antunes N.L., Khakoo Y., Matthay K.K., Seeger R.C., Stram D.O., Gerstner E., Abrey L.E., Dalmau J. Antineuronal antibodies in patients with neuroblastoma and paraneoplastic opsoclonus-myoclonus. *J Pediatr Hematol Oncol*. 2000;22(4):315–320. DOI: 10.1097/00043426-200007000-00007.
6. Giraldo S.J., Michaelis M., Kerr L. et al. Inaugural patient-reported registry of pediatric opsoclonus-myoclonus-ataxia syndrome: presentation, diagnosis, and treatment of 194 patients. *Pediatric Neurology*. 2024;188:128–134. DOI: 10.1016/j.pediatrneurol.2024.06.007.
7. Kinsbourne M. Myoclonic encephalopathy of infants. *J Neurol Neurosurg Psychiatry*. 1962;25(3):271–276. DOI: 10.1136/jnnp.25.3.271.
8. Koh P.S., Raffensperger J.G., Berry S., Larsen M.B., Johnstone H.S., Chou P., Luck S.R., Hammer M., Cohn S.L. Long-term outcome in children with opsoclonus-myoclonus and ataxia and coincident neuroblastoma. *J Pediatr*. 1994;125(5 Pt 1):712–716. DOI: 10.1016/s0022-3476(94)70062-1.
9. Korfei M., Fühlhuber V., Schmidt-Wöll T., Kaps M., Preissner K.T., Blaes F. Functional characterisation of autoantibodies from patients with pediatric opsoclonus-myoclonus-syndrome. *J Neuroimmunol*. 2005;170(1-2):150–157. DOI: 10.1016/j.jneuroim.2005.08.018.
10. Pang K.K., de Sousa C., Lang B., Pike M.G. A prospective study of the presentation and management of dancing eye syndrome/opsoclonus-myoclonus syndrome in the United Kingdom. *Eur J Paediatr Neurol*. 2010;14(2):156–161. DOI: 10.1016/j.ejpn.2009.03.002.
11. Panzer J.A., Anand R., Dalmau J., Lynch D.R. Antibodies to dendritic neuronal surface antigens in opsoclonus myoclonus ataxia syndrome. *J Neuroimmunol*. 2015;286:86–92. DOI: 10.1016/j.jneuroim.2015.07.007. Erratum in: *J Neuroimmunol*. 2016;300:74. DOI: 10.1016/j.jneuroim.2016.05.001.
12. Pranzatelli M.R. The neurobiology of the opsoclonus-myoclonus syndrome. *Clin Neuropharmacol*. 1992;15(3):186–228. DOI: 10.1097/00002826-199206000-00002.
13. Rossor T., Yeh E.A., Khakoo Y. et al. Diagnosis and management of opsoclonus-myoclonus-ataxia syndrome in children. *Neurol Neuroimmunol Neuroinflamm*. 2022;9(3):e1153. DOI: 10.1212/NXI.0000000000001153.
14. Rothenberg A.B., Berdon W.E., D'Angio G.J. et al. The association between neuroblastoma and opsoclonus-myoclonus syndrome: a historical review. *J Springer*. 2009;39(7):723–726. DOI: 10.1007/s00247-009-1282-x.
15. Tate E.D., Allison T.J., Pranzatelli M.R., Verhulst S.J. Neuroepidemiologic trends in 105 US cases of pediatric opsoclonus-myoclonus syndrome. *J Pediatr Oncol Nurs*. 2005;22(1):8–19. DOI: 10.1177/1043454204272560.

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