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Features of Rehabilitation Therapy for Patients with Symptomatic Epilepsy

Fayzullaeva Vazira Khamidovna
Bukhara State Medical Institute named after Abu Ali ibn Sina

Abstract: At present, comprehensive rehabilitation and social integration of children with disabilities is one of the priorities of the state social policy of the Republic of Uzbekistan. However, cerebral palsy is also accompanied by the so-called optional symptoms of damage to the central nervous system, which, although not included in the mandatory pathological symptom complex, significantly complicate treatment and worsen the rehabilitation prognosis of the disease.

Keywords: symptom complex, chronic diseases, symptomatic epilepsy.

The incidence of cerebral palsy (CP) in all countries of the world occupies one of the leading places in the structure of chronic diseases of children, ranging from 1.7 to 7 per 1000 children [22]. The prevalence of cerebral palsy in Uzbekistan, according to various authors, is 6.5-7.5 per 1000 newborns. One of the most common and difficult to treat "companions" of cerebral palsy is symptomatic epilepsy (SE). The presence of SE in a child with cerebral palsy has a negative impact on motor and psycho-speech development, and also significantly limits the possibilities of rehabilitation therapy. Predicting the risk of epileptic seizures in such children even in the early period is the most important guideline in choosing treatment tactics.

In the center of the clinical picture of cerebral palsy are movement disorders (paralysis, paresis, hyperkinesis, ataxia, etc.), on the basis of which the diagnosis is established [9, 10].

A lot of research has been devoted to the study of epilepsy in children with cerebral palsy. According to various publications, depending on the form of cerebral palsy, the risk of developing epilepsy in this disease varies from 15 to 90% [2, 3, 13, 16, 17, 20–23, 25]. In the therapeutic aspect, the main problem of the comorbidity of cerebral palsy and epilepsy is the difficulty of combining the active correction of motor disorders with the antiepileptic treatment regimen, which often leads to the termination of the rehabilitation process and aggravation of the patient's disability.

Risk factors for the development of epilepsy in children with cerebral palsy are: form of cerebral palsy (the highest risk of epilepsy is with spastic tetraplegia and hemiplegic form); the presence of neonatal seizures (NS) in history, which occur during the first 4 weeks of life of a full-term newborn (from the 1st to the 28th day); for premature babies, this period corresponds to the post-conception age; clinical features of neonatal seizures - whether they are fragmentary (oroalimentary, ocular, etc.), clonic, myoclonic, or tonic. Myoclonic seizures have the most severe prognosis; they may indicate the onset of infantile epileptic encephalopathy (Otahara syndrome) or early myoclonic encephalopathy in a child with CNS damage. Clonic NS may in some cases be indicative of benign familial NS or benign idiopathic NS. Up to 90% of cases, the cause of NS is CNS damage, and only in 10% of cases - hereditary factors (idiopathic NS). Risk

factors for the development of epilepsy in children can also include: low birth weight; low Apgar score; heredity burdened by epilepsy; detection of structural disorders of the brain (primarily malformations of the central nervous system, cortical dysplasia) [3, 13, 16, 26].

In general, the prognosis of NS is usually unfavorable; lethality in these cases reaches 40%. In all studies, NS is highlighted as a predictive pattern for the subsequent development of epilepsy in children. Of the surviving children with NS, 27% later develop epileptic seizures and 25% develop cerebral palsy [24].

According to neuroimaging data, structural anomalies of the brain (focal cortical dysplasia, atrophy, periventricular leukopathy, congenital malformations) are determined in 2/3 of cases among children born at term with cerebral palsy, according to neuroimaging data [14, 22]. In premature babies, the morphological basis for the occurrence of neurological disorders is periventricular leukomalacia, which is a local or widespread necrosis of the white matter of the brain, in the region of the anterior horns of the spinal cord, as well as around the occipital and temporal horns of the lateral cerebral ventricles [3, 5].

Patients with cerebral palsy are characterized by the presence of symptomatic forms of epilepsy. However, the possibility of developing an idiopathic form of the disease cannot be excluded. In this regard, when providing care to patients with cerebral palsy, a very thorough analysis of the anamnesis of the patient's parents and EEG is required, and in the future, long-term monitoring of the course of epilepsy with an assessment of the effectiveness of antiepileptic drugs (AEDs).

In 1989, the International Classification of Epilepsy, Epileptic Syndromes and Related Diseases was approved, but it was revised in 2019 by the International Commission on Classification and Terminology. The following clinical manifestations appear in the draft classification of epileptic seizures and epileptic syndromes (2019).

Seizures:

- generalized seizures — seizures are generalized from the very beginning, which is confirmed by EEG data [7]. These include tonic-clonic, clonic, typical absences, atypical absences, myoclonic absences, tonic, epileptic spasms, epileptic myo clonus, eyelid myoclonus, myoclonic-atonic, negative myoclonus, atonic, reflex generalized;
- focal seizures - there is a local nature of paroxysms, focal changes according to the EEG, MRI of the brain. These include focal sensory, focal motor, gelastic, hemiclonic, secondary generalized, reflex focal;
- they also distinguish ongoing seizures (status epilepticus), which are also differentiated as generalized and focal;
- reflex attacks.

Syndromes:

- idiopathic focal epilepsy;
- family (autosomal dominant) focal epilepsy;
- symptomatic (or probably symptomatic) focal epilepsy;
- idiopathic generalized epilepsy;
- reflex epilepsy;
- epileptic encephalopathy;
- progressive myoclonus epilepsy.

Diagnosis and treatment

For the successful treatment of epilepsy, reliable diagnosis and determination of the form of the disease are fundamentally important, since erroneous diagnosis is quite common. Thus,

according to H. Luders and S. Noachtar [19], from 10 to 40% of patients diagnosed with drug-resistant epilepsy suffer from non-epileptic paroxysms and there is reason to consider the diagnosis of epilepsy erroneous [19]. The occurrence of a single seizure in a patient, of course, requires close observation and examination. There are many reasons for the development of seizures in a child, and epilepsy is only one of them, but understanding the etiology of a seizure is essential to determine further treatment tactics. Seizures can be the result of metabolic disorders, the reaction of the immature brain of an infant to fever, one of the clinical manifestations of syncope or an affective-respiratory attack, etc., and at the same time mark the debut of epilepsy. A detailed analysis of the anamnesis, the study of all the details of the situation in which convulsions arose, often allow us to draw correct preliminary conclusions about the nature of the disease.

The diagnosis of epilepsy is based on two main components: the first is the clinical picture of the disease, suggesting the presence of persistent epileptic seizures, and the second is the characteristic changes in the electroencephalogram in the interictal period and during a seizure. In most cases, for example, at an outpatient appointment with a polyclinic neurologist, and often in a hospital, an opinion about the nature of seizures is formed solely from anamnestic data provided by the patient's relatives or witnesses. Since most patients have seizures less than once a day, it is not possible for the attending physician to become an eyewitness to a paroxysm, not to mention studying the nature of the EEG during an attack. In addition, according to our data, EEG performed according to the standard technique (15–20 min of continuous recording) does not reveal pathological changes in patients with epilepsy in about 50% of cases [1]. Thus, there are often difficulties in diagnosing epilepsy and determining its form, which in turn leads to diagnostic errors and the development of inadequate treatment tactics. The use of video-EEG monitoring makes it possible to avoid such errors in the vast majority of cases, as well as to reliably establish the localization of the epileptogenic focus.

The diagnosis of epilepsy in a child with cerebral palsy should in no way lead to the rejection of rehabilitation measures within the framework of the underlying disease, but the detection of concomitant pathology naturally dictates the need to correct the rehabilitation treatment plan. The first stage of the new rehabilitation scheme should be the implementation of drug control over epileptic seizures.

Therapy for epilepsy begins with the appointment of the drug of first choice with a minimum dose with a slow gradual increase in dose until a therapeutic effect is achieved or adverse reactions occur. The effectiveness of treatment is assessed clinically by the degree of relief of epileptic seizures and by the dynamics of the EEG pattern. Also, in dynamics, adverse events that occur while taking anticonvulsants are evaluated (assessment of a blood test, liver, kidney function, etc.).

If the drug is ineffective at the maximum therapeutic dose, another AED is prescribed, also from the minimum dose with a gradual increase to the therapeutic one. The first drug is gradually canceled after reaching therapeutic concentrations and the clinical effect of the second AED (Table 1).

If monotherapy with basic drugs is ineffective, polytherapy can be used [7]. In clinical practice, combinations are used: valproate + topiramate, valproate + levetiracetam, valproate + carbamazepine, carbamazepine + topiramate, valproate + lamotrigine, carbamazepine + levetiracetam, topiramate + levetiracetam. With secondary generalized seizures and the phenomenon of secondary bilateral synchronization, it is possible to prescribe a combination of topiramate or valproate with succinimides.

When choosing an anticonvulsant for combination therapy, it is necessary to take into account the initial somatic status of a child with cerebral palsy. As a rule, children with spastic and hyperkinetic forms of cerebral palsy are hypotrophic and have a variety of functional disorders of the gastrointestinal tract. When prescribing long-term combined antiepileptic treatment, it is

necessary to take into account the possible adverse events that the recommended therapy can cause. It is advisable to choose anticonvulsants for polytherapy - synergists in terms of therapeutic effect and antagonists in terms of adverse reactions. This is not always possible, however, there are drugs that are least interested in the metabolism of other anticonvulsants in the liver (levetiracetam), thus, the combination of levetiracetam with other AEDs (especially GABAergic ones) leads to a synergistic effect in preventing epileptic seizures in the absence of competitive interactions, causing additional side effects.

Epileptic syndromes in patients with one of the most severe forms of cerebral palsy, spastic tetraparesis (double hemiplegia), are represented with a high frequency by West syndrome (up to 15%) and Lennox–Gastaut syndrome [15, 17, 18, 21, 23]. West syndrome belongs to the group of age-dependent epileptic encephalopathies of infancy and is a symptomatic or presumably symptomatic generalized form of epilepsy. The debut of the disease is typical in the first year of life (usually at the age of 3 to 7 months). A feature of West's syndrome in most children with periventricular leukomalacia, according to O.N. Malinovskaya et al. [5], is the connection between the onset of seizures and active stimulating therapy (taking nootropic drugs or repeated courses of active massage). At the same time, on the interictal electroencephalogram in all patients with West syndrome before treatment, hypsarrhythmia is detected - typical or atypical [11].

When monotherapy is ineffective in patients with West syndrome, AED combinations are used: vigabatrin + valproate, valproate + topiramate, valproate + succinimides (ethosuximide at a therapeutic dose of 20-35 mg / kg per day in 2-3 doses), valproate + benzodiazepines.

Lennox-Gastaut syndrome belongs to the group of epileptic encephalopathies of childhood and is a symptomatic or presumably symptomatic generalized form of epilepsy. In many cases, the development of Lennox-Gastaut syndrome in a child is preceded by West syndrome. The onset of the disease is typical between the ages of 2 and 8 years. For the correction of seizures, combinations of AEDs are used: topiramate + valproate, valproate + suxinimides, valproate or topiramate + lamotrigine. The prognosis of the disease is usually poor, remission can be achieved in a very small percentage of cases.

Thus, patients with cerebral palsy can experience any type of seizures: neonatal, febrile convulsions, symptomatic partial epilepsy of any localization, myoclonic seizures, epileptic encephalopathies: West syndrome, Lennox-Gastaut syndrome. In all forms of cerebral palsy, secondary generalized tonic-clonic seizures are predominant. Among the earliest seizures in children with cerebral palsy, according to various international studies, myoclonic seizures and infantile spasms are noted. Secondary generalized seizures develop, as a rule, after myoclonic, partial seizures and infantile spasms. In children with spastic hemiplegia, partial and secondary generalized seizures occur with equal frequency. West's syndrome is observed with great frequency in patients with spastic tetraplegia.

The effectiveness of antiepileptic therapy in children with cerebral palsy, depending on the form of the disease, according to various researchers, ranges from 50 to 85%. Seizure control can be achieved with spastic tetraplegia in more than 60% of cases, with spastic hemiplegia - in 72.7% of cases, with spastic diplegia - in 83.3% of cases. The need for polytherapy most often occurs in cases of spastic tetraplegia - in 59.5% of cases [13]. An unfavorable factor in the resistance of epilepsy, in addition to the form of cerebral palsy, is the development of the first epileptic seizure in the first year of a child's life. Among the prognostically favorable factors for the course of epilepsy in cerebral palsy, there are: normal and subnormal intellectual development; rare seizures with a late onset; positive effect of monotherapy; spastic diplegia [ ].

Stages of rehabilitation of children with cerebral palsy and epilepsy. The difficulties of selecting multicomponent antiepileptic therapy in patients with resistant forms of epilepsy in cerebral palsy, as a rule, are undertaken by epileptologists, however, the main specialist who dynamically monitors a patient with cerebral palsy at various stages of its development and rehabilitation is

an outpatient neurologist. The competence of a polyclinic neurologist includes: prevention of epileptic seizures and referral for primary diagnosis of epilepsy, prescription of basic AEDs and referral to an epileptologist in case of failure of basic therapy, deciding on the timing and scope of rehabilitation and spa treatment of a child with cerebral palsy.

Thus, to optimize care for children with cerebral palsy and epilepsy, it is necessary to determine the general tactics of managing such patients by outpatient neurologists.

I stage. Identification of children with cerebral palsy with a maximum risk of presentation of convulsive conditions. For this purpose, all patients with cerebral palsy should undergo an EEG study at least once a year. When specific epileptic activity is detected in the EEG, the frequency of dynamic EEG conduction increases up to 1 time in 3-6 months, drug-induced (nootropic drugs with high epileptogenicity) and non-drug (intense massage, electroprocedures) rehabilitation are reviewed. The presence of epileptic activity in the EEG should in no case be the reason for stopping restorative treatment, however, a frivolous attitude to the identified changes is also unacceptable. Non-epileptogenic rehabilitation methods are: therapeutic exercises, positional treatment, orthotics, cognitive and speech rehabilitation. Non-aggressive acupuncture, hydrotherapy, balneotherapy, magnetotherapy, local thermotherapy with rational dosing can also be used in this category of patients.

II stage. First seizure. An outpatient neurologist analyzes in detail the situation in which the paroxysm occurred and its nature. If an epileptic seizure is suspected, the patient is prescribed an examination in the form of an unscheduled EEG and neuroimaging of the brain (CT / MRI). If the diagnosis of epilepsy is confirmed, the patient receives basic antiepileptic prescriptions from a neurologist or is referred to an epileptologist or a specialized epileptological hospital, depending on the severity of the condition and the frequency of seizures. At this stage, the main task of the neurologist is the diagnostic search and prevention of recurrent attacks, so the volume of rehabilitation treatment is temporarily reduced to a minimum, but does not stop.

III stage. The epileptologist analyzes the history, seizure semiotics, EEG patterns, and neuroimaging findings. Based on the data obtained, the form of epilepsy is established and the appropriate AED is selected. An adequate dose of AED is selected using the pharmacomonitoring method. Efficacy and tolerability of treatment is assessed. If there are indications, the patient is sent for examination to a neurogeneticist.

Against the background of effective antiepileptic therapy, carried out in an adequate dosing regimen, upon reaching remission lasting 3 months. the volume of rehabilitation can be gradually expanded. Regular dynamic analysis of electroencephalographic data makes it possible to regulate the intensity of rehabilitation loads in such patients.

IV stage. Observation by a neurologist of a polyclinic of a patient with an established diagnosis of epilepsy. Efficacy and safety of treatment are evaluated. In the case of taking a number of anticonvulsants, periodic monitoring of the functions of internal organs and body systems is required. For example, taking valproates requires regular monitoring of the functions of the liver, pancreas, and hematopoietic system. Carbamazepine, as well as valproate, can cause hematopoietic dysfunction. Topiramate can cause precipitation of salts in the kidneys, leading in some cases to the formation of calculi, which requires monitoring of the level of salts in the urine and periodic ultrasound examination of the kidneys. A control examination by a polyclinic neurologist under the condition of remission is carried out at least once every 3 months, and by an epileptologist at least once every 6 months. In case of recurrence of seizures, the adequacy of dosages of anticonvulsants is assessed or the treatment regimen is reviewed.

Pharmacoresistance of epilepsy is defined as the failure of two basic, appropriately prescribed anticonvulsants, used at maximum tolerated doses as monotherapy or in combination with each other. The fact of pharmacoresistance in older patients is recognized when the duration of epilepsy is more than 2 years. In infantile forms of symptomatic and cryptogenic epilepsy, or if the patient has one of the forms of symptomatic epileptic encephalopathy, drug resistance is

already established at the time of diagnosis. In case of drug-resistant course of epilepsy, the patient is necessarily sent to a specialized epileptological hospital for an in-depth examination and decision on further treatment tactics (surgical treatment, ketogenic diet, VNS-therapy).

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