

Cerebral Palsy

G. T. Madjidova

Samarkand State Medical University, 2nd Assistant of the Department of Internal Medicine and Cardiology, Samarkand Uzbekistan

G. B. Nuralieva, A. X. Buribaeva

Doctor's office, Samarkand branch of the Republican Scientific Center for Urgent Ambulance, Samarkand Uzbekistan

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Annotation: Cerebral palsy (CP) is a non-progressive neurological disorder resulting from damage to the central nervous system during the prenatal, perinatal, or neonatal periods. It is a leading cause of childhood disability, with a prevalence of 2-2.5 cases per 1000 children. This condition often coexists with various intellectual, speech, and paroxysmal disorders. Classification of CP is complex due to its heterogeneous nature and is based on predominant movement disorders, which can be spastic, dyskinetic, ataxic, or mixed. Approximately 50% of cases occur in children born prematurely, with risks increasing as gestational age and birth weight decrease. Risk factors for CP include maternal infections, coagulopathies, and complications during birth. Symptoms range from motor impairments to cognitive and behavioral disorders, with the diagnosis typically confirmed by 12-18 months of age. While CP cannot be cured, rehabilitation efforts can significantly improve functional outcomes. These efforts may include physiotherapy, occupational therapy, speech therapy, and pharmacological treatments aimed at symptom management. Surgical interventions may be considered in cases of severe spasticity or contractures.

Keywords: Cerebral palsy, childhood disability, motor disorders, rehabilitation, risk

factors, diagnosis, treatment, physiotherapy.

Introduction. The leading places in the structure of primary childhood disability are occupied by congenital developmental anomalies, diseases of the nervous system and mental disorders. Among diseases of the nervous system, the main cause of childhood disability is cerebral palsy, the prevalence of which is 2–2.5 cases per 1000 children [1].

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On average, the number of children with perinatal pathology in Uzbekistan is about 24% of the total child population.

In 86% of cases, neurological diseases leading to disability in children are a consequence of pathology of pregnancy and childbirth, and the outcome of such pathology in 30% of cases is recovery, in 30% - persistent disability, in 40% of cases - conditionally disabling conditions, which with timely and proper rehabilitation can be partially or even completely reversible.

At the same time, infancy is a unique time interval during which, with timely and adequate treatment, the outcome is most promising, whereas with age, the child's rehabilitation potential decreases, which is accompanied by a noticeable decrease in the response to therapy [2]. In our country, official statistical accounting of childhood disability was introduced in 1996. During the first reporting decade, the overall disability rates in the 0-4 age group increased in all federal districts, with the maximum increase values (+59.1%). The maximum increase in the overall disability rate in children aged 0-4 was recorded in the Jewish Autonomous Region (+280%). The latter indicator, on the one hand, is certainly associated with the growth of disabling diseases in the child population, and on the other hand, is indirect evidence of the early alertness of specialists and the high detection rate of these pathologies in children.

Classification Cerebral palsy (CP) is a term used to describe a group of non-progressive disorders of body posture and movement caused by damage to the central nervous system (CNS) that occurred in the antenatal, intranatal, or neonatal period. Movement disorders characteristic of CP are often accompanied by intellectual, mnemonic, speech, and paroxysmal disorders [3].

Since CP is not a single nosological form, it is difficult to classify. Various classifications of CP are based on the predominant movement disorders: for example, one classification suggests the presence of four main types of cerebral palsy (spastic, athetoid, ataxic, and mixed); another classification distinguishes three categories of movement disorders:

spastic - with increased muscle tone and tendon reflexes (upper or lower paraparesis, tetraparesis, unilateral or double hemiplegia);

dyskinetic - with impaired coordination and adequacy of muscle tone regulation (athetoid or hyperkinetic form);

ataxic - with impaired coordination of voluntary movements (atonic -astatic or mixed forms of cerebral palsy).

About 50% of all cases of cerebral palsy are disorders in children born prematurely. Achievements in healthcare in the field of nursing premature babies have significantly increased the survival rate of these fragile newborns. However, statistics inexorably show that as the gestational age and weight of the fetus at birth decrease, the risk of perinatal pathology resulting in cerebral palsy increases sharply. For example, compared to full-term children, the risk of cerebral palsy in children born at 37-41 weeks of pregnancy increases by 5 times, and in children born before 28 weeks of gestation - almost 50 times [4].

The increased risk of developing cerebral palsy in premature babies is associated with two main groups of factors. Firstly, prematurity is always associated with a high frequency of complications during nursing, such as intracerebral hemorrhages, infections, respiratory and cardiac disorders. Secondly, the onset of premature birth can be caused by pregnancy complications, which at the time of birth have already caused neurological damage to the fetus. More often, in the pathogenesis of severe perinatal lesions of the central nervous system of a premature baby, a combination of the listed factors occurs. Moreover, statistically shown tendencies to premature birth in some families, but the genetic mechanisms of familial forms of prematurity are not yet entirely clear.

Despite the fact that cerebral palsy is the leading cause of childhood neurological disability worldwide, and scientists and doctors around the world are concerned about the fight against this pathology, over the past 20 years, the incidence of cerebral palsy has tended not to decrease, but to increase. It should be noted that modern advances in medicine have a dual effect on cerebral palsy statistics. On the one hand, such risk factors for pathology as Rh-conflict between mother and fetus with subsequent development of bilirubin encephalopathy are currently well controlled and treated, which has somewhat reduced the incidence of hyperkinetic forms of cerebral palsy, on the other hand, the proportion of such risk factors for cerebral palsy as premature birth and multiple pregnancies has increased significantly.

Historically, at different times, various pathogenetic mechanisms were attributed to cerebral palsy. In the twentieth century, the main concept of the formation of cerebral palsy was considered to be mechanical birth trauma leading to asphyxia of the fetus and newborn. However, epidemiological studies have shown that mechanical birth trauma is the cause of only 5-10% of cerebral palsy cases [5].

Other putative mechanisms of cerebral palsy pathogenesis are: disruption of the formation of brain structures, prenatal factors that directly or indirectly damage neurons of the developing nervous system, as well as various "sufferings" of the brain during the first 7 days of life.

It should be noted that if domestic authors traditionally consider cerebral palsy to be the result of damage to the central nervous system that occurred in the perinatal period, then foreign authors, as a rule, include in the statistics of cerebral palsy all cases of ischemic -traumatic brain damage that occurred in the life of a fetus, newborn and a child of the first three years of life and led to the formation of a symptom complex of motor disorders characteristic of cerebral palsy.

Taking into account the leading etiopathogenetic causes of cerebral palsy, all cases of the disease can be conditionally divided into two large groups: genetic and non-genetic, but most patients will be somewhere in the middle. Therefore, it is preferable to use a classification based on the time of exposure to the pathological factor and to distinguish prenatal, intranatal and postnatal groups of causes of the disease. Cases of cerebral palsy associated with multiple pregnancies and premature births should be considered separately.

Risk factors for the development of cerebral palsy Prenatal factors During the complex process of antenatal formation, the fetal CNS is exposed to the potential risk of various anomalies. Some of these anomalies may affect the structure of the brain, while others, visually undetectable, may cause significant conduction disturbances in the cerebral cortex. Anomalies in the structure or conduction system of the CNS may be both hereditary and sporadic.

Some infectious diseases of the mother and fetus increase the risk of cerebral palsy, including rubella virus, herpes, cytomegalovirus (CMV), toxoplasmosis. Each of these infections is potentially dangerous to the fetus only if the mother encounters it for the first time during pregnancy or if the infection actively persists in her body. Most women are immune to these infections by the time they reach childbearing age, but it is advisable to determine a woman's immune status using a TORCH test (toxoplasmosis, rubella, cytomegalovirus, herpes) before and during pregnancy.

Just like an adult, a fetus can have a stroke during intrauterine development. Fetal stroke can be

either hemorrhagic (bleeding due to damage to a blood vessel) or ischemic (due to embolism of a blood vessel). Both children with cerebral palsy and their mothers are significantly more likely than the general population to have various coagulopathies, which cause a high risk of intrauterine episodes of hyper- or hypocoagulation. Both specific nosological pathologies of the blood coagulation system and deficiencies of individual coagulation factors, thrombocytopenia, etc. may be hereditary.

In general, any pathological factor affecting the fetus's central nervous system antenatally may increase the risk of subsequent developmental disorders in the child. In addition, any pathological factor that increases the risk of premature birth and low birth weight, such as alcohol, tobacco, or drugs, also exposes the child to the risk of subsequent physical, motor, and mental defects.

Moreover, since the fetus receives all nutrients and oxygen from the blood that circulates through the placenta, anything that interferes with the normal function of the placenta may adversely affect fetal development or increase the risk of premature birth. Therefore, pathological neoplasms or scars of the uterus, structural abnormalities of the placenta, premature detachment of the placenta from the uterine wall, and placental infections (chorioamnionitis) also pose a danger in terms of disruption of the normal development of the fetus and child.

Certain maternal illnesses or injuries during pregnancy may also pose a risk to fetal development, leading to the development of neurological pathology. Women with autoimmune antithyroid or antiphospholipid antibodies also have an increased risk of having a child with neurological disorders. A potentially key factor here is high levels of cytokines in the blood of the mother and fetus, which are proteins associated with inflammation, such as in infectious or autoimmune diseases, and can be toxic to fetal neurons.

Severe physical injuries to the mother during pregnancy may result in direct injury to the fetus or compromise the availability of nutrients and oxygen to the developing organs and tissues of the fetus.

Intranatal factors

Severe birth asphyxia is not so common in developed countries today, but it is quite sufficient to lead to the development of severe motor and mental disorders in the future. The causes of asphyxia may be mechanical: for example, tight umbilical cord entanglement around the fetus's neck, its prolapse and loss, as well as hemodynamic: bleeding and other complications associated with premature placental abruption or its pathological presentation.

Particular attention should be paid to infectious factors. It should be taken into account that infections are not necessarily transmitted to the fetus from the mother through the placenta; infection can occur directly during childbirth. Postnatal factors About 15% of cases of cerebral palsy in children are caused by factors affecting the child's body after birth.

Incompatibility of the mother and child in blood type or Rh factor can lead to fetal bilirubin encephalopathy (the so-called "nuclear jaundice"), fraught with the formation of hyperkinetic or dyskinetic syndromes. Prevention of this serious complication involves routine screening of Rh-negative women for anti-Rh antibodies after each birth of a Rh-positive child and the level of hemoglobin and bilirubin in the newborn.

Serious infections that directly affect the brain, such as meningitis and encephalitis, can also cause irreversible damage to the brain, leading to persistent disabling deficits in motor skills and mentality.

Neonatal seizures can both directly cause damage to the central nervous system and be a consequence of other hidden pathological factors (encephalitis, stroke, metabolic defect), which will also contribute to the formation of persistent deficits in motor skills and mentality.

When discussing postnatal causes of cerebral palsy, it is necessary to recall once again that in most foreign countries, CP is considered a symptom complex of persistent disabling motor disorders

that arise as a result of the impact of pathological factors on the central nervous system of the fetus and child antenatal, intranatal or postnatal before the child reaches the age of 3-4 years. Thus, according to foreign standards, the category of patients with CP may include patients with the consequences of physical injuries, drowning, suffocation, intoxication, which led to persistent neurological disorders [6].

Symptoms

Symptoms of CP can be conditionally divided into main and concomitant. The main symptoms should include the immediate results of damage to the structures of the central nervous system, leading to a violation of the motor and coordination function (paresis and spasticity of muscles, coordination and fine motor disorders, dystonic attacks and hyperkinesis).

Although cerebral palsy is not a progressive disease, the degree and severity of its main symptoms may change over time and may be accompanied by complications (for example, long-term spasticity may lead to contractures, abnormal postural settings, and joint and limb deformities that require surgical correction).

Associated neurological manifestations of cerebral palsy are also a consequence of CNS damage, often accompany cerebral palsy, but are not its obligatory companions and are not related to the motor sphere: Intellectual/cognitive disorders and behavioral disorders. Epilepsy and other paroxysmal disorders. Visual and hearing impairment. Speech (dysarthria) and nutritional disorders. Associated symptoms of cerebral palsy often reduce the quality of life of patients more than the main symptoms of the disease. As an example, the following statistics can be given: about 1/3 of patients with cerebral palsy have moderate and severe intellectual and cognitive impairment, about 1/3 have mild intellectual and learning disabilities, and only 1/3 of patients with cerebral palsy have intact intelligence. **Diagnosis** There is no universal diagnostic test for cerebral palsy, but a number of factors may attract the doctor's attention immediately after the child's birth: a low score on the Apgar scale, the presence of abnormal muscle tone and movements. However, most often the diagnosis is determined by the end of the first 12-18 months of the baby's life, when, compared with healthy peers, the pathology of the motor system becomes obvious [7]. In the diagnosis of cerebral palsy, paraclinical methods are actively used, for example, ultrasound, computer magnetic resonance neuroimaging (detection of periventricular leukomalacia, ventriculomegaly, foci of ischemia or hemorrhage, or structural abnormalities of the central nervous system). Neurophysiological studies (electroencephalography, electromyography, recording of evoked potentials) and laboratory (biochemical analyses, genetic tests) are usually used to identify pathological conditions often associated with cerebral palsy (optic nerve atrophy, hearing loss, epileptic syndromes) and differential diagnostics of cerebral palsy with many hereditary and metabolic diseases that debut in the first year of a child's life [8].

Rehabilitation treatment Cerebral palsy cannot be cured, which is why we are talking about restorative treatment or rehabilitation, but timely and correct restorative treatment can lead to a significant improvement in the functions impaired by the disease. The program of restorative treatment of a child with cerebral palsy depends on the severity, nature and predominant localization of symptoms, as well as on the presence or absence of hearing, vision, behavioral, cognitive impairment, etc. associated with cerebral palsy. The most serious obstacles to the rehabilitation of a child with cerebral palsy are concomitant impairment of intelligence and cognitive activity, which interferes with adequate interaction between the patient and the instructor, and epileptic seizures, which, in the absence of drug control, can create a risk of life-threatening complications for the child against the background of active stimulating treatment.

However, today special "soft" rehabilitation programs for children with epilepsy have been developed, as well as methods of communication with intellectually impaired patients with cerebral palsy, that is, for each patient an individual rehabilitation program can and should be developed, taking into account his capabilities, needs and problems. The duration of rehabilitation treatment for a patient with cerebral palsy is not limited, while the program must be flexible and

take into account the constantly changing factors of the patient's life. The main goal of rehabilitation in cerebral palsy is the adaptation of a sick person in society and his full and active life.

Physiotherapy, occupational therapy and speech therapy Muscle spasticity, paresis, posture and gait disorders significantly affect the mobility of patients with cerebral palsy. Physiotherapy for this disease is aimed at maximizing the development of the patient's existing motor abilities and compensating for existing motor disorders using so-called "technical rehabilitation devices", which include wheelchairs, walkers, shoe inserts, crutches, orthoses, and devices [9]. Speech therapy, both traditional and high-tech (implementation of computer-controlled communication devices for patients with severe dysarthria) can fundamentally change the quality of life of a patient with cerebral palsy, creating the possibility of social communication [10].

Drug treatment Drug treatment of cerebral palsy is exclusively symptomatic and should be aimed at correcting a particular symptom or complication of the disease. The main problem is that from the point of view of evidence-based medicine, only a few drug-based therapeutic methods have received statistical confirmation of their effectiveness and safety in patients with cerebral palsy. These methods include the use of antispasmodic drugs, in particular, botulinum toxin, nootropic drugs, anticonvulsants [11]. At the same time, a number of previously accepted drug-based methods are now completely discredited (for example, alcohol and phenol blockades causing local muscle necrosis), and some methods (for example, oxygen barotherapy) are used without ever having been specifically studied for this disease [12].

Surgical treatment Fixed contractures developing as a result of long-term muscle spasticity are usually considered from the point of view of the advisability of surgical intervention. The most frequently used surgical procedures for cerebral palsy are tenotomies, the purpose of which is the maximum possible return of the limb to a normal, supporting position. In cases of severe symmetrical spasticity that is not amenable to medication and is fraught with pain or joint complications, a neurosurgeon may use spinal rhizotomy to interrupt the transmission of pathological impulses from the spinal cord to the affected muscle groups [13,14].

Forecast Cerebral palsy affects the quality of life of a sick person at any stage, from childhood and adolescence to maturity and old age. And at each stage, a patient with cerebral palsy should be accompanied by active rehabilitation support. To date, the average life expectancy of 90% of patients with cerebral palsy does not differ from that in the population as a whole [15,16].
Prevention The main modern targets for the prevention of cerebral palsy in the population are: early diagnosis and treatment of coagulopathies and chronic infections in women of childbearing age, prevention and adequate treatment, as well as health education work among teenage girls about the need for proper nutrition, quitting alcohol and smoking, and the importance of timely treatment of chronic diseases.

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