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Status of Newborns with Perinatal Central Nervous System Lesions

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Abstract: According to United Nations estimates, disabled people make up 10% of the world's population, which causes a number of moral, social and economic problems, the level of their resolution is one of the most important features of modern society. Among childhood disabilities, diseases of the nervous system - 19.5%, mental diseases - 14.3% and congenital anomalies - 21% prevail. In 60% of cases, neurological disability in childhood is associated with the pathology of the perinatal period, while 24% are patients with cerebral palsy. Every third child in the Russian Federation has various neuropsychic diseases, 80% of which are caused by perinatal factors, which determines the high social importance of this problem.

Perinatal lesions of the nervous system represent a large group of pathological conditions combined with the time of negative impact on the fetus and newborn. The perinatal period begins from the 22nd full week of intrauterine life of the fetus and ends 7 days after birth. Intranatal - the period during childbirth. Neonatal - includes 28 days after birth. Embryo - from the time of conception to 10 weeks. Embryopathy is a defect between the 16th day after fertilization and the end of the 10th week of pregnancy. Fetopathy - damage from 11 weeks to the end of birth. (Professor EV Shnitkova. 2022).

Keywords: Toxic-metabolic disorders of the nervous system, Causes of hypoxia, Effects leading to PPCNS, Birth trauma.

This classification involves the division of neurological diseases of the newborn period into 4 main groups depending on the leading mechanism of injury: I hypoxic, II traumatic, III toxic-metabolic and IV infectious.

The following are distinguished in each of the groups: the main neurological symptoms and syndromes in nosological form; The clinical course of PPCNSL is divided into 3 main periods: acute (1 month of life), recovery, early (up to 4-5 months) and late (up to 1 year, in premature babies - up to 2 years) and residual period of secrets (or consequences) - a pathology in a child older than 2 years.

Before birth, the human brain is similar to the adult brain, and during life it increases 3 times, and the body weight increases 20 times. At the time of birth, the brain has a complete set of nerve cells released for life - about 150 billion neurons . During life, new neurons are not formed, and part of the information received from the birth of cells is systematically destroyed to ensure its stable operation.

1. Hypoxic injury of the central nervous system.

Chronic hypoxic distress syndrome or chronic hypoxia is a condition that occurs not as a result of direct damage to fetal tissues, but indirectly as a result of damage to the mother's body, placental tissues or umbilical cord. Chronic hypoxia takes a leading place among the causes of damage to the central nervous system in the perinatal period, which is an independent

nosological unit of diseases of the fetus and newborn, accounting for 50% of perinatal morbidity and mortality. Professor EV Shnitkova, 2022)

Causes of hypoxia:

- age of primiparous women (under 18 or over 30);
- Violation of the physiological course of pregnancy leads to the development of chronic intrauterine hypoxia: disorders of peripheral blood flow, deterioration of oxygen transport and somatic and infectious diseases of women with metabolic diseases; burdened obstetric history (abortions, abortions, stillbirths, infectious and endocrine gynecological diseases); wrong position; pathological course of pregnancy: gestosis, preeclampsia, hypertonicity of the uterus, impaired placental blood flow, fetoplacental insufficiency, venous thrombosis, preservation of uterine tone.

Asphyxia of the newborn (acute hypoxia): a syndrome characterized by a lack of breathing or irregular breathing in the presence of cardiac activity - an acute cessation or limitation of oxygen, an excess of carbon dioxide and poorly oxidized products a complex multiphase process resulting from planing. organic acids in the body.

Risk factors for the development of asphyxia in newborns: premature birth; diabetes mellitus, maternal obesity; gestosis (eclampsia); chronic hypertension (maternal hypertension); ultrasound signs of maternal Rh immunization, anemia or fetal hydrops; stillbirth or birth of previous children in a state of severe asphyxia; clinical signs of an infectious disease in the mother before or during childbirth (chorioamnionitis, fever before or immediately after childbirth); congenital infection of the fetus was suspected.

Intrapartum risk factors: preterm birth (less than 37 weeks); late birth (more than 42 weeks); acute fetal hypoxia during childbirth; uterine rupture; - amniotic fluid embolism; fall/shock of a woman during childbirth (of any etiology); separation of the placenta; previous position of the placenta; placenta accreta; loss of umbilical cord loops; pathological condition of the fetus; use of general anesthesia during childbirth; labor anomalies; the presence of meconium in the amniotic fluid; • violation of the fetal heart rhythm; shoulder dystocia; instrumental birth (obstetric forceps, vacuum extraction).

Consequences. The consequence of insufficient perfusion and oxygenation of tissues can lead to damage or death of the brain and other organs, deterioration of cerebral blood circulation leads to increased intracranial pressure, which often leads to extensive necrosis of brain tissue.

The leading basis of the pathogenesis of perinatal hypoxic lesions of the central nervous system in newborns: structural disorganization of cell membranes, changes in transmembrane transport activity.

In the brain of a child who has experienced a hypoxic effect, two processes occur in parallel: the restoration of nerve tissue and its degeneration. In this regard, the recovery phase can continue indefinitely. The phenomenon of delayed psychoneurological disorders is observed. Neurological recovery cannot be equated with recovery. Children who have experienced hypoxia in the perinatal period have brain weakness, which is determined under increased stress (neuroinfection, stress, trauma, etc.).

2. Birth trauma.

This is a birth due to the mechanical impact of mechanical forces directly on the fetus, not on the placenta or umbilical cord, manifested by a harmful effect in the form of rupture, fracture, stretching, dislocation. local damage to fetal tissues during childbirth. as well as local blood circulation disorders in the presented part (venous stagnation, stasis, edema, bleeding thrombosis).

Causes of birth injury: inconsistency between the size of the pelvis of the mother and the size of the fetus; incorrect location or insertion of the fetus; pathology of pelvic organs and bones of the mother.

Anomaly of labor: fast, fast, protracted labor; obstetric intervention: vacuum extraction, extraction by the pelvic cavity, use of forceps during cesarean section, iatrogenics; prolonged labor in combination with perinatal hypoxia, rapid rotation of the head, forced extraction of the fetus.

Causes of spinal cord injury, coagulopathy. Vascular malformations and tumors are predisposing factors. and abnormalities in the appearance of the fetus (breast); incorrect performance of obstetric care, excessive lateral traction or rotation of the body with a rigid head; predisposing factors - hypoxia, coagulopathies, vascular malformations.

Causes of birth damage to the peripheral nervous system: abnormal (extensor) appearance of the fetus, surgical delivery - incorrect use of abdominal (less protruding) obstetric forceps

Temporary metabolic diseases: a critical level of indirect bilirubin in the blood (isoimmunization, hemolysis, bleeding, infections, polycythemia, liver damage, etc.).

3. Toxic-metabolic disorders of the nervous system.

Toxic damage to the nervous system of the fetus and newborns when using anesthesia and analgesics for the mother during pregnancy, labor and delivery, when the mother takes opiates and tranquilizers, when she consumes tobacco, alcohol and other drugs in the perinatal period. addiction.

4. Damage to the central nervous system due to infectious diseases of the perinatal period.

- Damage to the central nervous system in neonatal sepsis.
- Damage to the central nervous system during intrauterine infections (TORCH or STARCH syndrome): cytomegalovirus infection, toxoplasmosis, congenital rubella, ECHO viruses, syphilis.

The danger of STARCH syndrome and other viral infections is that they cause 80% of birth defects in children. Among them, damage to the central nervous system, congenital defects of the heart and kidneys, other organs and systems of the body were found to lead to infection of the fetus in the first trimester of pregnancy, micro-, hydrocephalus, heart defects, gastrointestinal tract, genitourinary systems, skeletons, cataracts were found. , the development of deafness. Infection in the second and third trimesters causes fetal hepatosplenomegaly, anemia, jaundice, malnutrition, pneumonia, meningoencephalitis, and sepsis.

The classification of the consequences of perinatal lesions of the nervous system in children in the first year of life by the Russian Association of Perinatal Medicine Specialists (2005) is presented in Table 2.

Etiopathogenetic groups of effects leading to PPCNS (ICD-10 codes are shown in parentheses) (Studenikin VM, Khachatryan LG, etc.). Five etiopathogenetic groups of effects have been identified:

hypoxia (ischemia - P91.0, hemorrhage - P52.0);

birth injury (brain - P10.0, spinal cord - P11.5, peripheral nerves - P14);

metabolic diseases (carbohydrates - P70, Ca/Mg - P71.0, hypovitaminosis K - P53);

toxic effects on the nervous system (P04);

infectious and parasitic diseases (viral - P35, bacterial sepsis - P36, parasitic - P37).

Severity of PPNS: In children in the first year of life, there are two main periods of PPNS: the period of formation of a neurological defect (1-3 months) and the recovery period (duration 3-12 months). For premature babies, the recovery period of PPNS can be extended up to 24 months.

Clinical syndromes in the formation of a neurological defect:

brain excitability syndrome - P91.3; brain depression syndrome - P91.2; syndrome of vegetative-visceral disorders - G90.8; cerebrovascular distension syndrome (intracranial hypertension) - G91.8; convulsive syndrome - P90.0; congenital hypertonicity - P94.1; congenital hypotonia - P94.2; other disorders of muscle tone - P94.8. Hypertensive-hydrocephalic syndrome is the initial stage of active hydrocephalus, which can be called "syndrome of expansion of cerebral blood vessels" and later (during recovery) is considered hydrocephalus (during the formation of this disease).

Clinical syndromes of recovery period of PCNSL (up to one year).

Clinical syndromes of the recovery period of PPNS include: delayed stages of psychomotor development - R62.0; emotional and behavioral disorders - F98.9; disorder of psychospeech development -F84.8; disorders of motor development (monoplegia or monoparesis of hand - G83.2, monoplegia or monoparesis of leg - G83.1, diplegia - G83.0, flaccid hemisyndrome - G81.0, spastic hemisyndrome - G81.1, unspecified hemisyndrome - G81 .9, paraplegia or free paraparesis - G82.0, paraplegia or spastic paraparesis - G82.1, tetraplegia or free tetraparesis - G82.3, tetraplegia or spastic tetraparesis - G82.4); also symptomatic hydrocephalus - G91.8, convulsive syndrome - R56, non-convulsive paroxysms - G98.0, autonomic dysfunction - G90.8, parasomnias - G47.0. It is important to identify non-convulsive paroxysms (motor, psychomotor, metabolic, etc.), because a convulsive syndrome cannot always be equivalent to epilepsy, just like non-epileptic seizures.

Delay in psychomotor development of PMTCT (motor activity, psyche, speech formation in children) is usually characterized by degrees of severity: mild - characterized by a three-month (or less) delay in development compared to healthy children, not related. can be easily corrected by damage to the central nervous system; moderate - a maximum delay of six months, a consequence of oxygen starvation (ischemia) of the brain. Compensation is possible with early treatment; Severe - a delay of more than six months, the result of malformation of the central nervous system (CNS), genetic pathologies, damage to the fetal brain due to intrauterine infection, decreased thyroid function. Disruptions are permanent and may increase as the child grows.

Classification (syndromological) of the consequences (results) of PPNS in children older than 12 months (VM Studenikin, MD, LG Khachatryan, MD, etc.).

Practically possible outcomes of PPNS are reduced to four options: recovery (up to 30%), organic diseases (about 30%), functional disorders (about 40%) and death (rarely).

1. Organic consequences of PPNS are divided into four main categories: with the predominance of motor disorders; with mental illnesses; symptomatic epilepsy; hydrocephalus.

1.1. Organic consequences dominated by motor disorders include three main groups of diseases:

- infantile cerebral palsy (CP) - G80.0 (spastic diplegia - G80.1, infantile hemiplegia - G80.2, dyskinetic cerebral palsy - G80.3, ataxic cerebral palsy - G80.4, mixed type of cerebral palsy - G80. 8);
- damage to nerve roots and plexuses - G54.8; It is based on damage to nerve roots, plexuses or individual nerves that occur during the intrapartum period. The clinical presentation during the recovery period depends on the location and severity of the injury (ischemia, sprain, tear, rupture). Damage to the brachial plexus often occurs, mainly in full-term infants. Usually, the stretch or ischemia and resulting motor impairment (paresis) recover during the neonatal period. And tears and ruptures of the roots or plexuses lead to permanent motor

deficits (paralysis, plegia) in the limbs, perinatal damage to the central nervous system, peripheral paresis and paralysis (G54.0, G54.1, G56.2,) . G83.0, G83.1, G83.2).

- other diseases of the peripheral nervous system - G64.0. The peripheral nervous system includes nerves and nerve plexuses outside the brain and spinal cord (that is, outside the central nervous system). The main function of the peripheral nerves is to connect the central nervous system with the rest of the body and internal organs. Incoming and outgoing impulses are carried through nerves like telephone wires. Sensory nerves carry information about the environment such as temperature, touch, pain, and body position. Motor nerves send bioelectric impulses to muscles, causing them to contract and move. Unlike the brain, peripheral nerves are not protected by the bones of the spine or skull, and there are no special tissue barriers (eg, the blood-brain barrier) between the blood and the brain. Thus, they are more susceptible to toxins and mechanical damage. According to the International Classification of Diseases, tenth revision (ICD-10), polyneuropathy is coded G60 - G64. This class includes the names of polyneuropathies and other lesions of the peripheral nervous system.

1.2. Organic consequences of PPNS with mental disorders correspond to the diagnosis in practice: unspecified mental retardation (F79), the determination of its degree is within the competence of child psychiatrists and medical psychologists (F79). Oligophrenia (dementia) is an underdevelopment of mental functions due to reasons that existed before the child was born or occurred in the first years of his life. Oligophrenia is characterized by the fact that the formation of most functions of the nervous system occurs with a significant delay, many functions that are important for adaptation are not formed at all. Characteristic dynamics of oligophrenia without the development of intellectual disability. On the contrary, it is necessary to talk about evolutionary dynamics expressed in gradual maturation and accumulation of certain experiences, partial adaptation in specially created conditions. It is customary to divide oligophrenia into stupidity, insanity and weakness according to the degree of severity.

1.3. Symptomatic epilepsy includes three main categories: epilepsy with simple partial seizures - G40.1; epilepsy with complex partial seizures - G40.2; generalized epilepsy - G40.4.

1.4. Hydrocephalus as a result of PPNS is characterized by four concepts: communicative hydrocephalus - G91.0; residual ventriculomegaly with hydrocephalus - G91.8; hydrocephalus, unspecified -G91.9; symptomatic hydrocephalus - G91.8.

2. Functional disorders (results of PPNS) are considered in four major sections:

2.1. Motor disorders (specific motor function disorders - F82.0); Dyspraxia is a specific movement disorder in which there is no paralysis or impaired muscle tone, but the child has difficulty coordinating and performing complex and purposeful movements. The disorder cannot be explained by mental retardation or any specific congenital or acquired neurological disease. Among children aged 5 to 11 years, the prevalence is about 6%. It occurs mostly in boys. Possible etiological factors are divided into two groups: developmental factors and factors of changes in brain structures. The developmental hypothesis suggests that the disorder is caused by a disruption of communication between the cortical and executive motor systems, as well as by different levels of their maturity.

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