CLINICAL AND NEUROLOGICAL MANIFESTATIONS OF ENCEPHALITIS IN CHILDREN

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Abstract*. The article presents the results of clinical and neurological studies of children with primary and secondary encephalitis, characterized by polymorphism of neurological symptoms. Analysis of clinical and neurological parameters of children with primary and secondary encephalitis showed more severe and persistent organic disorders in primary encephalitis.*
Keywords*: encephalitis, children, convulsive syndrome, neurological disorders.*

Relevance: in childhood, the incidence of viral encephalitis in the world ranges from 3.9 to 7.5 per 100,000 children (1,2,3). According to WHO, 75% of cases of viral diseases of the central nervous system (meningitis, encephalitis) occur in children under 14 years of age (Leshchinskaya E.V., Martynenko I.N. 1990). Among children abuse, this pathology is characterized by particular severity, a high frequency of neurological complications of a disabling nature with high mortality (4). The course of encephalitis depends on the necrotic process, resulting in the formation of foci of decay of the brain matter with subsequent cyst formation, especially in the temporal lobes of the brain (1,2,3). WHO data indicate that diseases caused by the herpes simplex virus (HSV) rank second (15.8%) after influenza (35.8%) as a cause of death from viral infections. Relatively often, infectious lesions of the nervous system are the cause of neonatal seizures in 3-17% of cases.

Conversely, neonatal seizures may be the first symptoms of brain inflammation. The authors also pointed out the facts that intracranial vasculitis in children, as a cause of hemorrhagic stroke, is more often caused by bacterial meningitis or viral encephalitis (3). Depending on the nature of the damage to the nervous system, two types of encephalitis are distinguished. The main link in the pathogenesis of primary encephalitis (herpetic, tick-borne, etc) is direct damage by the virus to neurons, endothelium of blood vessels and meninges, and in the case of secondary or allergic (against the background of a rash, viral respiratory infections) encephalitis, the brain is affected indirectly, through autoimmune damage to the myelinated structures of the central nervous system, endothelium, neuroglia, being in most cases a reversible process. [1,2,3,4].

Purpose of the study: to study the clinical and neurological characteristics of encephalitis in children.

Materials and methods of research: to solve the set problems in a hospital setting, we examined 50 children aged 1 to 5 years with primary and secondary encephalitis. The study main group included 26 children with primary encephalitis (15 boys and 11 girls), the second group included 24 children with secondary encephalitis (14 boys and 10 girls). All children underwent a targeted clinical-neurological, laboratory and instrumental examination.

The diagnosis was made based on the results of clinical and neurophysiological (EEG, computer and magnetic resonance imaging of the brain) studies.

Results of the study: in the study group with secondary encephalitis, the provoking concomitant diseases were ARVI, thus having a viral etiology, while the group with primary

encephalitis consisted of children with herpetic encephalitis 16 children (67%) and rubella encephalitis 8 children (33%).

It should be noted that the children were hospitalized in the ICU. In 70% of patients, the first complaints were convulsive syndrome, against the background of general cerebral symptoms.

When collecting anamnesis in a group of children with primary and secondary encephalitis, as a rule, complaints about deterioration of the condition appeared 3-7 days after the onset of the disease. Upon admission to the hospital, the main complaints made by parents of children with secondary encephalitis, in addition to complaints about the underlying disease, were lethargy, lethargy, and drowsiness in 10 children (50%); 8 children (40%) had seizures; 9 children (45%) had general cerebral symptoms in the form of nausea and vomiting, 3 older children (15%) complained of headaches; impaired consciousness was observed in 5 (25%) children (soporous state). All patients were hospitalized in the ICU due to their serious condition.

According to the literature, the most severe viral lesions of the brain include herpetic encephalitis. The formation of necrosis in the frontal and temporo-parietal lobes of the brain contributes to such clinical manifestations of the disease as impaired consciousness, behavioral disorders and partial seizures, as well as its outcomes.

In all children with primary encephalitis for 1-3 days. a coma developed lasting 26±6 days, which was preceded by partial or secondary generalized seizures. It should be noted that 83% (20) of children with primary encephalitis were under 2 years of age.

As we have already noted, 8 children (33%) of the group with primary encephalitis had rubella encephalitis. Symptoms of deterioration in the condition in the form of convulsive syndrome in 7 children (87%) and disturbances of consciousness: in 50% stupor and in 3 children (38%) coma of 1-2 degrees occurred on days 2-7 from the onset of manifestations of rubella, the rash was preceded by hyperthermia.

The premorbid background in children with PE and VE was studied (Table 3.3.). The condition of 24 children with primary encephalitis and 26 children with secondary encephalitis group was aggravated by such concomitant diseases as anemia (33% and 23%), malnutrition (25%), respiratory distress syndrome (75% and 19%), heart failure (50% and 19%). 15%), endotoxemia. The manifestation of one or more foci of infection (bronchopneumonia - 83% and 50%, sepsis -50% and 19%) significantly worsened the somatic status. This was manifested by symptoms of intoxication: an increase in temperature to sub febrile and febrile levels, lack of weight gain, regurgitation, bloating due to intestinal paresis, and enlarged liver. In 6 (25%) children with primary encephalitis of the group, signs of DIC syndrome were observed, which was manifested by increased bleeding from injection sites and pinpoint skin hemorrhages.

Table 1

Premorbid background in children with primary and secondary encephalitis.

Analyzed indicators	Children with PE n=24		Children with VE n=26	
	abs (%)	abs (%)		abs (%)
ARVI	18	75	15	58
Pneumonia and bronchopneumonia	20	83	13	50
Intestinal infections	18	75	16	62

TORCH carriage	22	92	15	58
Anemia	8	33	6	23
Sepsis	12	50	5	19
DIC syndrome	6	25	1	4
Respiratory disorders	18	75	5	19
Heart failure	12	50	4	15

TORCH carriage was determined in 92% of patients with primary encephalitis and 58% with secondary encephalitis.

A comparative analysis of the neurological status of patients with primary and secondary encephalitis was carried out.

In the group with primary encephalitis, a moderate course was observed in 14 children and a severe course in 10 children of this group, impaired consciousness corresponded to a coma of 2-3 degrees, age varied up to 2 years. The first symptoms of the disease were disturbances of consciousness and convulsions, which were more of a partial motor nature in 16 children (67%), while 17% had generalized tonic-clonic convulsions. A wide range of organic disorders was characteristic of children with primary encephalitis. In 16 children (75% of children (18) had bulbar disorders, meningeal symptoms - stiff neck, Kernig's sign, which were leveled. Damage to the cranial nerves was noted in all children, depending on the severity of the disease. Oculomotor nerves - decreased convergence in 16 children (67%), lack of reaction to light in 10 children, convergent strabismus in 13 children (57%), horizontal nystagmus in 16 children (57%), central paresis of the facial nerve was observed in 18 children (75%), deviation of the tongue. in 10 children (42%) symptoms of oral automatism were observed in 8 children (33%).

Increased muscle tone was noted in 12 children, while 10 children had muscle hypotonia with decreased tendon reflexes. The formation of paresis and paralysis was observed in 23 children (96%) with primary encephalitis. 71% of children had pathological reflexes and foot signs. Sensory disorders in the form of hypoesthesia were noted in 10 (67%) children and hyperesthesia in 11 (42%) children,4 children (17%) had emotional-volitional disorders, tearfulness and dysarthria. These children were older, the course of the disease was of moderate severity.

When examining 26 children with secondary encephalitis, impaired consciousness was noted in 6 children (23%), a state of coma of the 1st, 2nd degree, and in 6 (23%) children a state of stupor was noted. In 6 children (23%) meningeal symptoms were observed - stiff neck, but were not pronounced. Damage to the cranial nerves was observed in all children, depending on the severity of the disease. Oculomotor nerves – decreased convergence in 5 children (19%), convergent strabismus in 6 children (23%), divergent strabismus in 1 child (4%), horizontal nystagmus in 10 children (50%). Central paresis of the facial nerve was observed in 7 children (35%), decreased pharyngeal and palatal reflexes in 6 children (30%), deviation of the tongue in 5 children (19%). Over time, convulsions were observed in 14 children (54%) and were the first signs of the disease; at low-grade fever in 6 children (23%), they were clonic-tonic in nature; in 8 children (31%) convulsions were partial, motor attacks.

Thus, the course of primary encephalitis was accompanied by a disturbance of consciousness in the form of a 2-3-degree coma. It should be noted that the fatal outcome in children with primary encephalitis was 10 children, the coma period was on average 26-30 days. The remaining children had organic disorders and symptomatic epilepsy. While in the group with secondary encephalitis a milder course was noted, the period of grade 1-2 coma lasted about 10-

12 days. Persistent organic deficiency was observed in 2 children in the form of paresis and paralysis; the overall outcome was favorable.

Conclusion: analysis of clinical and neurological parameters of children with primary and secondary encephalitis showed more severe and persistent organic disorders in primary encephalitis.

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