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HERPETIC ENCEPHALOMYELITIS. CASE FROM PRACTICE

Acute inflammatory diseases of the central nervous system are a heterogeneous group of diseases and conditions in clinical manifestations and prognosis, united by one of the main pathomorphological signs - primary demyelination. This symptom, in turn, causes other general symptoms, united by the predominant damage to the central nervous system (CNS) in the group under consideration, since the myelin of the central nervous system differs in origin and molecular antigenic composition from the myelin of the peripheral nervous system, and predominant damage to the white matter of the brain and spinal cord, where myelin quantitatively predominates over other components of the nervous tissue.

Another common pathomorphological sign is inflammation, against the background of which (or as a result of which) demyelination develops. Another common feature of diseases in this group today is their idiopathic nature. The latter circumstance most likely means that these diseases are multifactorial and develop with the participation of a number of endogenous, primarily genetic, factors, as well as exogenous etiological factors or triggers, including infections. The listed probable pathogenetic variants of the relationship - demyelination, inflammation and infection - emphasize the insufficient knowledge and heterogeneity of infectious diseases of the central nervous system. This largely determines the relevance of their study, and from a clinical point of view, the significance and difficulties of differential diagnosis.

In the structure of viral encephalitis, lesions of herpetic nature (herpetic encephalitis (HE)) occupy one of the main places [Tansarli GS, Chapin KC. 2019. Diagnostic test accuracy of the BioFire(R) FilmArray(R) meningitis/encephalitis panel: a systematic review and meta-analysis. Clin Microbiol Infect https://doi.org/10.1016/j.cmi.2019.11.016.]. Its most common form is considered to be acute meningoencephalitis with predominant damage to the anterior (temporal, frontal, central) parts of the brain. Focal neurological symptoms of the nervous system manifest themselves in the form of mono- and hemiparesis [8]. Often, epileptic seizures are the first signs of central nervous system damage in HE [16]. One of the widespread herpes viruses is the Varicella-zoster virus. Every year, the incidence of herpes zoster varies depending on the age and immune status of the sick, ranging from 0.4-1.6 per 1000 people under the age of 20 years and from 4.5 to 11 cases under the age of 80 years and older [8] . The clinical picture of GE is characterized by an acute onset with high fever for 3-4 days, followed by the development of neurological symptoms. A frequent sign of GE is impaired consciousness up to the development of coma due to rapidly increasing cerebral edema. In 90% of cases, damage to the temporal regions is noted [3].

The following describes a clinical case of a patient with manifestations of viral encephalitis of mixed origin.

Clinical case. Patient Zhamila Abdullaeva, born in 1972, was admitted to the Department of Neurology 1 of the ASMI clinic with a diagnosis of Acute encephalomyelitis.

Complaints upon admission: severe headaches, nausea, vomiting, increased body temperature up to 39 ° C, chills, pain throughout the body, weakness in the arms and legs on the left, difficulty speaking, choking.

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Anamnesis morbi: From the anamnesis it is known that she suffered from herpes zoster localized in the lumbar region, after which she began to notice a feeling of pain and burning in this area. 5 days later, she noted a short-term numbness in her left leg, then weakness developed in her left arm, but she did not see a doctor or receive treatment. A day later, speech began to become difficult; her relatives turned to a specialist, where she was urgently hospitalized.

The general condition is serious, he is unconscious. Visible skin and mucous membranes are unchanged. Peripheral lymph nodes are not enlarged. Breathing is even, through the nose. Weak vesicular breathing is heard in the lungs. Heartbeats are rhythmic. Blood pressure 90/60 mmHg. pulse 78 beats per minute. The language is clean, uncoated. The liver and spleen are not enlarged. Urination is carried out through a urinary catheter.

When examining the neurological status, the following were revealed: (given the serious condition of the patient) uniformly narrow pupils, photoreaction and weak corneal and conjunctival reflexes. The face is symmetrical. There is no deviation. Dysphagia is observed (when trying to swallow water). Motor sphere: tetraplegia. Muscle strength in the arms and legs is 3 points, muscle tone is increased in the extremities. Tendon reflexes are increased. Pathological reflexes: upper, lower Rossolimo, Babinsky on the left are positive. Sensitivity: deep and superficial sensitivity, tetrahypoesthesia. Coordinator samples could not be verified. Meningeal signs: undecided. VND consciousness is soporous. Glasgow scale: 10 points.

Analysis results:

1. General analysis: Hb - 88.4 g/l, erythrocytes - 3.56, Color - 0.8, leukocytes - 62 g/l, ESR - 30 mm/h.

2. Indicators of a comprehensive clinical blood test: blood glucose level 5.04 mmol/l (N=3.89-5.83 mmol/l) – within normal limits. Biochemical blood test: LDL, cholesterol, triglycerides – within normal limits; increased indicators: total cholesterol. – up to 5.8 (N=0-5.2 mmol/l), HDL cholesterol – up to 2.24 (N=1.0-2.1 mmol/l). fibrinogen, prothrombin, INR, thrombin time are normal.

3. Analysis of cerebrospinal fluid: quantity - 2.0, color - colorless, protein - 1.32, cytosis - 18, Pandi reaction - +++, lymphocytic pleocytosis.

4. ECG: sinus tachycardia, horizontal position of the electrical axis of the heart.

5. C reactive protein – 48

6. Blood immunology: IL-1(β)-32.52, IL 6-11.8, TNF(α)-3.52, large CEC-68, small CEC-120.

7. To clarify the nature of the disorders and exclude the demyelinating process, consultation with an ophthalmologist is recommended. The ophthalmologist's conclusion: the optic discs are pink, the boundaries are clear. retinal vascular angiopathy.

8. Results of paraclinical methods of studying MRI of the brain: in the white matter of both hemispheres, mainly subcortically, multiple foci of increased MR signal with clear contours, 2-8 mm in size, without perifocal edema are determined; a single focus of 3 mm is in the radiance of the corpus callosum on the left.

Conclusion: The MRI picture of multifocal white matter lesions, most likely of vascular origin, should be differentiated from demyelinating disease MRI of the spinal cord infiltration of the spinal cord at the level of the cervical spine.

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Based on the anamnesis, clinical, MRI and laboratory data, a diagnosis was made: Acute encephalomyelitis, viral etiology.

Complication: mixed tetraparesis. Bulbar palsy.

Based on the diagnosis, the following treatment was carried out:

antiviral drugs (Zovirax); decongestants (sorbilact, mannitol); drugs that improve microcirculation; antioxidants (tivortin, neurox); neuroprotectors (cerebrolysin); hormone therapy (prednisolone); antibiotic therapy (ceftriaxone); anticholinesterase drugs (neuromidin); vitamin therapy (dematon).

Study of the immunological status of PCR (polymerase chain reaction) and other infections: DNA of CMV, type I herpes virus was detected. Thus, an increase in antibody titers to CMV confirms its participation in the etiology of the disease. Antibodies to HSV-1, which were determined to indicate connection with HSV-1 as the causative agent of the disease.

Repeated MRI of the brain showed multifocal areas of demyelination of the white matter, without negative dynamics in comparison with the MRI that was performed previously. On examination: there is no negative dynamics in the neurological status. Works successfully in kindergarten. Final diagnosis: viral encephalitis associated with cytomegalovirus and herpes simplex virus type 1. The patient was observed by a local doctor and was seen by a neurologist for a year. She complained of rashes on the body, in the oral cavity, increased body temperature, and pain. Antiviral drugs were prescribed, which improved the condition.

In conclusion: we present a rare case of a patient with a slow course of viral encephalitis caused by cytomegalovirus and herpes simplex virus. Diagnosis of encephalitis in the described patient was based on the determination of specific antibodies to herpes virus type 1 and cytomegalovirus in the blood. The difficulty in interpreting the manifestations of the disease is due, first of all, to the fact that in this patient most of the symptoms are “erased” in nature due to the duration of the disease, and there is also no reliable data on the onset of the disease and the first manifestations of the disease. Negative results were obtained during PCR. Although, currently, most neuroinfections are diagnosed using the PCR method, which has great advantages, it is not without its disadvantages, since it can be seronegative in the first 3-4 days of illness and its sensitivity decreases significantly after the 10-20th day of illness. But there were exceptions after encephalitis in the convalescent stage of viral complications [2], such as rashes on the face, herpetic sore throat. Despite the bilateral brain damage, this patient was of mild severity, and its positive outcome was due to constant treatment with antiviral drugs (Zovirax and Ergoferon).

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