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MODERN ETIOLOGY, DIAGNOSIS AND TREATMENT OF VIRAL ENCEPHALITIS

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The summary: Viral encephalitis diffuse or focal inflamattory changes in brain structures caused by the penetration of viral agents into them. The clinical picture is variable, depends on the type of virus and the state of the immune system of the diseased, is made up of general infectious, cerebral and focal manifestations. The diagnostic algorithm includes EEG, Echo-EG, CT, MRI of the brain, lumbar puncture and liquor analysis, polymerase chain reaction to identify the pathogen. Combined treatment: antiviral, antiedematous, anticonvulsant, antihypoxant, neuroprotective, psychotropic.

Key words: encephalitis, meningoencephalitis, virus, epilepsy, epilepsy syndrome.

Viral encephalitis is a viral etiological inflammation of the brain tissue, accounting for about 20% of all viral neuroinfections. The global incidence rate of acute viral encephalitis ranges from 4 to 7.5 per 1000,000 axoli. According to the World Health Organization, 75% of all viral lesions of the central nervous system are observed in children under 14 years of age [12,48]. Another reason for the relevance of this topic is the severe course of the disease, especially in children, a high incidence of neurogenic complications and a high mortality rate (10- 20%) [17,18].

The diagnosis of the erta encephalitis virus requires a thorough examination and medical examination of patients [10, 20]. Currently, the clinic is diagnosing encephalitis encephalopathy virus, as well as diagnosing a number of diseases of the central nervous system. The yarim hollard talvasalar encephalitis virus has been detected. This is due to the fact that the viral infection common in Uzbekistan has a specific epidemiological history. General infectious signs may even be accidental, and, as a rule, belgilarning may have varying degrees of reliability [11,16]. As a doctor, immunologist, virologist, neurologist and many other specialists, you can undergo an examination.

Encephalitis virus is an antigen of the encephalitis virus affecting the nervous system, the main component of the immunological tolerant drug myeling [1,22]. Birak, ushbu factni clinic zhikhatdan baholanmagan. The viral load on the main protein, myeling, affects only the herpes group, dogwood, and adenovirus groups, although there is a theory [8,9].

The main place in the pathogenesis of viral encephalitis is occupied by allergic inflammation [2,3]. These viruses dramatically weaken the immune system, causing immune imbalance [26,27,46]. However, it can be assumed that Ig E is involved in the development of immunopathological reactions in the central nervous system in acute viral encephalitis. This can lead to the development of the autoimmune demielinization process in some cases [13,36,44,48]. The prevalence of viral encephalitis in various etiologies is climatic and geographically diverse [25,30]. So, Japanese fly encephalitis is very common in the states of Japan and Asia, St. Louis encephalitis is most common in the United States, lethargic Economo encephalitis is most common in western Europe, and duct encephalitis is most

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common in the forested provinces of Eastern Europe. The risk of developing deep complications in viral encephalitis, taking into account the difficulty of diagnosis and problems in the identification of the pathogen, remains an urgent problem of modern applied neurology [19, 23,24].

Causes of viral encephalitis

Common herpes virus is common among neurotrophic viruses that cause encephalitis. The cause of damage to the cerebral substance can also be pathogens of other herpes viruses: cytomegalovirus, chickenpox virus, infectious mononucleosis (Epstein –Barr virus). Etiological factors of viral encephalitis include enterovirus (including poliomyelitis virus), adenoviruses, epidemic mumps virus, influenza A, measles, Thrush, arboviruses, reoviruses, Arena - and Bunyaviruses are also involved [6,9,14].

Infection from the patient is transmitted directly through air droplets, contact, oral-fecal passages or transmissively – in the carrier sting (mosquito, mite). In the latter case, the infection reservoir can be birds and animals. The incidence of a neurotrophic virus in the body may be a consequence of obtaining a live attenuated vaccine (e.g. antirabic, anti-polio, anti-smallpox) [15,21,28].

In the human body, the inflammatory process developed by the entry of the virus, the state of its immune system, reactivity are important [29,31,32]. Also, the age of the patient (in children, the elderly), the presence of a state of immune deficiency disease or immunosuppression, an increase in the likelihood of developing encephalitis, and one of the factors contributing to its severe course, is noted. Hence, the human immunodeficiency virus (HIV) cannot usually be a direct causative agent of the disease, but causes a state of immunodeficiency and increases the likelihood of the appearance of viral encephalitis [4,5].

Symptoms of viral encephalitis

The clinical picture will depend on the type of encephalitis and the nature of its rejection [33,38,40]. The disease begins in the form of general signs of infection: fever, impotence, myalgia, pain/itching in the throat or a decrease in constipation, and the observation of unpleasant sensations in the abdomen. Against the background of them later, common brain symptoms appear: cephalgia (headache), nausea unrelated to nutrition, vomiting, increased sensitivity to light, dizziness, epileptic paroxysms, etc. Cephalgia is usually observed in the forehead and eye area. Psychosensory disorders, meningeal syndrome, benign disorder States (seizures, sopor, coma States), psychomotor arousal, delirium, amentium can be observed [7, 34, 37].

In parallel with the growth of the above-mentioned signs, an acute neurological deficit occurs. Symptoms of spastic paresis, ataxia, aphasia, cranial nerve damage (decreased hearing, impaired visual field and acuity, changes in eyeball motion, bulbar paralysis), cerebellum syndrome (discoordination, wobbling, large stride, muscle hypotonia, intentionalist tremor, dysarthria) may be observed along with hypesthesia [39,41].

The above-mentioned cases can be observed in any encephalitis. However, in some viral encephalitis, the observation of a characteristic combination of specific clinical signs or

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signs makes it possible to distinguish them from many other diseases. Hypersomnia for lethargic encephalitis, severe impairment of consciousness for Japanese encephalitis, for measles encephalitis –gallucination and psychomotor arousal, cerebral ataxia for smallpox encephalitis, major disorders of ES retardation and meningeal signs for St. Louis encephalitis are characteristic[42, 43].

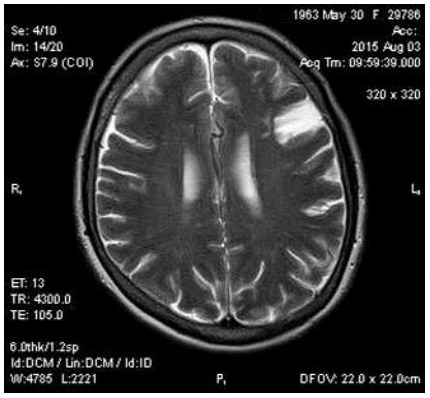
Typical of the course of viral encephalitis, it can be asymptomatic, abortive or lightning fast. The asymptomatic form of the disease is accompanied by periodic cephalgia, irregular Genesis fever, transistor dizziness, and/or episodic diplopia. There are no neurological changes in the abortive type, signs of gastroenteritis or respiratory infection can be observed. The course of the disease at lightning speed is characterized by a rapidly developing coma and death condition [45].

Viral encephalitis diagnosis

The absence of specific clinical signs and other lesions of the central nervous system (acute encephalopathy, acute diffuse encephalomyelitis, bacterial encephalitis, etc.) similarities cause a number of problems with viral encephalitis diagnosis. Therefore, the doctor should rely on the anamnestic and epidemiological data of the neurologist patient, clinical features and the results of additional examination.

Exo-encephalography usually determines hypertension of the liquor, diffuse changes against the background of the dominance of EEG – slow wave activity, epileptic activity in some cases. Ophthalmoscopy detects changes in the optic nerve disc. In the process of conducting a lumbar puncture, an increase in the pressure of the tserebrospinal fluid is observed, the color does not change. A distinctive feature of viral Genesis encephalitis is the detection of lymphocytic pleocytosis in the cerebrospinal fluid [47]. However, in the early days of the disease, such changes may not be observed, so it is necessary to re-take the liquor for analysis after one day [1,2].

Confirmation of the presence of encephalitis, cranial computed tomography (CT) and magnetic resonance imaging (MRI) in the detection of foci of inflammation are of great importance (Figure 1). Cranial CTSI provides the ability to visualize damage sacs with low density of cerebral tissue, HYPERINTENSIVES in MRI T1 order – hypointensive, MRI T2 order. Figure 2. Encephalitic damage to the bark of the chunk.



Since MRI has high sensitivity, it is considered extremely important in the early stages of the disease. In the early stages of the disease, specific changes appear in the brain of the head. These include encephalitic damage to the

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medial soxes of the pectoral fin (tonsils, hippocampus), mediobasal soxes of the forelock, periventricular edema, enlargement of the cerebral ventricle, etc. (Figure 2) [38,39].

Blood analysis is an additional method for detecting encephalitis. In most cases, in inflammatory processes in the human body, the immune system synthesizes special antibodies that can be detected in venous blood. In Bloodline mite encephalitis, G and M provide an opportunity for early serological diagnosis by detecting immunoglobulins, and in autoimmune types of encephalitis, antibodies to cranial tissue [6,9,32].

Until the polymerase chain reaction (PZR) method is created in the spinal fluid, brain biopsy has been considered the gold standard of diagnosis in the United States, the United Kingdom, Canada and other countries for decades (1970-1990) and has been used quite widely. The pzr method is considered a very convenient, common, optimal and Fast Result test, and in practice it is used to identify the DNA or RNA s of the virus in the cerebrospinal fluid for early identification of the pathogen [18,20].

In parallel with PZR, it is advisable to carry out a serological examination in biological fluids (blood and spinal fluid), since viral DNA is detected in the initial periods of the disease (on days 10-14), while the patient can also refer to the doctor at later times. In this case, PZR can be pseudonegative or pseudopositive, while detection of parallel-state antitana increases the outcome of the investigation [28]. **Treatment of viral encephalitis**

Complex treatment involves etiotropic, pathogenetic, symptomatic and rehabilitative treatment. In the treatment of etiotropic, pharmaceutical drugs against viruses are recommended: in herpetic encephalitis – acyclovir and gantsiclovir, arboviruslida –ribavirin. In parallel, interferon and its analogues are also used in the treatment. Spesefic immunoglobulin may also be used.

Pathogenetic treatment is carried out in the recovery of vital activity (cardiac glycosides, vasoactive drugs, oxygenotherapy, as prescribed by IVL), by the use of anti-tumor drugs (mannitola, furosemide, acetazolamide), antihypoxants (ethylmethylhydroxypyridine succinate, meldonium), neuroprotectors (oxymethylethylpyridine succinate, pyracetam, glycine, scopolamine). In most cases, there is a need to recommend glucocorticoids with anti-inflammatory and anti-tumor effects. Anticonvulsants (carbamazepine, valproates, diazepam), anti-vomiting medication, depending on the need for symptomatic treatment.

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