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# ENCEPHALITIS OR STROKE? MISTAKES IN THE DIAGNOSIS OF VIRAL ENCEPHALITIS

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Currently, there are about 20 known types of viral encephalitis that cause acute and or chronic processes in the brain. According to the opinions of most scientists, inflammatory agents can cause different degrees of damage, different localization, which may possibly cause incorrect timely differential diagnosis of the disease. Often, an erroneous diagnosis can be made by general practitioners, emergency physicians, which is important for hospitalization of a patient in a hospital. It is important to conduct a thorough clinical, neurological examination and fully examine the medical history. This will help the doctor to orient himself to the further management of the patient.

Encephalitis is an acute or chronic inflammatory disease of the brain characterized by the presence of organic lesions in the patient. It is known that the brain is protected from the ingress of immune system cells from the periphery by the blood-brain barrier (BBB), but at the same time has its own protective potential. The penetration of the pathogen or its antigens into the central nervous system causes an innate immune response of perivascular macrophages, microglia and astrocytes. Antigen-specific T and B lymphocytes are formed on the periphery during the systemic adaptive immune response and are recruited to the central nervous system through the stimulated epithelium of the BBB [1]. But even in the early stages of inflammatory diseases, the difficulty of determining the type of T-lymphocytes puts neurologists and general practitioners in a difficult state. Although repeated tests of cerebrospinal fluid give a leukocyte shift, but you can miss the timely start of treatment of inflammation, mistakenly diagnosing a brain stroke.

Acute cerebral vascular diseases are known for their high mortality and disability among the population. The inflammatory reaction resulting from anaerobic glycolysis leads to the death of neurons in brain tissue. Interleukins are secreted by macrophages and cause fever, activate T and B lymphocytes, and neutrophils underlying inflammation. An increase in the number of leukocytes provokes damage to the vascular wall and the nervous system due to the release of reactive oxygen species, which are not utilized due to cerebral ischemia and lead to apoptosis of neurons. Russian scientists have also studied the pathogenetic and prognostic aspects of the involvement of interleukins in the development of inflammatory processes in hemorrhagic stroke [2]. In their opinion, the increased content of interleukins on the third to tenth days of hemorrhagic stroke is determined by its participation in the reactions of local and systemic inflammation, which is accompanied by brain damage.

Cytokines also activate adhesion mechanisms on the endothelium of the cerebral capillaries, which leads to a breakthrough of the blood-brain barrier (BBB) with the development of an inflammatory reaction in brain tissue and subsequent death of neurons. Astrocytes are also important in the violation of the structure of endothelial cells of the microvessels of the brain, the proliferative reaction of which is normally aimed at strengthening the structures of the BBB, inactivation and elimination of the pathogen, and replacement of lesions. It is the reaction of astrocytes at all stages of the infectious process that is one of the decisive factors for a favorable outcome in viral encephalitis. In contrast to systemic immune reactions with a local response, humoral and cellular reactions in the central nervous system are associated with the activation of micro- and astroglia, the production of neuropeptides, neurohormones, neurotrophic factors, and nitric oxide. An essential component of the pathogenesis of encephalitis is edema and swelling of the brain.

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Tumor necrosis factor alpha (TNF-a) plays an important role in the formation of an antiviral immune response. It is a multifunctional cytokine with pronounced pleiotropy, which participates in the formation of protective reactions of the body, stimulates the phagocytic and cytotoxic activity of cells, regulates the processes of immune inflammation, promotes the utilization of destructive material, and regulates cell apoptosis [3]. TNF hyperproduction is one of the main mechanisms of activation of the infectious process during its transition from a latent state to a phase of clinical manifestations and indicates the progression of the disease. Elevated TNF-a levels in blood plasma were found in exacerbation of chronic infection induced by hepatitis B and C viruses, HIV, herpes simplex, Epstein-Barr, cytomegaly, influenza, polio, tick-borne encephalitis, etc. [4]. The inhibition of TNF-a production at the stage of chronization of the infectious process is the result of deep dysregulation of the immune response.

Radiation diagnostics is extremely important in the management of patients with acute encephalitis, strokes and other diseases. These imaging techniques help to determine the level of lesion, the involvement of certain structures of the central nervous system in the pathological process, contribute to the correct verification of the diagnosis (and are of particular value in early diagnosis), allow you to detect complications of the underlying process (for example, mass effect), assume the etiology of the disease, track the response to therapy and conduct dynamic monitoring in case of transition of the process to a chronic form. It cannot be said that encephalitis caused by a certain pathogen has a specific clinical and radiation picture, however, there are characteristic signs, knowledge of which will help to quickly direct the diagnostic search in the right direction.

In case of encephalitis of the brain, an MRI scan is recorded:

• Signs of cerebral edema – smoothness of the gyrus, reduction of differences between the cortex and the white matter of the brain (mass effect).

• In the subacute period, hemorrhage foci in the form of signal amplification on T-2 dependent MRI images and loss of the MR signal in GRE/SWI sequences.

• In the later stages, foci of necrosis appear in the form of hypointensive areas without clear boundaries.

• In chronic course, scattered nodules from microglia are detected.

• Signs of transferred inflammation of the brain include calcifications, areas of softening of the brain along the course of the gyrus or diffusely located. They may look like cysts. In this case, the ventricle of the brain on the affected side may increase compensatorily.

To clarify the data, an MRI with contrast is performed. The contrast agent accumulates along the convolutions, along the course of the subarachnoid space, diffusely or annularly. [5]. Although modern methods of neuroimaging inflammatory brain lesions and cerebral strokes also give an erroneous diagnosis. For example, in the early stages of inflammation and strokes, especially ischemic ones, do not provide clear information about the state of brain tissue.

**A case from practice.** We hospitalized a 73-year-old patient with a diagnosis of acute ischemic stroke, with left-sided hemiparesis. The consciousness is soporose. From anamnesis (according to relatives), he had the flu 20 days ago, after the condition returned to normal, on the 7th day the patient lost consciousness, there were convulsive phenomena, a slight increase in blood pressure. The patient was hospitalized and diagnosed with acute ischemic cerebral circulation disorder. He received therapy at his place of residence, but since the patient's condition remained severe, he was transferred to the regional department.

An MRI of the brain without contrast on day 6-7 gave a picture of an ischemic stroke. Despite the treatment, the patient's condition worsened. On the 10th day, the patient had vesicular rashes in the oral cavity, which made it possible to suspect encephalitis. On the same day, a control spinal puncture was performed, an analysis of the cerebrospinal fluid for the determination of

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proinflammatory cytokines gave high indicators. Next, the patient was prescribed antiviral and antibacterial therapy, drugs aimed at the inflammatory process, with correction of the immune system, rehabilitation of foci of infection. An MRI image with contrast revealed foci of destruction of brain tissue and hemorrhage in the parenchyma of the brain. On the 4th-5th day after the start of etiotropic treatment, the patient's condition began to improve. His mind was clear, he answered questions clearly, although he remained detached. The rashes of the oral cavity began to dry out gradually. Anti-inflammatory cytokines were found in the blood above the reference values (IL-4, IL-10), and the number of pro-inflammatory cytokines decreased sharply (IL-1, IL-6, TNF). On the 20th day, the patient was discharged and sent for rehabilitation treatment at his place of residence. Upon discharge from the hospital, mild hemiparesis persisted.

We also examined sera for the determination of proinflammatory cytokines IL-1, IL-6, TNF-α in 30 patients with encephalitis of various etiologies (with CMV, HSV-1), as well as 38 with ischemic and 32 with hemorrhagic stroke.

Immunological examination parameters (pg/ml)

| № | Nosological unit | Number of patients | | IL-1β | IL-6 | TNF-α |
|---|---|---|---|---|---|---|
| | | Abs number | % | | | |
| 1 | Encephalitis | 30 | 100 | 19,2±0,7 | 16,2±0,5 | 18,3±0,5 |
| 2 | Ischemic stroke | 38 | 100 | 18,1±09 | 17,4±1,2 | 17,8±0,8 |
| 3 | Hemorrhagic stroke | 32 | 100 | 20,2±06 | 21,6±0,3 | 19,2±0,2 |

As can be seen from the table above, in all conditions, the cytokine level was high and significantly exceeded the level of reference values. Therefore, an increase in cytokine levels determines the degree of inflammation of brain tissue, and not the disease itself. The relationship between infection by inflammation and stroke is twofold, bacteremia or viremia, the occurrence of endotheliitis or arteritis, hypercoagulation and thrombosis and vice versa can lead to a stroke.

Reducing the activation of high levels of proinflammatory cytokines plays a crucial role in the pathogenetic therapy of strokes. Based on this, the mandatory inclusion in the treatment of drugs that affect neuroprotection and the immune response of the body.

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