NEUROIMMUNOLOGICAL ASPECTS OF SUPPRESSION OF ENCEPHALOMYELITIS BY CELLS OF THE ALLOGENEIC DEVELOPING BRAIN

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Introduction.

Among all viral neuroinfections, the share of acute viral encephalitis is about 20%. The incidence of acute viral encephalitis in the world ranges from 4 to 7.5 per 100,000. According to the WHO, 75% of all cases of viral lesions of the central nervous system occur in children under 14 years of age (Leshchinskaya E.V., Martynenko I.N., 1990).

The relevance of studying the problem of viral encephalitis is due to the particular severity of this pathology, a high mortality rate (1020%) and a high frequency of neurological complications with a clearly unfavorable prognosis (Pokrovsky V.I., Lobzin Yu.V. 2007, Sorokina M.N., Skripchenko N.V. 2004, Cunha B. 2004).

Acute viral encephalitis refers to diseases requiring urgent medical interventions and, accordingly, early diagnosis (Timchenko V.N. 2002, Ivanova V.V. et al. 2005, Kim Y. 2004).

The clinical picture of various etiological forms of acute viral encephalitis at the initial stage is largely similar to each other and to a number of diseases of the central nervous system. In about half of cases, acute viral encephalitis manifests itself with seizures. It is extremely difficult to differentiate the etiology with such an onset of the disease, since it is extremely difficult to assume the etiology of the process with an acute onset of neurological symptoms is possible only if there is a certain epidemiological history, information about an acute viral infection transferred the day before. The presence of these symptoms does not necessarily mean an infectious genesis and may be a coincidence, just as the absence of general infectious signs does not exclude the absence of inflammatory changes in the brain. All this determines the need to use a whole range of methods: clinical, immunological, virological, special methods of examining the neurological status and a number of others for the correct diagnosis.

Encephalomyelitis as a model of multiple sclerosis, has been successfully used in preclinical testing of new agents for the treatment of human demyelinating diseases. Suppression encephalomyelitis of can be achieved by intraperitoneal injection of enriched in the bodies of nerve cells suppression of the developing allogeneic brain (Markova. Belskaya, 2000). The cells of the developing brain combine tissue antigenicity with other unique properties of cells of fetuses and embryos, on the basis of which drugs of a new class are created.

The purpose and objectives of the study.

The aim of the investigation was to study the immunological mechanisms of encephalomyelitis suppression upon administration of allogeneic brain cells of newborn animals.

Materials and methods.

Encephalomyelitis was induced by the introduction of brain tissue in complete Freund's adjuvant (Davydova 1969). Enrichment of neonatal brain cell fractions was carried out by preplating (Frischbach 1972). Mononuclear cells of the spleen and leukocytes infiltrating the central nervous system (Sedgwick 1991) were the objects of the study. The

functional activity of natural killer cells and spleen macrophages was assessed (Melnikov, Zayats 1999), the viability of leukocytes infiltrating the CNS when stained with ethidium bromide. The EAE suppressing effect of brain cells was compared with the effect of allogeneic liver cells.

Main results.

It has been established that suppression of encephalomyelitis upon intraperitoneal injection of neurons from the allogeneic developing brain is accompanied by activation changes in the immune system of animals (increased cytotoxicity of natural killer cells and spleen macrophages). The leukocytes infiltrating the central nervous system lose their viability (the specific gravity of cells stained with bromide of the epidium is 42.3 + 2.7 after the use of neurons, 25.2 + 2.3 in the comparison group). Intraperitoneal injection of glial cells did not sutress encephalomyelitis and was not accompanied by activation changes in the immune system. The introduction of liver cells only facilitated, but did not suppress the development of encephalomyelitis, was not accompanied by a change in the viability of leukocytes infiltrating the CNS.

Conclusion.

The results obtained allow us to consider the suppression of encephalomyelitis as a consequence of the antigen-induced deletion of encephalitogenic lymphocytes in the target organ in response to the introduction of cells of the nervous system. Deletion leads to a change in the balance of Th1 / Th2 cells, a change in the balance of pro- and anti-inflammatory cytokines. The absence of a suppressive encephalomyelitis effect upon the introduction of glial cells of the developing brain is apparently associated with the previously described differences in the immunosimilar properties of neurons and glial cells of the central nervous system (Lisyaniy, Markova 1999; Markova 1995).

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