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THE PATHOGENIC ROLE OF VIRUS-SPECIFIC ANTIBODY SECRETING CELLS IN THE CNS OF RATS RESISTANT AND SUSCEPTIBLE TO CORONAVIRUS-INDUCED ENCEPHALITIS

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Amongst the different animal models developed to study primary demyelination of the CNS, the MHV4 infection in rats is of particular interest since immune reactions are involved in the pathology. The most interesting courses of infection are observed in susceptible Lewis-(LE) rats which develop neurological disorders and in resistant Brown Norway-(BN) rats which remain clinically healthy. In order to study the role of the local humoral immune-reaction on the course of the infection we analyzed the kinetics of virus neutralizing antibody titers in the CSF and quantitated virus-specific antibody secreting cell (AbSC) from the brain parenchyma using the spot-ELISA-assay. The subclinical inflammation in the CNS of BN rats was accompanied by an early rise of neutralizing antibodies in the CSF. At the same time, LE rats developed signs of neurological symptoms in the absence of such antibodies. However, with the rise of neutralizing antibodies in the CSF of LE rats, these animals recovered from disease. The CNS-parenchyma of both rat strains harboured equivalent numbers of IgM secreting cells but in BN rats, virus-specific IgGSC appeared earlier and reached higher numbers compared to LE rats. Moreover, BN rats developed antibodies to viral antigens of higher affinity than was found in the LE rats. The data suggest that an early virus-specific antibody response in the CNS limits the spread of the virus and thus contributes to the subclinical course of infection in the BN rat. In contrast, the absence of such antibodies favours the spread of virus to large areas resulting in severe neurological disease as observed in LE rats.

INTRAVENTRICULAR RECOMBINANT ALPHA-INTERFERON IN SUBACUTE SCLEROSING PANENCEPHALITIS

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Since July 1986, we began to treat with intraventricular (IV) recombinant alpha-2a-interferon (IFN) patients affected with subacute sclerosing panencephalitis (SSPE). We treated only subject still at an early phase of the disease (stages 1 or 2), and subcutaneous (SC) IFN administration was always added to IV. Maintenance weekly doses, reached after gradual increment, were (in million units=mU): 1,5mU twice plus 9mU SC in patients 1 and 2; 1mU twice IV plus 4mU SC in patient 3; 3mU IV plus 12mU in patient 4 (SC dose was always subdivided in four administrations). The course of the disease was monitored by means of periodic controls of clinical and laboratory parameters, the most relevant of the latter being: a) antimeasles antibody titers and b) number of oligoclonal bands at isoelectrofocusing in CSF and serum; c) dot-blot hybridization of measles virus RNA on PB lymphocytes. IFN levels in ventricular CSF were also monitored. In all cases an arrest of the progression of the disease with improving of biological parameters was observed for several months. However, patients 1 after 17 months in stage 2, and patient 2 after 4 mo. in stage 2, had septic meningitis and rapidly deteriorated and deceased after withdrawal of the IV catheter and IV IFN administration. Patient 3 (first signs of SSPE in July 1988; IV IFN started after one mo.) and patient 4 (first signs in Febr.90, IV IFN started in Oct.90) are still living in clinical conditions of stage 2.