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Oral Presentation

Inflammation in Brain and Spinal Cord

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Abstract

our goal in this paper is to describe and compare basic immunopathologic pattern of common demyelinating disorder, that is very important to choose the best treatment. The most common disorders are multiple sclerosis, neuromyelitis optica,Anti MOG associated disease,ADEM and autoimmune encephalitis. ADEM consists of ‘‘sleeves’’ of demyelination centered on small, engorged venules.Significant inflammatory infiltrates consist of myelin-laden macrophages.Variable T and B lymphocytes,and occasional plasma cells and granulocytes May coexit ,Perivenous demyelinating lesions may coalescence to form larger areas of demyelination,but the MS-characteristic joint areas of demyelination, macrophage infiltration, and reactive astrocytes are not typically seen in ADEM.Multifocal cortical-microglial aggregates scattered throughout the cortex, not associated with cortical demyelination. NMOSD is an autoimmune astrocytopathy that causes secondary demyelination..The presence of vasculocentric pattern of immunoglobulin. and complement activation in active NMO lesions suggested a humoral-mediated injury. The water channel aquaporin-4 (AQP4), expressed on pericapillary foot process of astrocytes, has been identified as the target antigen in NMO. T-helper 17 (Th17) cells is very important and elevated serum and CSF levels of interleukin 6 (IL-6), with elevated IL-17 in CSF were seen. In biopsy, active demyelination with macrophages containing myelin oligodendrocyte glycoprotein (MOG), immunoreactive myelin debris, were seen adjacent to periplaque white matter. AQP4 is lost in the active lesion but retained in the periplaque white matter, unlike MS, that show increased AQP4 expression on astrocytes.We can see loss of immunoreactivities to astrocytic proteins, glial fibrillary acidic protein (GFAP) matter. ANTI MOG-IgG demyelination with relative axonal preservation and the presence of complement activation markers, which is similar to some histopathological descriptions of MS pattern II demyelination. Distinct from AQP4-IgG-associated NMOSD, no astrocytopathy is seen in these patients. Multiple sclerosis is an inflammatory, demyelinating, and is not only an autoimmune disease in which autoreactive immune cells against myelin, damage axons and nerves in the CNS , but also a neurodegenerative disease of the CNS. CD4+ helper T (TH) cells types 1 and 17 release cytokines and Inflammatory mediators that cause tissue damage, while CD4+ TH2 cells might be involved in modulation of these effects.Macrophages that containing proteolipid protein-immunoreactive myelin debris, were seen adjacent to periplaque white matter .Confluent demyelNination admixed with reactive astrocytes (Creutzfeldt-Peters cells)are considerable.Evidence suggests significant humoral immunity involvement in the disease process(Bcell) .Lymphocytic inflammatory infiltrates are composed mainly of CD8-positive cytotoxic T lymphocytes, and fewer CD4-positive helper T cells.No MS spesicific autoantigen has been identified. Autoimmune Encephalitis envolvement of cortical and white matter disease cause by antibodies against intra and extracellular antigen.In biopsy clusters of mononuclear inflammatory cells mainly composed of CD3+ , CD4+ , and CD8+ T cells.B cells are scarce and mainly restricted to the perivascular space and meninges .Syndromes are associated with antibodies against intracellular (onconeuronal) antigens, appear to be mediated by cytotoxic T-cell responses,against extra cellular antigen related disease that is prominently Bcell type. we can treat MS and ADEM patients with both Bcell and Tcell type associated treatment against Autoimmune encephalitis and NMOSD and Anti MOG associated disease that must be treated with Bcell associated ones.

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