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Neurobiology of Multiple Sclerosis

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About the Study

Multiple sclerosis (MS) is an immune-mediated neurodegenerative disorder which affects the human central nervous system (CNS). It is the most common neurological cause of disability in young adults. Generally, MS is not lethal, but imposes devastating neurological and psychiatric limits on patients. It is important to realize that MS is not a single disease but a conglomerate of different neurological syndromes with different pathological bases and hence dissimilar responses to therapeutic intervention. Relapsing-remitting MS (RRMS), the most common form of MS, affects females, twice as often as males. Many patients with RRMS with or without treatment will later develop secondary progressive MS (SPMS). SPMS is characterized as a neurodegenerative syndrome with less clinical relapses, less response to immunomodulatory treatments and persistent progression of the disease with accumulation of disability.

Primary progressive MS (PPMS) is manifested by an insidious onset and steady disease progression. Patients with PPMS show the least responsiveness to therapy. Neuropathological studies have shown that each or these different forms of MS show different patterns of morphological and histological alterations in the CNS. For example, RRMS is pathologically characterized by the predominant presence of inflammatory cells, while the neuropathology of PPMS mainly consists of less severe inflammation. More prolonged T-cell infiltration, more pronounced oligodendrocyte loss, and an ongoing low level of axonal damage in PPMS. The factors responsible for this diverse underlying neuropathology remain largely unknown but most likely reflect compound inter actions between individual genetics and environmental antigenic "triggers," all of which provoke immune responses and/or increased levels of susceptibility to the inflammatory cascade. In addition to these initiating factors, chronic inflammation also alters and impairs mechanisms related to repair and restitution in the CNS.

On the other hand, the majority or neuroscientists believe that MS is triggered only after an individual's exposure to certain environmental factors like viral agents. This initial viral exposure/infection activates CD4+ T cells against CNS tissue antigens. These imprinted cells eventually gain access to the CNS microenvironment, which inturn perpetuates ongoing cycles of neuroinflammation and neurodegeneration. Most neuroimmunologists favor the peripheral onset model of MS based on competition of cellular infiltrates or the CSF and MS lesions, and data obtained from experimental allergic (autoimmune) encephalomyelitis (EAE). In EAE and perhaps in MS, CD4+ T cells somehow become sensitized against myelin basic proteins (MBPs), and eventually cross the endothelial barrier of the blood brain barrier (BBB). The concept of MS pathogenesis further supported by the fact that certain class II HLA molecules act as the antigen-presenting molecules to the activated pathogenic CD4+ T cells.

The dramatic increase in research into MS pathogenesis during the last two decades has expanded our knowledge of the involvement of other cellular/molecular elements in pathogenesis of this elusive and complicated neurological disease. In recent years, we have learned more about the virus roles of B cells, CD8+ T cells, cerebral endothelial cells, and various pro- and anti-inflammatory cytokines in the development and maintenance of the continuous pathology of MS. The roles of B cells, CD4+ T cells and CD8+ T cells should be described and focus on the role of cerebral endothelial cells and their interactions with activated leukocytes in pathogenesis of MS in further research.