Multiple Sclerosis: New Hypotheses Are Needed

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Short Communication

Multiple Sclerosis (MS) was recognized as a distinct disease involving both the brain and spinal cord and associated with disseminated demyelinating plaques ("la sclérose en plaques") and axonal damage more than 145 years ago by French neurologist Jean-Martin Charcot [1]. Since that time, the world has witnessed many remarkable discoveries. Due to progress in physics and mathematics, MRI has become a powerful tool in neurology allowing us to follow the progression of demyelinating lesions in living patients. Based on progress made in the field of immunology, a number of immunomodulatory drugs have been discovered, tested in clinical trials and become standards of care for the treatment of patients with the disease but only delay its activity and progression. DMTs have limited clinical efficacy and may have significant adverse effects. At present, we do not know what causes MS and, therefore, we cannot develop the MS cure.

It has been well documented that MS pathogenesis involves both inflammation and neurodegeneration. Inflammation is more common in relapsing-remitting form of MS compared to primary-progressive form of MS and, in the past, was considered a primary feature of MS, as in progressive multifocal leukoencephalopathy, or initiate immunopathological demyelination, as in animals infected with Theller's murine encephalomyelitis virus or coronaviruses [12]. However, no MS-specific virus has yet been isolated from the brains of patients.

The second hypothesis suggests that MS is an infectious disease. For example, it was hypothesized that a certain virus might reactivate after years of latency and lyse oligodendrocytes in the CNS of patients with MS, as in progressive multifocal leukoencephalopathy, or initiate immunopathological demyelination, as in animals infected with Theller's murine encephalomyelitis virus or coronaviruses [12]. However, no MS-specific virus has yet been isolated from the brains of patients.

The third hypothesis suggests that patients with MS have chronic cerebrospinal venous insufficiency (CCSVI) causing delayed venous outflow from the brain and spinal cord [13]. However, the initial promising results of Dr. Zamboni and coauthors could not be reproduced by other researchers. It was later reported that CCSVI occurs rarely in both patients with MS and in healthy people [14].

All three hypotheses mentioned above provide a reasonable explanation why the disease selectively involves the brain and spinal cord and does not affects other organs. However, they do not explain the key epidemiological finding of decreased MS prevalence among Asians. It is very likely that new hypotheses addressing the cause of MS are going to emerge in the near future. Therefore, two mandatory criteria are proposed for a new biological factor/mechanism hypothesized to be a MS cause:

**Criterion 1.** The new hypothesis has to explain why the disease is restricted to the CNS and causes both inflammation and neurodegeneration.

**Criterion 2.** The new hypothesis has to explain why the disease has low prevalence in Asia.

References