Abstract

Multiple sclerosis (MS) is an inflammatory disorder, in which neurons become demyelinated. To date, its etiology has remained unknown. Nevertheless, certain features are inspected to provoke MS. For instance, improper function of immune cells is widely believed to be the basis of such disorder. In this concept, MS is stated as an autoimmune disease, which was asserted by major of studies, as CD8+ T-lymphocyte reacts to any agent containing major histocompatibility complex class 1 (MHC 1). They pass epithelium of brain capillaries to access locations where oligodendrocytes abnormally contain production of such gene. In this hypothesis, most articles assign peripheral immune system for disrupting the structure of blood brain barrier (BBB), which involves series of sequential mechanisms regulated by numerous genes of endothelium of BBB, as well as immune system. During last few decades other etiologies have been proposed, which are in intimate relationship with BBB. In 2015, a new pathway was discovered in which leukocytes might recruit and travel more easily in and out of the brain tissue. These newly discovered lymphatic vessels also might be in association with MS. The aim of this paper is to present the role of BBB in pathogenesis of MS. In conclusion, autoimmunity of MS is well asserted by most studies, which aimed to propose further etiological facts of MS. However, importance of each molecules and genes leading to its autoimmunity or disruption of BBB is yet to be more particularly determined.

Keyword: Blood brain barrier, Multiple sclerosis, Neuroinflammation

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