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Molecular mechanism of disruption of blood brain barrier in Experimental autoimmune encephalomyelitis

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Abstract

Introduction: Blood brain barrier (BBB) is made by endothelial capillary cells. These cells use complex tight junctions to restrict paracellular diffusion. In EAE (Experimental autoimmune encephalomyelitis), animal model of Multiple sclerosis, we increase permeability of blood brain barrier. Many articles show that angiogenesis has a significant role in disruption of BBB. Astrocytes, pericytes, neurons, endothelial cells and prevascular microglia are the cells that present in BBB environment. Disruption in signaling pathway between these cell cause breakdown of BBB. What is happening is cooperation of proinflammatory cytokines or angiostatic agents. Astrocytes balance endothelial stability and permeability.

Materials and Methods: We gather information from articles related to angiogenesis, tight junction and blood brain barrier by searching in pub med and Google scholar from the year 1990.

Results: Up regulation of Astrocyte derived VEGF-A, cause decrease in CLN-5 and OCLN molecules which participate in tight junction, thus weakening it, in other hand VEGF-A by influencing VEGFR positive cells, macrophage, causes production of angiogenic factors, including IL-1, and angiogenesis happens.

Conclusion: ALL in all, there is a circling pathway between angiogeneic agents and cells present in BBB environment. Endothelial cells and Astrocytes show significant role in controlling breakdown of BBB by angiogenic factor and tight junction molecules. Having the knowledge of these mechanisms could be helpful for designing more sophisticated future studies of Multiple Sclerosis, and also it can help to design new approaches to facilitate drug delivery in the case of Central Nervous System diseases.

Key words: *Tight junction, Angiogenesis, Occludin, Endothelial cell, Proteins claudin-5.*