The Role of Inflammation in the Seizure Occurrence

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Abstract

Most common hypotheses of seizure initiation are increased neural excitation, decreased inhibition or both. But, the conditions that lead to these activation states not to be clear yet. Recent studies challenge traditional concepts and indicate new evidence that a key epileptogenic process may actually begin in the blood vessel. Seizures could be initiate by a variety of insults to the brain, such as trauma, infection, hypoxia, fever. Recent research has highlighted putative role of blood-brain barrier (BBB) permeability in the evolvement of epilepsy. Local or global changes of the brain’s homeostatic environment can pathologically alter neural activity via admission of intravascular proteins such as albumin and altered electrolyte levels such as potassium. Investigations showed immune-mediated damage to the nervous system is extruding as an important contributor to epileptogenesis, both directly through inflammation and indirectly by causing BBB leakage. Pilocarpine administration as a valid temporal lobe epilepsy in the animal models increased in cell adhesion molecules in brain blood vessels, which are important moderators for leukocyte extravasation during inflammatory processes. Chiefly, expression of the some intercellular adhesion molecule such as: vascular cell adhesion molecule-1 (VCAM-1), P-selectin and E-selectin was increased at one day and seven days after status epilepticus, compared to control groups. In the similar studies researchers expressed enhanced leukocyte adhesion in the central nervous system vessels. These findings indicate that seizure activity is accompanied with leukocytic inflammatory alteration in the central nervous system vasculature. More important point is that leukocyte-endothelial interactions determined whether seizures occurred, and subsequent extent of structural, cognitive and physiological damage, including the development of epilepsy.

Keywords: Inflammation, Seizure Occurrence, blood-brain barrier

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