Update and Future Directions in Poststroke Epilepsy

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Published: 24 August, 2018

Abstract

Concepts of latency and epileptogenesis form an important basis for clinical understanding of the early versus late seizures in stroke patients. Two models of epileptogenesis is postulated in stroke patients. First, loss of neurovascular unit integrity (BBB disruption) leads to local metabolic disturbances without disturbance in neuronal networks causes seizures immediately after a stroke. Late seizures may be secondary to gliosis and development of meningoencephalitis scarring. Changes in membrane properties, differentiation, selective neuronal loss, and collateral sprouting may result in hyperexcitability and neuronal synchrony sufficient to elicit seizures and predispose to epilepsy. These models of seizure carry different risks of seizure recurrence for post-stroke epilepsy, early and late. Recent studies show effect of drugs in the processes of PSE. Statin with anti-inflammatory and antioxidant properties is postulated to have role in reduction of epileptogenicity in early post-stroke epilepsy. LEV by inhibition of inflammatory responses and reduction of reactive gliosis in the hippocampus and piriform cortex in a rat model of epilepsy could be an important agent in the prevention of epileptogenesis far from as anti-epileptic drug. Future studies should aim to clarify the impact of AED treatment on vascular-risk profile and rehabilitation. Role of biomarkers and neuroimaging in prevention of PSE, reduction of brain damage and deterioration in neurological function, is going on.

Keywords: Biomarkers, PSE, Function, BBB, Brain Damage.

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