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Poster Presentation

Temporal Lobe Epilepsy and Mitochondrial Dysfunction

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Abstract

Introduction: Epilepsy is a neurological disorder characterized by recurrence seizures resulting in disturbing effects on patients. Human temporal lobe epilepsy (TLE) is a common destructive neurological disorder described by recurrent seizures and selective susceptibility of hippocampal neurons in specific parts of the hippocampus. The emphasis was placed on the role of mitochondrial dysfunction in epilepsy, with the fact that epilepsy is often inherited with mitochondrial disorders, such as myoclonic epilepsy with ragged red fibers (MERRF) epilepsy and also it is related with encephalopathy in children. Damage to proteins, lipids, mitochondrial DNA is considered as evidence for consequences of mitochondrial dysfunction in different animal models of TLE. The mitochondrial oxidative stress has been considered as the leading cause of epileptic seizures. Mitochondria is one of the most important organelles in cells and control neuronal excitability by controlling adenosine triphosphate (ATP) production, oxidation of fatty acid, amino acid, and neurotransmitter biosynthesis regulation. The first place where reactive oxygen species (ROS) is produced in mitochondria. On the other hand, mitochondria are too susceptible to oxidative damage which controls neuronal excitability. Initial trauma associated with TLE induces complex molecular, biochemical, physiological, and structural changes in the brain mitochondria that contribute to epileptogenesis and the subsequent onset of spontaneous and recurrent seizures. Mitochondrial damage is shown in studies from human imaging and tissue analysis from TLE patients. The mitochondrial malfunction can be considered as a central factor for seizures. In addition, mitochondrial oxidative stress may have a pathologic role in temporal lobe epilepsy. Another reason which attributes mitochondrial oxidative stress to seizure generation in TLE is age-dependent epileptogenesis and increasing in mitochondrial oxidative stress with age. **Conclusion:** It is proposed mitochondrial dysfunction high relevance in seizure generation in temporal lobe epilepsy with Ammon's horn sclerosis. Furthermore, mitochondria should be regarded as a promising target for future neuroprotective strategies in epilepsy.

Keywords: Temporal Lobe Epilepsy, Mitochondrial Oxidative Stress.

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