SHORT COMMUNICATION

Vitamin E as a therapy method for epilepsy and neurodegeneration

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ABSTRACT

Epilepsy is the most common neurological illness, with about 50 million people suffering from it globally. The disorder might be caused by a hereditary predisposition or acquired as a result of an acute insult that alters cellular and molecular pathways. Hyperexcitability is a result of an imbalanced state in which increased excitatory glutamatergic and decreased inhibitory GABAergic signalling is thought to be responsible for seizures-related damage, according to the latest and current knowledge in regard to the mechanisms underlying molecular and cellular change. However, the role of neurodegeneration in epileptogenesis is becoming more widely recognised. GABAergic neuronal and receptor modifications, neuroinflammation, axonal transport changes, oxidative stress, excitotoxicity, and other cellular and functional changes are all at the forefront of neurodegenerative changes during epileptogenesis. Vitamin E as an antioxidant, anti-inflammatory, and neuroprotective agent may show to be one of the therapeutic options beneficial in the treatment of epilepsy. We analyse and debate seizure-induced events as a link for the development of epilepsy's neurodegenerative and pathological repercussions in this paper. We also provided an overview of the possible use of vitamin E therapy in the treatment of epilepsy.

Key word: *Epilepsy; Neurodegeneration; Oxidative stress; Excitotoxicity neuroinflammation; Vitamin E*

SHORT COMMUNICATION

Epilepsy is a prevalent neurological illness that affects about 50 million people throughout the world. Recurrent seizures are a symptom of the condition, which are caused by abnormal neuronal networks that cause synchronous discharges, disrupting normal neuronal activity. The condition manifests several signs and symptoms, including electro clinical seizures, which include abnormal levels of consciousness and motor manifestations. The phrase refers to a wide range of disorders that have no clear cause and can be accompanied with neurological, psychological, cognitive, and social consequences that might damage patients' quality of life when they do not respond to typical treatment [1]. The condition can occur as a result of a genetic predisposition or as a result of an acute insult that causes changes in cellular and molecular mechanisms in the affected region, resulting in seizures that occur immediately or after a latent period and last until the occurrence of recurring seizures.

Temporal Lobe Epilepsy (TLE) is the most common type of epilepsy in adults, accounting for around 30% of all seizures [2]. It is characterised by spontaneous recurring motor seizures, particularly in the hippocampus. Brain damage, infection, status epilepticus (SE), brain tumours, and genetic or congenital anomalies are among factors that contribute to acquire TLE.

Based on current knowledge of the mechanisms causing molecular and cellular changes, neuronal hyperexcitability is thought to be the result of an imbalance including increased excitatory glutamatergic transmission and decreased inhibitory GABAergic signalling. Despite the fact that these hypotheses are widely accepted, standard medications that target these pathways have proven ineffective [3].

The neurodegenerative implications of seizures are the topic of this review. We examine the literature to see if seizure-induced events are linked to the development of epilepsy's neurodegenerative and pathological repercussions. This review also provides some insight into the potential for vitamin therapy to be used in the treatment of epilepsy.

On the basis of common memory problems, a link between epilepsy and neurodegeneration has been established. According to the study, around 50 percent of epilepsy sufferers have cognitive abnormalities

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[4]. Excess deposition of amyloid to hyperexcitability, resulting in neuronal loss, altered synaptic plasticity, and neuronal circuit remodelling, appears to be involved in the process of epileptogenesis, the progressive alteration of a normal to a hyperexcitable epileptic brain even after the stimulus is removed, in dementia. In addition, the most prevalent link between epilepsy and dementia in Alzheimer's disease is the temporal lobes. Alzheimer's disease patients have seizures, and 13.4% of epileptic patients had AD, according to neuropathological research.

Tau protein has also been demonstrated to cause toxicity in epilepsy patients with dementia [5].

Thus, substantial changes in neuronal plasticity, cellular and molecular modifications, and neurogenesis may cause neuronal loss in the epileptogenic brain, leading to various neurobiological disorders. Other clinical and experimental data has linked epilepsy to a variety of neurodegenerative pathways, including the Mammalian Target of Rapamycin (mTOR) (mTOR). Furthermore, data from experimental models of acquired epilepsy provided a better understanding of the role of different neurodegenerative pathways in excitotoxicity, neurogenesis, and cell death, which could aid in the development of anti-epileptic therapies by identifying alternative pharmacological targets to traditional treatments [6].

The bidirectional association between neurodegeneration in dementias and epilepsy could be explained by these similarities. Neuron loss and activated astrocyte, improved blood-brain barrier permeability, axonal sprouting, and neuroinflammation are all direct links between epilepsy and numerous neurodegenerative abnormalities in areas engaged in repetitive seizures. Similar neurodegenerative changes have been documented as a result of frequent convulsions in various animal models of seizures.

Superoxide Radical (SOD), Hydroxyl Radical (•OH), Hydrogen Peroxide (H2O), and singlet oxygen are examples of Reactive Oxygen Species (ROS) that are created during aerobic respiration when electrons leave complex I and III of the ETC in the mitochondria (Odouble bondO). Enzymatic (e.g., glutathione reductase, superoxide dismutase, glutathione peroxidase, and catalase) and nonenzymatic (e.g., glutathione peroxidase, glutathione peroxidase, and catalase) antioxidant defence mechanisms scavenge typical physiological levels of ROS (e.g., vitamin E and C and decreased glutathione). Excess ROS production and/or a reduction in defensive ability, on the other hand, might result in high levels of oxidative stress. Due to high oxygen consumption, a large number of mitochondria, and a lack of antioxidant defence ability, the human brain is more vulnerable to ROS-induced damage than other organs [7,8].

ROS causes cellular damage and functional changes, as well as neuronal death. Protein oxidation can cause essential enzymes to become inactive. Lipid peroxidation can damage membrane structure, affecting membrane fluidity and permeability, as well as protein activity. As a result, the pathophysiology of neurodegenerative illnesses is regarded to be complicated [9].

Traditional antiepileptic drugs are designed to stop seizures, therefore they only provide symptomatic relief and have no effect on the disease's progression; also, there is no recognised treatment aimed at neuroprotective techniques to reverse neurodegeneration. Indeed, early seizure cessation combined with an appropriate strategy aimed at inhibiting components and pathways that cause neurodegeneration may aid neurodevelopment. As a result, neuroprotective therapy can be used as an epilepsy treatment technique. As a result, recent research has focused heavily on treatments that protect neuronal cells, resulting in fewer seizures or seizure suppression. Tocopherol, or vitamin E, is a lipophilic alcohol having biological action. Because it accounts about 90% of tocopherol in animals, the -form is thought to be the most physiologically active component. Vitamin E's positive effects on oxidant stress have been attributed to its antioxidant capabilities. However, research has recently discovered that it may potentially modulate pathogenic variables in epilepsy that are not related to oxidative stress, such as neuroinflammatory enzymes and signalling cascades [10].

-T has been found to inhibit the activity of Protein Kinase C (PKC), an enzyme that reduces GS expression in the hippocampus, independent of its antioxidant properties.

CONCLUSION

This expanded review focuses on the role of oxidative stress, excitotoxicity, and neuroinflammation in epilepsy-induced neuron damage and their putative role in epileptogenesis. Neurodegeneration causes various alterations, including the release of Ca2+, oxidative stress, and apoptotic factors, which causes astrocytes and microglia to release inflammatory cytokines, resulting in hyperexcitability and epileptogenesis. Using vitamin E as an antioxidant, anti-inflammatory, and neuroprotective to target neurodegeneration could help manage recurrent epilepsy. Continued research in this area will lead to a better understanding of the role of neurodegeneration in the pathogenesis of seizures, as well as how vitamin E's neuroprotective mechanism targeting seizure-induced neurodegeneration can be used to help patients who are taking current antiepileptic drugs..

REFERENCES

1. Black LC, Schefft BK, Howe SR, et al. The effect of seizures on working memory and executive functioning performance. Epilepsy Behav. 2010; 17(3):412-419.

2. Engel J. A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE task force on classification and terminology. Epilepsia. 2001; 42(6): 796-803.

3. Fisher RS, Van W, Blume W, et al. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). Epilepsia. 2005; 46(4): 470-472.

4. Patel DC, Tewari BP, Chaunsali L, et al. Neuron–glia interactions in the pathophysiology of epilepsy. Nat Rev Neurosci. 2019; 20(5): 282-297.

5. Mrcp MM, Hart YM, Sander JWAS, et al. National general practice study of epilepsy (ngpse): partial seizure patterns in a general population. Neurology. 1992; 42(10): 1911-1917.

6. Herman ST. Epilepsy after brain insult: targeting epileptogenesis Neurology. 2002; 59: S21-S26.

7. McNamara JO. Emerging insights into the genesis of epilepsy. Nature. 1999; 399(6738): A15-A22.

8. Sharma AK, Reams, RY, Jordan WH, etal. Mesial temporal lobe epilepsy: pathogenesis, induced rodent models and lesions. Toxicol Pathol. 2007; 35(7): 984-999.

9. Dalby NO, Mody I. The process of epileptogenesis: a pathophysiological approach. Curr Opin Neurol. 2001; 14(2): 187-192.

10. Kwan P, Brodie MJ. Refractory epilepsy: mechanisms and solutions. Expert Rev Neurother. 2006; 6(3), 397-406.