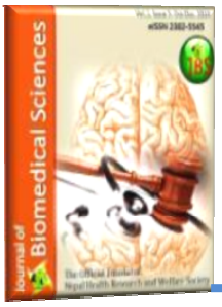

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EDITORIAL

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Editorial

Epilepsy is a commonly found neurological disorder which is known to occur from various biochemical and molecular changes. Related to these changes oxidative stress is found to play an important role in the etiology of seizure-induced neuronal death. A mitochondrial disease arising due to defects in oxidative phosphorylation is often characterized by epileptic seizure like activities [1]. Studies showed that epileptic seizures lead to excess production of reactive oxygen species (ROS) thereby resulting in the development of oxidative stress [2]. This causes increased secretion of corticotrophin releasing hormone (CRH) which in turn causes increased secretion of glucocorticoids and thereby inactivation of macrophages leading to immunosupression. Glucocorticoids suppress the immune response via negative feedback mechanism. Oxidative stress leads to macrophage inactivation and thereby immunosuppression. As macrophage inactivation in turn modifies the production of IL-1, this alteration mutilates cell mediated immunity (CMI) as well as humoral immunity thereby, devastating the antibody production as well as cell mediated cytotoxic killing. Thus, contributing to immunosuppression, this is finally responsible for worsening the epileptic condition [3]. The white blood corpuscle (WBC) of the immune system manipulates the activity of the central nervous system (CNS) by the production of cytokines which is responsible for activation of the Hypothalamus- Pituitary – Adrenal axis (HPA axis). Though cytokines modulate hypothalamus but at the same time immune system is also modulated by the hypothalamus. This occurs through two routes, direct innervations and endocrine mechanism. Organs like thymus and spleen have been found to receive a direct impulse from the autonomic nervous system [4].

The observations regarding the epileptic seizures reveals, the dysfunction that occurs within the mitochondria lead to more oxygen consumption to make ATP. In such cases the mitochondria is studied to produce highly ROS called free radicals. These excess free radicals damage the mitochondrial functions by changing the mitochondrial DNA, proteins, and membranes as the mitochondria fails to detoxify them. The continuation of such process may lead to cell apoptosis. Similar deficiencies of oxidative phosphorylationhas been studied in epileptogenic brain regions of therapy-resistant focal epilepsies, such temporal lobe epilepsy with Ammon's horn sclerosis [5]. Seizures develop due to ROS under some pathophysiological conditions and seizure-induced neurodegeneration. Research

evidenced that there is the production of ROS in amygdala-kindled rat models which led to the development of seizure and seizure-induced neuronal loss [6]. Investigations established a relationship between the free radical and scavenger enzymes of our body. The superoxide dismutase and catalase activities were studied to be increased during the chronic phase. In addition, lipid peroxidation and nitrite levels were also found to increase during spontaneous recurrent seizures. Normal levels of superoxide dismutase with increase in catalase activities, hippocampal lipid peroxidation and nitrite concentrations were observed in research with animals showing seizure activities and submitted to 24 h of status epilepticus. The studies with pilocarpine model of epilepsy evidenced a link between the lipid peroxidation and nitrite during seizure activity and it was also established that these can be the cause behind the neuronal damage in the hippocampus of rats. [7]. The clinical and experimental findings claims that increase in lipid peroxidation in the epileptogenic foci contribute to the production of ROS. An increase in the products of lipid peroxidation in the cerepbrospinal fluid (CSF) and in peripheral blood plasma in the epileptic cases found to be responsible for antioxidant deficiency and thereby development of oxidative stresses [8]. These findings can be linked with the decrease in the glutathione peroxidase activity and decreased in haptoglobin, transferrin and ceruloplasmin functions [9]. Thereby, the crumbling condition in epileptic cases can be complimented with the help of antioxidants.

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