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# Assessment of Reactive Oxygen Species Status and Neuroprotection by Vitamin E in Chronic Cerebral Hypoperfusion Induced Neurodegeneration in Rats

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**Background:** Chronic cerebral hypoperfusion has been linked with cognitive decline in ageing and Alzheimer's disease (AD). A reduced cerebral blood flow (CBF) is considered to be a predictive marker for the progression into Alzheimer's disease. In order to tackle the neuropathological consequences of such a reduction in CBF a similar condition was created in experimental animals. Chronic cerebral hypoperfusion induced neurodegeneration was produced by common carotid artery occlusion (2 vessel occlusion, 2VO) in rats. Since oxidative stress, leading to neuronal apoptosis and death, is one of the mechanisms which is thought to play a significant role in chronic degenerative neurological disorders, the present study was designed to assess the neuroprotective role of Vitamin E in chronic cerebral hypoperfusion-induced neurodegeneration.

**Methods:** After acclimatization, thirty Sprague Dawley rats weighing 200-250 g were equally divided into three groups. Group A – sham control, Group B – 2VO, and Group C – 2VO-E (treated daily with Vitamin E, 100 mg/kg, orally following 2VO). On the 8$^{th}$ week, all the rats were euthanized and the hippocampi were isolated. Viable neuronal cell count in the hippocampal CA-1 region was estimated.

**Results:** There was significant difference in neuronal cell death in 2VO group as compared to sham control group. In Vitamin E-treated 2VO (2VO-E) rats, the viable neuronal cell count of the hippocampal CA-1 region was significantly higher as compared to the untreated 2VO group.

**Conclusion:** Vitamin E is an effective neuroprotective agent in chronic cerebral hypoperfusion-induced neurodegeneration in rats and can be effectively utilized in the management of AD. The antioxidant activity of Vitamin E is in progress and will be evaluated by the measurement of Isoprostane F2 through Enzyme-Linked Immunosorbent Assay.

**Keywords:** Vitamin E, chronic cerebral hypoperfusion, neurodegeneration, Alzheimer's disease, oxidative stress