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Sinapic acid reduces pentylenetetrazol induced seizures in rats

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Seizure is known to induce oxidative stress which may initiate neuronal death. Oxidant-antioxidant imbalance often leads to mitochondrial dysfunction, inflammation, and apoptosis in the brain which may further result in the development of seizure. Phenolic compounds such as curcumin and rosmarinic acid are reported to control convulsions and seizures in pentylenetetrazol induced seizures models by suppressing seizure time, oxidative stress and inflammation indirectly. Sinapic acid (SA), a polyphenolic product of hydroxycinnamic acid found in various plants, exhibits anti-inflammatory, antioxidant and anxiolytic effects. In this study, we investigated the effects of sinapic acid on pentylenetetrazol induced seizures in rats through oxidative stress, inflammation, apoptosis, and neurotrophic factor. A total of 28 male Wistar Albino rats weighing 200-220 g were divided into four equal groups (n=7/group). The treatment groups received 10 mg/kg and 20 mg/kg SA, respectively, by oral gavage for five consecutive days along with pentylenetetrazol (45 mg/kg, intraperitoneal) to induce seizures. The levels of Total oxidant status (TOS), Total antioxidant status (TAS), TNF-α, IL-1β, and Brain-derived neurotrophic factor (BDNF) were measured in the cortex and hippocampus. Additionally, caspase 3 and caspase 9 levels, as well as the immunoreactivity of Cleaved caspase 3, were determined in the hippocampus. The results showed that pretreatment with 20 mg/kg SA delayed the latency of generalized tonic-clonic seizures (GTCS) and first myoclonic jerk, reduced GTCS duration, and improved seizure score and cognitive function. Importantly, the 20 mg/kg SA pretreatment resulted in decreased levels of TOS, TNF-α, IL-1β, and BDNF in the cortex and hippocampus, while increasing TAS levels in these brain areas. Moreover, the 20 mg/kg SA reduced hippocampal caspase 3 and caspase 9 levels, as well as the immunoreactivity of Cleaved caspase 3 in rats with pentylenetetrazol-induced seizures. These findings suggest that the anti-seizure effects of SA are mediated by BDNF modulation, as well as its antioxidant, anti-inflammatory, and anti-apoptotic properties.

Keywords: Anticonvulsant activity, Antiseizure activity, Apoptosis, Brain-derived neurotrophic factor (BDNF), Epilepsy, Experimental acute seizure model, Generalized tonic-clonic seizures (GTCS), Inflammation, Oxidative stress, Racine convulsion scale (RCS)