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Neuroprotective effects of clenbuterol against experimentaly induced epileptic seizures in rats

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Epilepsy is a neurological condition described by repeated unpredictable interruptions of normal brain function, named epileptic seizures. Oxidative stress, nitrosative stress, inflammation and apoptosis are considered as potential mechanisms underlying the pathogenesis of epilepsy. In this study, we used Levetiracetam (LEV) as a standard anti-epileptic treatment. Clenbuterol (CLEN), a lipophilic β2-adrenoceptor agonist is a therapeutic drug for asthma and COPD. The possible neuroprotective actions of CLEN were investigated by unveiling its potential in preventing oxidative stress, inflammation and apoptosis compared to LEV. Rats were allocated into 4 groups. In the convulsive group, rats received a single i.p. injection of 3 mEq/kg lithium chloride 20 hours prior to a single i.p. injection of 150 mg/kg pilocarpine-hydrochloride. The control group received 3 mEq/kg lithium-chloride dissolved in normal saline. The third and fourth group, rats received i.p. 500 mg/kg LEV 30 minutes and 0.5 mg/kg CLEN for 7 days before lithium-pilocarpine (Li-PIL) administration respectively. Both drugs alleviated Li-PIL seizures and motor deficits, represented as open field parameters, in rats, which attributed to preservation of hippocampal reduced glutathione and decrease in total thiobarbituric acid reactive substances and nitric oxide contents. Furthermore, a reduction in tumor necrosis factor-α, interleukin-1β was observed. Moreover, both drugs protected hippocampal neurons against apoptotic death assured by a decrease in caspase-3 and cytochrome-c levels. Collectively, our results suggest that CLEN might possess a promising therapeutic effect in Li-PIL -induced epileptic experimental model and other pathological aberrations in epilepsy via significant neuroprotective effects mediated by antioxidant, anti-inflammatory and anti-apoptotic effects.

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