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The effect of some immunomodulatory and anti-inflammatory drugs on Li-pilocarpine-induced epileptic disorders in Wistar rats

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Evidence shows that inflammatory and immune processes within the brain might account for the pathophysiology of epilepsy. Therefore, developing new antiepileptic drugs that can modulate seizures through mechanisms other than traditional drugs is required for the treatment of refractory epilepsy. This study aims to determine the relationship between brain inflammation and epilepsy, to examine the contribution of some biochemical parameters involved in brain inflammation, and to address the effect of pharmacological interventions using some anti-inflammatory and immunomodulatory drugs in an experimental epilepsy model. Adult male rats were divided into seven groups of 20. G1 was the normal, non-treated control. G2 was the epileptic, non-treated group. G3–G7 were treated with celecoxib, methotrexate, azathioprine, dexamethasone, and valproate, respectively, for a period of three weeks. Induction of status epilepticus (SE) by Li-pilocarpine was performed on groups G2–G7. EEG tracing was conducted, and inflammatory mediators (brain and serum IL-1ß, IL 6, PGE2, HSP70, TGF-β2, and IFNγ) were measured. The induction of SE increased the amplitude and frequency of EEG tracing and inflammatory mediators more than in the normal control group. Treatments of epileptic rats reduced the frequency and amplitude of EEG tracing and significantly decreased the levels of inflammatory mediators in some treated rats compared to G2. These findings demonstrate that some anti-inflammatory and immunomodulatory drugs can lower the frequency and amplitude of seizures and reduce some inflammatory mediators in epilepsy treatments, strengthening the possibility that targeting these immunological and inflammatory pathways may represent another effective therapeutic approach to preventing epileptic seizures.

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