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Review Article

Rats With Epilepsy Caused by Kainic Acid or Quinolinic Acid Were Used to Test the Neuroprotective and Cognitive-Enhancing Effects of A Herbal Extract

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ABSTRACT

Background: Recurrent seizures, neuronal degeneration, oxidative stress, neuroinflammation, and cognitive impairment are the hallmarks of epilepsy, a long-term neurological condition. Because they resemble the neuropathological and behavioural characteristics of temporal lobe epilepsy, excitotoxic substances like quinolinic acid (QA) and kainic acid (KA) are frequently employed to create experimental epilepsy in mice. In experimental models of epilepsy, herbal extracts rich in polyphenols, flavonoids, and alkaloids have shown encouraging neuroprotective, antioxidant, anti-inflammatory, and cognition-enhancing qualities. **Objective:** The current study sought to assess the neuroprotective and cognitive-enhancing benefits of a standardized herbal extract in rats with epilepsy caused by KA or QA and to explore potential mechanisms of action. **Methods:** Five groups (n = 6–8) of adult Wistar rats were randomly assigned to receive either standard therapy, epileptic control, normal control, or two treatments with herbal extract. According to the experimental methodology, a single injection of either QA or KA (10 mg/kg, i.p.) caused epilepsy. For 14–21 days, the herbal extract was taken orally. Seizure scoring, locomotor activity, passive avoidance, Y-maze, and Morris water maze tests were among the behavioral evaluations used to assess memory and learning. To identify oxidative stress indicators including malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase, biochemical studies were carried out.

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Additionally assessed were neuroinflammatory indicators such as interleukin-1 β (IL-1 β), tumor necrosis factor- α (TNF- α), and neuronal damage markers. To evaluate neuronal deterioration and neuroprotection, hippocampus areas were examined histopathologically.

Results: When compared to the epileptic control group, treatment with the herbal extract significantly decreased the frequency and severity of seizures ($p < 0.05$). By improving memory retention and spatial learning in behavioral paradigms, the extract enhanced cognitive function. MDA levels were significantly lowered and endogenous antioxidant defenses, such as GSH, SOD, and catalase levels, were significantly restored. Additionally, the herbal extract shielded hippocampus neurons from excitotoxic injury and reduced the generation of neuroinflammatory cytokines. The treated animals' hippocampus architecture was preserved and there was less neuronal loss, according to histopathological results. These effects were similar to those seen with the common antiepileptic medication.

Conclusion: The results indicate that the herbal extract has important neuroprotective and cognitive-enhancing properties against seizures in rats caused by KA or QA. Its anti-inflammatory, anti-apoptotic, and antioxidant qualities may be responsible for the reported advantages. As a result, the herbal extract could be a viable treatment option for the treatment of neuronal damage and cognitive impairment brought on by epilepsy

INTRODUCTION

One of the most common chronic neurological conditions in the world, epilepsy affects around 50 million individuals and has a significant impact on society, the economy, and healthcare. Recurrent, unprovoked seizures brought on by aberrant, excessive, and synchronized brain neuronal discharges are the hallmark of the illness. Almost one-third of patients still have uncontrollable seizures despite the availability of various antiepileptic medications (AEDs), and many endure side effects such as drowsiness, cognitive impairment, mental disorders, hepatotoxicity, and drug resistance. Therefore, one of the biggest challenges in epilepsy research continues to be the creation of safer and more successful treatment techniques. [1-8]

Epilepsy is becoming more widely acknowledged as a condition linked to oxidative stress, neuroinflammation, progressive neuronal damage, and cognitive impairment in addition to repeated seizures. Deficits in learning, memory, attention, and executive function are common in patients with temporal lobe epilepsy, which greatly lowers their quality of life. Excitotoxic neuronal damage, a pathogenic process driven by excessive activation of glutamate receptors that results in calcium overload, mitochondrial malfunction, oxidative stress, and ultimately neuronal death, is mostly responsible for these neurological problems. As a result, current research focuses on preventing neurodegeneration and maintaining cognitive function in addition to seizure management. [9-16]

To study the processes behind epileptogenesis and to assess possible treatments, experimental models of epilepsy have been widely used. Because they closely mimic numerous clinical and behavioral characteristics of real temporal lobe epilepsy, kainic acid (KA) and quinolinic acid (QA)-induced epilepsy are the most extensively recognized of these models. As a strong agonist of ionotropic glutamate receptors, especially kainate receptors, kainic acid causes oxidative stress, gliosis, neuronal degeneration, extended seizure activity, and hippocampus injury. Learning and memory losses are severe when KA is administered because it causes selective neuronal death in hippocampus areas including CA1, CA3, and the dentate gyrus. Similar to this, quinolinic acid, an endogenous metabolite of the kynurenine pathway, functions as a selective agonist of the N-methyl-D-aspartate (NMDA) receptor and causes excitotoxic neuronal damage by generating reactive oxygen species and excessive calcium influx. QA-induced neurotoxicity is a useful paradigm for researching neuroprotective therapies since it is linked to neuronal



degeneration, neuroinflammation, mitochondrial malfunction, and cognitive impairment.^[17-34]

A growing body of research indicates that oxidative stress is a key factor in the pathophysiology of epilepsy and neuronal damage brought on by seizures. Lipid peroxidation, protein oxidation, DNA damage, and neuronal death are the outcomes of natural antioxidant defense mechanisms being overloaded by excessive formation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) during epileptic episodes. Both clinical research and experimental epilepsy models have shown elevated levels of malondialdehyde (MDA) and decreased activity of antioxidant enzymes such glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD). Moreover, pro-inflammatory cytokines such as interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) are released when seizures activate microglia and astrocytes. These cytokines cause neuronal hyperexcitability and neurodegeneration. Thus, medicinal substances with anti-inflammatory and antioxidant qualities may be quite helpful in reducing epileptic brain damage.^[34-40]

Due to their long history of traditional usage, acceptable safety profiles, and multitarget pharmacological activities, medicinal plants and herbal medicines have garnered significant attention as complementary and alternative therapy alternatives for neurological illnesses. Preclinical studies have shown that a variety of phytochemicals, including as flavonoids, polyphenols, alkaloids, terpenoids, and phenolic acids, have neuroprotective, anticonvulsant, antioxidant, anti-inflammatory, and cognition-enhancing properties. These bioactive substances have the ability to control calcium homeostasis, limit oxidative stress, inhibit neuroinflammatory signaling pathways, alter neurotransmitter

systems, and stop neuronal death. As a result, plant extracts are intriguing options for treating epilepsy and the neurological issues it causes.^[37-45]

Recent studies have shown that a number of herbal extracts provide protection against cognitive impairments and experimentally produced seizures. Enhancement of endogenous antioxidant defenses, decrease of lipid peroxidation, attenuation of inflammatory cytokine production, regulation of glutamatergic and GABAergic neurotransmission, and maintenance of hippocampal neuronal integrity have all been linked to these effects. However, there is still much to learn about the therapeutic potential of many medicinal plants, especially when it comes to neurodegeneration and cognitive impairment linked to seizures.^[41-47]

Many bioactive phytoconstituents with antioxidant and neuroprotective qualities are found in the herbal extract used for this study. According to preliminary pharmacological research, the extract may have strong anti-inflammatory, free radical scavenging, and cognitively enhancing properties. Nevertheless, a thorough assessment of its effectiveness against excitotoxin-induced seizures and associated neuronal injury has not yet been conducted. It is thus necessary to conduct a thorough evaluation of its potential for neuroprotection and cognitive enhancement. The goal of this study was to assess the herbal extract's neuroprotective and cognitive-enhancing properties in rats with epilepsy caused by kainic acid or quinolinic acid. Learning, memory, and seizure severity were all assessed behaviorally. Oxidative stress and antioxidant levels were assessed biochemically, while hippocampus architecture and neuronal integrity were examined histopathologically. Treatment with the herbal extract was predicted to improve cognitive function, reduce seizure-induced



neuronal damage, enhance antioxidant defences, and reduce neuroinflammatory responses. The results of this study may aid in the creation of

innovative plant-based treatment approaches for the treatment of epilepsy and related cognitive deficits.^[44-50]

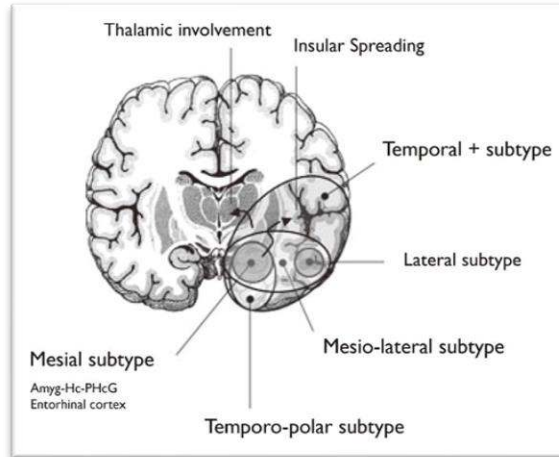


Image No.01: Epilepsy and Hippocampal

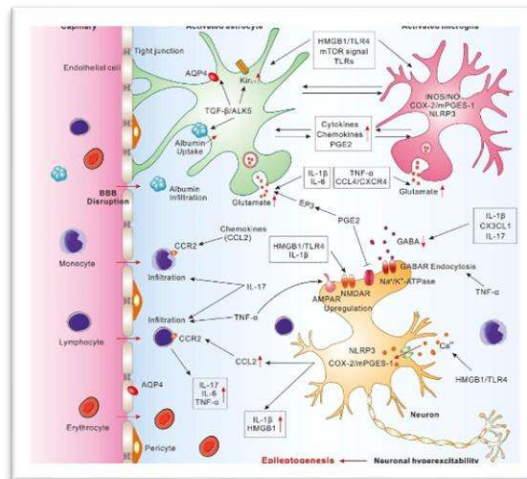


Image No.02: Quinolinic Acid-Induced Excitotoxicity

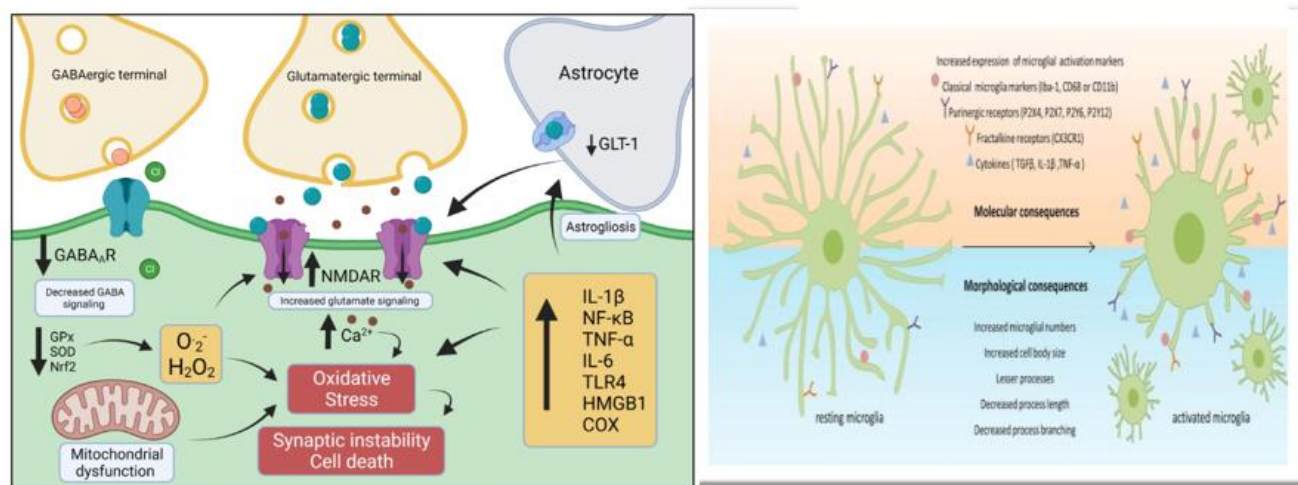


Image No.03: Oxidative Stress and Neuroinflammation in Epilepsy

Types of epilepsy:

A. Based on Seizure Onset

1. Focal Epilepsy (Partial Epilepsy)

- Focal aware seizures
- Focal impaired awareness seizures
- Focal to bilateral tonic-clonic seizures

2. Generalized Epilepsy

- Absence seizures
- Tonic-clonic seizures
- Myoclonic seizures
- Tonic seizures
- Clonic seizures
- Atonic seizures

3. Combined Generalized and Focal Epilepsy

- Dravet syndrome
- Lennox-Gastaut syndrome

4. Unknown Epilepsy

- Unclassified or undetermined seizure onset

B. Based on Etiology (Cause)

1. Structural epilepsy
2. Genetic epilepsy
3. Infectious epilepsy
4. Metabolic epilepsy
5. Immune-mediated epilepsy
6. Unknown etiology epilepsy

C. Clinically Important Types of Epilepsy

1. Temporal lobe epilepsy (TLE)
2. Frontal lobe epilepsy
3. Occipital lobe epilepsy
4. Parietal lobe epilepsy
5. Juvenile myoclonic epilepsy
6. Childhood absence epilepsy
7. Benign Rolandic epilepsy
8. Reflex epilepsy
9. Status epilepticus
10. Drug-resistant (refractory) epilepsy

Signs and Symptoms of Epilepsy

- Recurrent seizures (repeated episodes of abnormal brain activity)
- Loss of consciousness or awareness

- Uncontrolled jerking movements of the arms and legs (convulsions)
- Muscle stiffness or rigidity
- Staring spells (blank stare for a few seconds)
- Confusion during or after a seizure
- Temporary loss of responsiveness
- Sudden falls due to loss of muscle tone
- Tongue biting
- Loss of bladder or bowel control
- Memory problems
- Difficulty concentrating
- Fatigue and drowsiness after a seizure (postictal state)
- Headache following a seizure
- Behavioral, emotional, or mood changes such as anxiety or irritability

Models used in the treatment of epilepsy:

1. Maximal Electroshock Seizure (MES) Model

For testing antiepileptic medications against generalized tonic-clonic seizures, the MES model is among the most established and trustworthy experimental models. In this technique, a regulated electrical shock is applied through corneal or ear electrodes to Wistar rats in order to produce seizures. Tonic hind-limb extension, which is the main endpoint for evaluating anticonvulsant activity, is produced by the electric shock. Medications that stop or lessen hind-limb extension are thought to be useful in preventing generalized seizures. Since several therapeutically useful antiepileptic medications, such as carbamazepine and phenytoin, exhibit action in this test, the model is highly repeatable, easy to apply, and has great predictive validity. Compounds that limit the spread of seizures by blocking sodium channels or altering excitatory neurotransmission can be found using MES.

Advantages

- Simple and reproducible.
- Suitable for screening anticonvulsant drugs.
- Mimics generalized tonic-clonic seizures.

Limitation

- Does not reproduce chronic epilepsy or cognitive impairment.

2. Pentylentetrazole (PTZ) Model:

One of the most used models for chemically inducing seizures in Wistar rats is the PTZ model. PTZ, a GABA_A receptor antagonist, causes neuronal hyperexcitability and seizures by reducing inhibitory neurotransmission in the brain. Animals have myoclonic jerks, clonic convulsions, tonic seizures, and even status epilepticus after intraperitoneal injection. This model is very helpful for researching myoclonic epilepsy and absence seizures, as well as for assessing medications that increase GABAergic neurotransmission. The paradigm is ideal for anticonvulsant screening because PTZ-induced seizures have a quick onset and reliable behavioral symptoms. A kindling paradigm that simulates epileptogenesis may also be established by repeatedly administering subconvulsive doses of PTZ.

Advantages

- Easy to establish.
- Useful for evaluating GABA-mediated anticonvulsant activity.
- Suitable for acute and kindling studies.

Limitation

- Limited ability to mimic hippocampal neurodegeneration.



3. Kainic Acid (KA) Model:

One of the finest experimental models for temporal lobe epilepsy (TLE) is the Kainic Acid model. A strong glutamate receptor agonist, kainic acid causes excitotoxicity and excessive neuronal excitement. When KA is administered, it causes status epilepticus, which is followed by a latent phase and the emergence of spontaneous recurring seizures that resemble temporal lobe epilepsy in humans. Selective hippocampal neuronal loss, mossy fiber sprouting, gliosis, oxidative stress, neuroinflammation, and cognitive impairments are the hallmarks of the model. The KA model is often used to assess neuroprotective therapeutics, antioxidant treatments, anti-inflammatory substances, and medications that improve cognition because of these pathological characteristics. It is particularly pertinent to research on hippocampal injury and memory loss.

Advantages

- Closely mimics human temporal lobe epilepsy.
- Produces hippocampal neurodegeneration.
- Useful for studying cognitive impairment and neuroprotection.

Limitation

- Higher mortality rate than MES and PTZ.
- Requires careful monitoring of animals.

SUMMARY:

Recurrent seizures, cognitive decline, oxidative stress, neuroinflammation, and neuronal degeneration are all hallmarks of epilepsy, a long-term neurological condition. To study epileptogenesis and assess possible treatments, experimental models including Maximal Electroshock Seizure (MES), Pentylentetrazole

(PTZ), Kainic Acid (KA), and Quinolinic Acid (QA) are frequently utilized. These include KA and QA models, which closely resemble hippocampal injury and temporal lobe epilepsy. Significant neuroprotective, antioxidant, anti-inflammatory, anticonvulsant, and cognitive-enhancing properties have been shown by herbal extracts high in flavonoids, polyphenols, and alkaloids. These characteristics imply that herbal treatments may provide viable substitute methods for lessening neuronal damage brought on by seizures and enhancing cognitive results in epilepsy.

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