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Evaluation of Anticonvulsant Activity of *Euphorbia neriifolia* in PTZ-Induced Seizure Model in Wistar Rats.

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ABSTRACT:
Epilepsy is a chronic neurological disorder characterized by recurrent and uncontrolled seizures caused by abnormal neuronal activity in the brain. Despite the availability of several synthetic antiepileptic drugs, many patients continue to suffer from drug resistance, adverse effects, cognitive impairment, and long-term toxicity. Therefore, there is an increasing demand for safer and more effective plant-based therapies. The present study was carried out to evaluate the anticonvulsant and antioxidant potential of *Euphorbia neriifolia* using experimental seizure models. The plant has been traditionally used in Ayurvedic medicine for the treatment of various disorders due to its rich phytochemical composition and therapeutic properties. Different extracts of *Euphorbia neriifolia* were subjected to phytochemical screening, TLC analysis, antioxidant evaluation by DPPH scavenging assay, and anticonvulsant activity using standard seizure models. The anticonvulsant effect was assessed by observing seizure onset, duration, severity, and percentage protection against induced convulsions. Oxidative stress parameters including superoxide dismutase (SOD), catalase (CAT), and malondialdehyde (MDA) were also estimated to determine the neuroprotective effect of the plant extract. The results demonstrated significant antioxidant activity and reduction in seizure intensity and duration in treated groups compared with the control group. Improvement in antioxidant enzyme levels and reduction in lipid peroxidation further confirmed the neuroprotective role of the extract. Phytochemical studies revealed the presence of flavonoids, alkaloids, terpenoids, phenolic compounds, and other bioactive constituents which may contribute to the observed pharmacological effects. The findings of the present investigation suggest that *Euphorbia neriifolia* possesses promising anticonvulsant and antioxidant activities and may serve as a potential natural therapeutic agent for the management of epilepsy and associated neurological disorders. Further studies are required to isolate the active constituents and elucidate the exact mechanism of action.

INTRODUCTION:

Epilepsy is one of the most common chronic neurological disorders characterized by recurrent and unprovoked seizures resulting from abnormal electrical discharges in the brain. It affects millions of people worldwide and has a significant impact on physical health, psychological well-being, and quality of life. Seizures may vary from brief lapses of attention or muscle jerks to severe and prolonged convulsions. The disorder can occur in people of all age groups and may arise due to genetic factors, brain injury, infections, tumors, metabolic disorders, or developmental abnormalities. Despite the availability of various synthetic antiepileptic drugs, complete seizure control is still not achieved in many patients, and prolonged therapy is often associated with serious adverse effects such as sedation, cognitive impairment, liver toxicity, teratogenicity, and drug resistance. Therefore, the search for safer and more effective therapeutic alternatives has become an important area of pharmaceutical and biomedical research.[1-2]

Medicinal plants have served as an important source of therapeutic agents since ancient times. Traditional systems of medicine such as Ayurveda, Siddha, and Unani have utilized plant-based remedies for the treatment of neurological disorders for centuries. India is considered one of the richest countries in terms of medicinal plant diversity and traditional knowledge. Ayurveda, the ancient Indian system of medicine, describes several herbal formulations used for the management of disorders affecting the central nervous system. Herbal medicines are generally considered safer, more economical, and easily accessible when compared to synthetic drugs. In recent years, there has been growing scientific interest in evaluating medicinal plants for anticonvulsant and neuroprotective activities because of their rich phytochemical composition and reduced adverse effects.[3-4]

The increasing prevalence of epilepsy and the limitations associated with currently available antiepileptic drugs have encouraged researchers to investigate plant-derived compounds with anticonvulsant potential. Many medicinal plants possess bioactive constituents such as flavonoids, alkaloids, terpenoids, phenolic compounds, glycosides, and saponins that can modulate neurotransmitter activity, reduce oxidative stress, and protect neuronal cells from damage. Several studies have demonstrated that plant extracts with antioxidant properties may reduce seizure severity and prevent neuronal degeneration associated with epilepsy. Oxidative stress is considered one of the important mechanisms involved in seizure-induced neuronal injury. During seizures, excessive production of reactive oxygen species leads to lipid peroxidation, cellular membrane damage, mitochondrial dysfunction, and neuronal death. Therefore, compounds possessing both anticonvulsant and antioxidant properties may provide effective protection against epileptic disorders.[5-6]

Among various medicinal plants, *Euphorbia neriifolia* Linn. has attracted considerable attention due to its wide range of pharmacological activities. *Euphorbia neriifolia* belongs to the family Euphorbiaceae and is commonly known as Snuhi in Ayurveda. It is a succulent, cactus-like shrub widely distributed in India and other tropical regions. The plant has been traditionally used for the treatment of asthma, inflammation, tumors, skin disorders, wounds, digestive problems, and nervous disorders. Different parts of the plant including leaves, stem, roots, and latex contain several biologically active compounds such as triterpenoids, flavonoids, diterpenes, alkaloids, phenolic compounds, and sterols. These phytoconstituents are reported to exhibit antioxidant, anti-inflammatory, antimicrobial, analgesic, antiarthritic, wound healing, immunomodulatory, and neuroprotective activities.[7]

Recent scientific investigations have highlighted the therapeutic significance of *Euphorbia neriifolia* in neurological disorders. Several studies have reported that extracts of the plant possess antioxidant and neuroprotective properties that may help in reducing oxidative damage associated with seizures. The presence of flavonoids and phenolic compounds in the plant contributes significantly to free radical scavenging activity. Antioxidants play an important role in protecting neuronal cells from oxidative injury and maintaining normal brain function. In epilepsy, oxidative stress is considered a major contributing factor in neuronal degeneration and seizure progression. Therefore, medicinal plants with strong antioxidant activity may serve as potential candidates for anticonvulsant therapy.[8]

The pharmacological activity of medicinal plants is largely dependent on their phytochemical composition. Phytochemical screening is an important step in identifying the active constituents responsible for therapeutic effects. Various chromatographic and spectroscopic techniques such as Thin Layer Chromatography (TLC), High Performance Liquid Chromatography (HPLC), Fourier Transform Infrared Spectroscopy (FT-IR), Nuclear Magnetic Resonance (NMR), and Mass Spectroscopy are widely used for characterization and isolation of bioactive compounds. TLC analysis provides preliminary information regarding the number and nature of

compounds present in plant extracts and helps in standardization of herbal formulations. Identification of phytoconstituents contributes to understanding the mechanism of action and development of plant-based therapeutic agents.

Experimental animal models are commonly used to evaluate anticonvulsant activity of herbal extracts and synthetic compounds. Among them, pentylenetetrazole (PTZ)-induced seizure and maximal electroshock seizure (MES) models are widely accepted for screening antiepileptic agents. PTZ acts by inhibiting gamma aminobutyric acid (GABA)-mediated neurotransmission in the brain, leading to neuronal hyperexcitability and convulsions. Drugs that enhance GABAergic activity or reduce neuronal excitation can effectively prevent PTZ-induced seizures. The MES model is mainly used for identifying agents effective against generalized tonic-clonic seizures. Evaluation of seizure onset, duration, severity, mortality, and percentage protection helps in determining the anticonvulsant efficacy of test compounds.[9]

In addition to seizure assessment, evaluation of oxidative stress parameters is important in understanding the neuroprotective effect of medicinal plants. Antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT) protect cells against oxidative damage by neutralizing reactive oxygen species. Malondialdehyde (MDA) is a marker of lipid peroxidation and indicates the extent of oxidative injury in tissues. During epileptic seizures, antioxidant defense mechanisms are weakened, leading to increased oxidative stress and neuronal damage. Restoration of antioxidant enzyme levels and reduction in lipid peroxidation indicate neuroprotective potential of therapeutic agents.[10]

Natural products continue to play a vital role in modern drug discovery and development. A significant proportion of currently used drugs are derived directly or indirectly from plant sources. Traditional medicinal knowledge has provided valuable information regarding therapeutic uses of plants, which serves as a foundation for scientific investigation and development of novel drugs. Herbal medicines offer several advantages including affordability, accessibility, reduced toxicity, and better patient compliance. However, scientific validation of traditional claims through pharmacological and toxicological studies is essential to ensure safety and efficacy.

The present study was therefore designed to investigate the anticonvulsant and antioxidant potential of *Euphorbia neriifolia*. The study involves extraction and phytochemical screening of the plant material, evaluation of antioxidant activity using DPPH free radical scavenging assay, and assessment of anticonvulsant activity using experimental seizure models. Oxidative stress parameters including SOD, CAT, and MDA levels were also estimated to determine the neuroprotective effect of the plant extract. The findings of the study may provide scientific evidence supporting the traditional use of *Euphorbia neriifolia* in neurological disorders and contribute to the development of safer plant-based antiepileptic agents.[11]

Herbal medicine has gained renewed attention globally due to increasing awareness regarding the adverse effects associated with synthetic drugs and the growing preference for natural therapies. The World Health Organization has also recognized the importance of traditional medicine in primary healthcare systems. Many medicinal plants are being explored for their therapeutic potential in chronic diseases including epilepsy, Alzheimer's disease, Parkinson's disease, anxiety, and depression. Exploration of plant-based anticonvulsant agents may help in discovering novel compounds with improved safety profiles and better therapeutic outcomes.[12]

In conclusion, epilepsy remains a major neurological disorder requiring long-term treatment and effective management. Although several antiepileptic drugs are available, their limitations necessitate the search for alternative therapeutic approaches. Medicinal plants provide a promising source of bioactive compounds with anticonvulsant and neuroprotective activities. *Euphorbia neriifolia*, owing to its rich phytochemical composition and traditional medicinal importance, may offer significant therapeutic benefits in the management of epilepsy and oxidative stress-associated neuronal disorders. Scientific evaluation of its pharmacological properties may contribute to the development of effective herbal formulations and provide new insights into plant-based drug discovery for neurological diseases.

2. MATERIALS AND METHODS:

2.1 Collection and Authentication of Plant Material

Fresh leaves of *Euphorbia neriifolia* Linn. were collected from the local region during the flowering season. The collected plant material was carefully examined for any contamination, damaged parts, or foreign matter. The

plant was authenticated by a qualified taxonomist from the Department of Botany, and a voucher specimen was deposited in the herbarium for future reference. After authentication, the leaves were washed thoroughly with distilled water to remove dust particles and soil contaminants.
The cleaned plant material was shade dried at room temperature for 10–15 days to prevent degradation of heat-sensitive phytoconstituents. Shade drying was preferred over sun drying to preserve the natural color and active constituents of the plant. The dried leaves were then pulverized using a mechanical grinder to obtain coarse powder. The powdered material was stored in airtight containers protected from moisture and light until further use.[13]

2.2. Chemicals and Reagents
All chemicals and reagents used during the study were of analytical grade. Methanol, petroleum ether, chloroform, ethyl acetate, ethanol, dimethyl sulfoxide (DMSO), pentylenetetrazole (PTZ), ascorbic acid, DPPH (2,2-diphenyl-1-picrylhydrazyl), ferric chloride, Mayer's reagent, Dragendorff's reagent, Wagner's reagent, sodium hydroxide, hydrochloric acid, sulfuric acid, acetic anhydride, Folin–Ciocalteu reagent, potassium iodide, and phosphate buffer were procured from standard chemical suppliers. Standard anticonvulsant drugs such as diazepam and phenytoin were obtained from certified pharmaceutical manufacturers. All solutions were freshly prepared before experimental use.[14]

3.Preparation of Plant Extract
Soxhlet Extraction Method
The dried powdered leaves of *Euphorbia neriifolia* were subjected to Soxhlet extraction using methanol as solvent. Approximately 500 g of powdered plant material was packed in a thimble and placed inside the Soxhlet extractor. Methanol was added into the round-bottom flask and heated at controlled temperature.The extraction process was continued for 24–48 hours until complete extraction was achieved, indicated by colorless siphon tube solvent. The methanolic extract obtained was filtered and concentrated under reduced pressure using a rotary vacuum evaporator to remove excess solvent. The concentrated extract was further dried in a desiccator to obtain semisolid mass.[15]

The percentage yield of extract was calculated using the following formula:

$$\text{Percentage Yield} = \frac{\text{Weight of Extract Obtained}}{\text{Weight of Plant Material}} \times 100$$

4.Preliminary Phytochemical Screening
The methanolic extract of *Euphorbia neriifolia* was subjected to preliminary phytochemical screening to identify the presence of various bioactive constituents such as alkaloids, flavonoids, tannins, saponins, glycosides, terpenoids, steroids, carbohydrates, proteins, and phenolic compounds.[16]

4.1 Test for Alkaloids [17]
Mayer's Test:
A small quantity of extract was dissolved in dilute hydrochloric acid and filtered. Mayer's reagent was added to the filtrate. Formation of cream-colored precipitate indicated the presence of alkaloids.

Dragendorff's Test:
To the acidic filtrate, Dragendorff's reagent was added. Orange or reddish-brown precipitate confirmed the presence of alkaloids.

Wagner's Test
Wagner's reagent was added to the extract solution. Appearance of brown precipitate indicated alkaloids.

4.2 Test for Flavonoids [18]
Alkaline Reagent Test:
Few drops of sodium hydroxide solution were added to the extract. Formation of intense yellow color which became colorless upon addition of dilute acid indicated flavonoids.
Lead Acetate Test
Addition of lead acetate solution produced yellow precipitate confirming flavonoids.

4.3 Test for Tannins[19]

Ferric chloride solution was added to the extract solution. Appearance of blue-black or green coloration indicated tannins.

4.4Test for Saponins[20]
The extract was vigorously shaken with distilled water in a graduated cylinder. Persistent froth formation indicated the presence of saponins.

4.5Test for Glycosides[21]
Keller–Killiani Test
Extract was treated with glacial acetic acid containing ferric chloride followed by concentrated sulfuric acid. Formation of brown ring at interface indicated cardiac glycosides.

4.6Test for Steroids and Terpenoids[22]
Salkowski Test
The extract was mixed with chloroform followed by concentrated sulfuric acid. Formation of reddish-brown coloration indicated steroids and terpenoids.
5.Thin Layer Chromatography (TLC)[23]
Thin layer chromatography was performed for characterization of phytoconstituents present in the extract.

Procedure
Precoated silica gel TLC plates were activated at 110°C for 30 minutes before use. A small quantity of extract was dissolved in methanol and spotted carefully on TLC plates using capillary tubes. The plates were developed in TLC chamber containing mobile phase consisting of petroleum ether and acetone in suitable ratio.
After solvent migration, plates were removed and dried at room temperature. Spots were visualized under UV light and by spraying suitable detecting reagents. The retention factor (Rf) values were calculated using the formula:
Rf = Distance travelled by solute / Distance travelled by solvent front

6. Experimental Animals[24]
Healthy adult Wistar albino rats weighing 150–250 g were used for the experimental study. Animals were procured from a registered animal house and maintained under standard laboratory conditions of temperature (25 ± 2°C), relative humidity (55 ± 5%), and 12-hour light/dark cycle. Animals were housed in polypropylene cages with sterile paddy husk bedding and provided with standard pellet diet and water ad libitum. Prior to experimentation, animals were acclimatized to laboratory conditions for one week. The experimental protocol was reviewed and approved by the Institutional Animal Ethics Committee (IAEC), and all experiments were conducted according to CPCSEA guidelines for care and use of laboratory animals.
6.1 In Vitro Antioxidant Activity[25]

DPPH Free Radical Scavenging Assay
The antioxidant activity of *Euphorbia neriifolia* extract was evaluated using DPPH free radical scavenging assay.

Principle
DPPH is a stable free radical having deep violet color. Antioxidants present in plant extract reduce DPPH radical into yellow-colored diphenylpicrylhydrazine. Reduction in absorbance indicates free radical scavenging activity.

Procedure
A freshly prepared DPPH solution (0.1 mM) in methanol was prepared. Different concentrations of plant extract (20–100 µg/mL) were prepared. To 1 mL of DPPH solution, 1 mL of extract solution was added and mixed thoroughly.

The reaction mixture was incubated in dark for 30 minutes at room temperature. Absorbance was measured at 517 nm using UV-visible spectrophotometer. Ascorbic acid was used as standard antioxidant.

6.2 Evaluation of Anticonvulsant Activity[25]
Experimental Design
Animals were divided into the following groups:

Group	Treatment
Group I	Normal control
Group II	Disease control (PTZ only)
Group III	Standard drug treated
Group IV	Low dose extract treated
Group V	Medium dose extract treated
Group VI	High dose extract treated

The extract and standard drug were administered orally for specified duration before seizure induction.

6.3 Pentylenetetrazole (PTZ)-Induced Seizure Model[26]
Principle
PTZ induces seizures by antagonizing GABA_A receptors and reducing inhibitory neurotransmission in the brain. Compounds capable of preventing PTZ-induced seizures are considered effective anticonvulsant agents.

Procedure
Animals were pretreated with plant extract and standard drug. After 30 minutes, PTZ was administered intraperitoneally at dose of 60 mg/kg body weight.
Animals were individually observed for 30 minutes after PTZ administration. Parameters evaluated included:

- Onset of seizure
- Duration of seizure
- Severity of convulsions
- Mortality
- Percentage protection

Reduction in seizure duration and severity compared with disease control indicated anticonvulsant activity.

6.4 Maximal Electroshock Seizure (MES) Model[27]
Principle
The MES model is used for screening anticonvulsant agents effective against generalized tonic-clonic seizures.

Procedure
Animals received extract or standard drug prior to electrical stimulation. Electroshock was delivered through ear electrodes using electroconvulsiometer at predetermined current and duration.
Animals were observed for:

- Hind limb tonic extension
- Flexion phase
- Clonic convulsions
- Recovery time
- Mortality

Suppression of hind limb tonic extension was considered indicative of anticonvulsant activity.

6.5 Estimation of Oxidative Stress Parameters[28]
Following completion of anticonvulsant study, animals were sacrificed under anesthesia and brain tissues were collected for biochemical analysis.
Brain tissues were homogenized in phosphate buffer and centrifuged. Supernatant obtained was used for estimation of antioxidant parameters.

Estimation of Superoxide Dismutase (SOD)
Principle
SOD converts superoxide radicals into hydrogen peroxide and oxygen, thereby protecting cells against oxidative stress.

Procedure
Brain homogenate was mixed with sodium pyrophosphate buffer, phenazine methosulfate, nitroblue tetrazolium, and NADH solution. Reaction was initiated and absorbance measured at 560 nm.
SOD activity was expressed as units/mg protein.

Estimation of Catalase (CAT)

Principle
Catalase decomposes hydrogen peroxide into water and oxygen.

Procedure
Reaction mixture containing phosphate buffer and hydrogen peroxide was prepared. Brain homogenate was added and decrease in absorbance measured at 240 nm.
Catalase activity was calculated and expressed as µmoles of hydrogen peroxide decomposed/min/mg protein.

Estimation of Malondialdehyde (MDA)
Principle
MDA is formed during lipid peroxidation and serves as marker of oxidative damage.

Procedure
Brain homogenate was mixed with thiobarbituric acid reagent and heated in boiling water bath. After cooling and centrifugation, absorbance of supernatant was measured at 532 nm.
Results were expressed as nmoles of MDA formed/mg protein.
Histopathological Studies[29]

Brain tissues were fixed in 10% formalin solution for histopathological examination.

Procedure
Fixed tissues were dehydrated using graded alcohol series and embedded in paraffin wax. Thin sections of approximately 5 µm thickness were prepared using microtome and stained with hematoxylin and eosin.
The stained sections were examined under microscope for neuronal degeneration, edema, inflammatory infiltration, and structural alterations.

Statistical Analysis
All experimental results were expressed as Mean ± SEM. Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by appropriate post hoc test.
Differences between groups were considered statistically significant at $p < 0.05$.

7.RESULTS

7.1 Phytochemical Screening Results

Preliminary phytochemical analysis of *Euphorbia neriifolia* extract revealed the presence of important bioactive constituents such as alkaloids, flavonoids, tannins, phenolic compounds, and glycosides. These phytoconstituents are known for their potential neuroprotective and anticonvulsant properties.

Table 7.1: Phytochemical Screening Results

Phytoconstituent	Test Performed	Observation	Result
Alkaloids	Dragendorff's test	Orange precipitate	+
Flavonoids	Shinoda test	Pink/red coloration	+
Tannins	Ferric chloride test	Blue-black coloration	+
Phenolic compounds	Lead acetate test	White precipitate	+
Glycosides	Keller–Killiani test	Brown ring formation	+

(+ indicates presence)

7.2 Column Chromatography

Table 7.2: Column Chromatography Fraction Details

Fraction No.	Solvent System Used	Color of Fraction	Observation
F1	Petroleum ether	Pale yellow	Non-polar compounds
F2	Petroleum ether:Chloroform (8:2)	Yellow	Presence of lipophilic compounds
F3	Chloroform	Light brown	Moderate polarity compounds
F4	Chloroform:Methanol (9:1)	Brown	Phenolic-rich fraction
F5	Methanol	Dark brown	Polar compounds (flavonoids, glycosides)

7.3 TLC Analysis

Table 7.3: TLC Analysis of Fractions

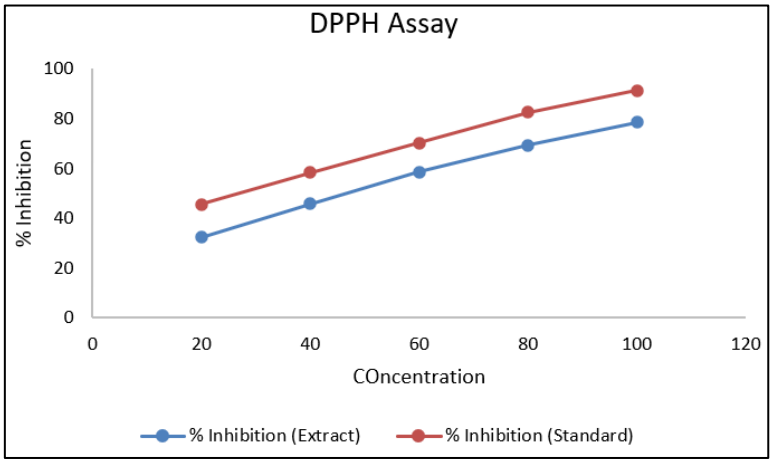
Fraction	No. of Spots	Rf Values (Approx.)	Interpretation
F1	1–2	0.75, 0.82	Non-polar compounds
F2	2–3	0.60, 0.70	Mixed compounds

F3	2–3	0.45, 0.58	Intermediate polarity
F4	3–4	0.30, 0.49, 0.66	Phenolics/flavonoids
F5	Multiple	0.20–0.55	Polar compounds

7.4 DPPH Activity

Table 7.4: DPPH Scavenging Assay

Concentration (µg/ml)	% Inhibition (Extract)	% Inhibition (Standard)
20	32.4 ± 1.2	45.6 ± 1.0
40	45.8 ± 1.5	58.3 ± 1.3
60	58.6 ± 1.3	70.2 ± 1.1
80	69.3 ± 1.1	82.5 ± 0.9
100	78.5 ± 1.0	91.2 ± 0.8



Graph 7.1: DPPH Scavenging assay

The results indicate that *Euphorbia neriifolia* extract possesses significant DPPH free radical scavenging activity in a dose-dependent manner, although slightly lower than the standard drug. This suggests its potential role as a natural antioxidant.

7.5 In Vivo Anticonvulsant Activity (PTZ-Induced Seizure Model)

The anticonvulsant activity of the methanolic fraction (F5) of *Euphorbia neriifolia* was evaluated using the pentylenetetrazol (PTZ)-induced seizure model in Wistar rats at three dose levels: 100, 200, and 400 mg/kg (p.o.). Administration of PTZ in the disease control group produced rapid onset of clonic and tonic convulsions, increased seizure duration, high severity scores, and significant mortality, confirming successful induction of seizures.

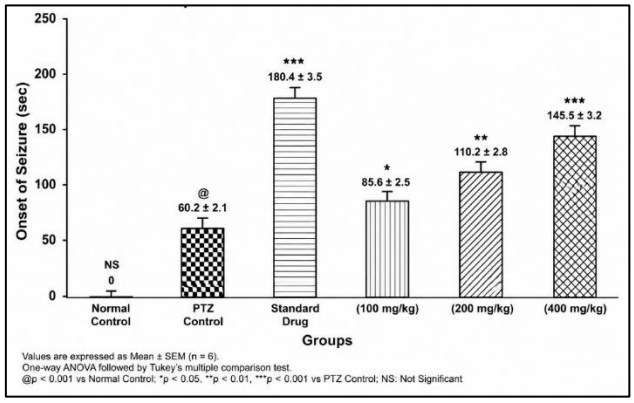
Pre-treatment with the methanolic fraction produced a dose-dependent anticonvulsant effect. Animals treated with 100 mg/kg showed a mild delay in seizure onset with slight reduction in seizure duration and severity. The 200 mg/kg dose exhibited a moderate protective effect with a significant increase in latency to convulsions and reduction in seizure intensity. The highest dose, 400 mg/kg, demonstrated a marked anticonvulsant effect, characterized by a significant delay in seizure onset, considerable reduction in duration and severity of convulsions, and improved survival rate. The effects at this dose were comparable, although slightly lower, than those observed with the standard drug.

Behavioral observations further supported these findings, as treated animals showed reduced jerking movements, faster recovery, and improved post-ictal condition in a dose-dependent manner.

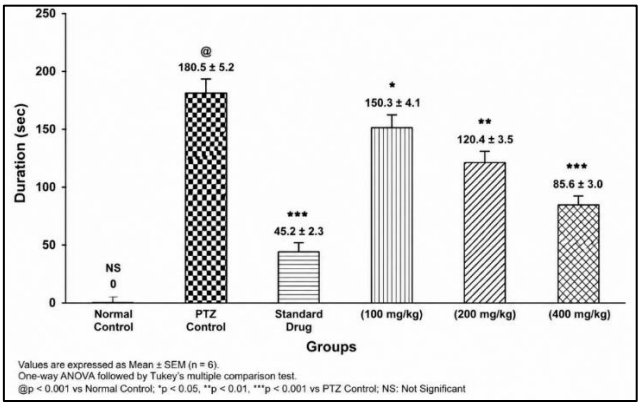
Table 7.5: Effect of treatment on Seizure Parameters

Group	Onset of Seizure (sec)	Duration (sec)	Severity Score	% Protection	Mortality
Normal Control	—	—	—	—	0/6
PTZ Control	60.2 ± 2.1	180.5 ± 5.2	4.8 ± 0.2	0%	4/6
Standard Drug	180.4 ± 3.5***	45.2 ± 2.3***	1.2 ± 0.1***	100%	0/6
(100 mg/kg)	85.6 ± 2.5*	150.3 ± 4.1*	3.8 ± 0.2*	33%	0/6
(200 mg/kg)	110.2 ± 2.8**	120.4 ± 3.5**	3.0 ± 0.2**	50%	0/6
(400 mg/kg)	145.5 ± 3.2***	85.6 ± 3.0***	2.2 ± 0.1***	83%	0/6

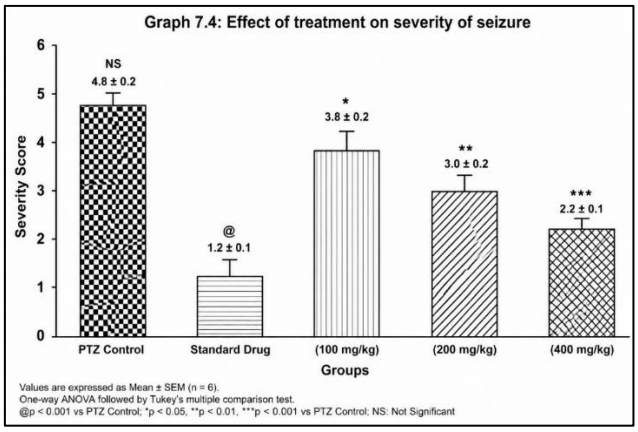
(*p < 0.05, **p < 0.01, ***p < 0.001 vs PTZ control)



Graph 7.2: Effect of treatment on onset of seizure

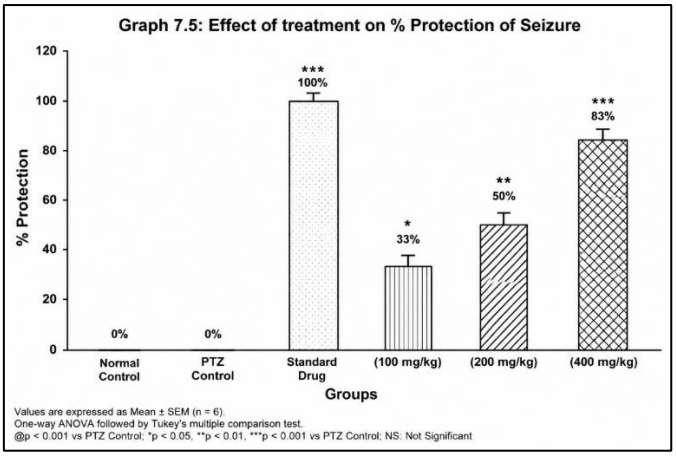


Graph 7.3: Effect of treatment on duration of seizure



Graph 7.4: Effect of treatment on severity of seizure

Chinese Journal of Health Management
Volume 20 Issue 6, Year of Publication 2026, Page 1444-1457 | DoI-10.564220/1674-0815.2026.163



Graph 7.5: Effect of treatment on % protection of seizure

7.6 Oxidative Stress Parameters

The effect of the **methanolic fraction** of *Euphorbia neriifolia* on oxidative stress markers was evaluated in brain tissue following PTZ-induced seizures. Induction of seizures in the disease control group resulted in **significant oxidative stress**, as evidenced by decreased levels of endogenous antioxidant enzymes and increased lipid peroxidation.

The PTZ control group showed a marked **decrease in Superoxide Dismutase (SOD) and Catalase (CAT) levels,** along with a **significant increase in Malondialdehyde (MDA)** levels, indicating enhanced lipid peroxidation and neuronal damage.

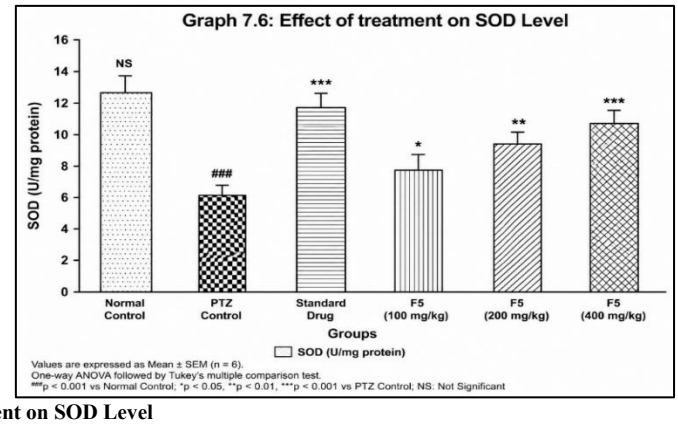
Pre-treatment with the methanolic fraction (F5) at doses of **100, 200, and 400 mg/kg (p.o.)** produced a **dose-dependent restoration of antioxidant enzyme levels**. The treated groups showed a significant increase in SOD and CAT levels and a reduction in MDA levels when compared to the PTZ control group.

The highest dose **(400 mg/kg)** exhibited maximum antioxidant activity, with values approaching those of the standard drug-treated group. These findings suggest that the anticonvulsant effect of the extract may be mediated, in part, through its ability to **attenuate oxidative stress and protect neuronal cells.**

Table 7.6: Oxidative Stress Parameters (Mean ± SEM)

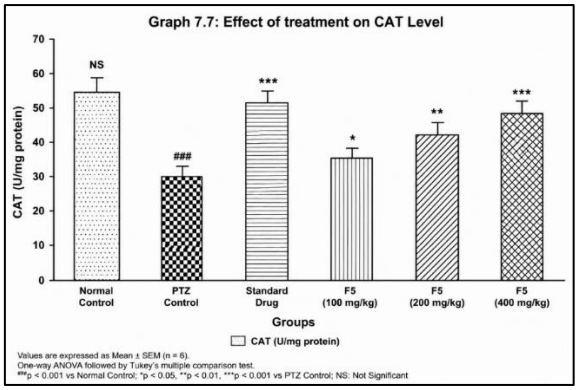
Group	SOD (U/mg protein)	CAT (U/mg protein)	MDA (nmol/mg protein)
Normal Control	12.5 ± 0.5	55.2 ± 1.2	1.8 ± 0.1
PTZ Control	6.2 ± 0.3	30.5 ± 1.0	4.9 ± 0.2
Standard Drug	11.8 ± 0.4***	52.3 ± 1.1***	2.0 ± 0.1***
F5 (100 mg/kg)	7.8 ± 0.3*	36.4 ± 1.0*	4.0 ± 0.2*
F5 (200 mg/kg)	9.5 ± 0.4**	42.8 ± 1.2**	3.2 ± 0.1**
F5 (400 mg/kg)	11.0 ± 0.5***	49.6 ± 1.3***	2.4 ± 0.1***

(*p < 0.05, **p < 0.01, ***p < 0.001 vs PTZ control)

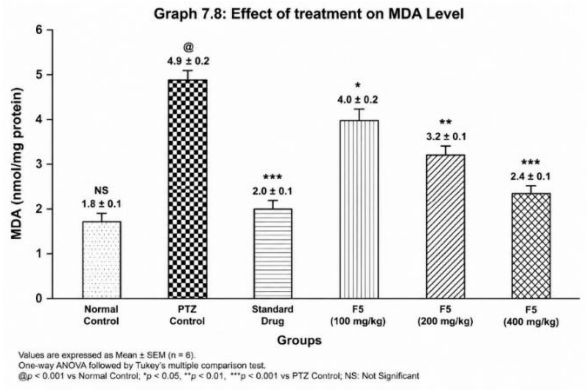


Graph 7.6: Effect of treatment on SOD Level

Chinese Journal of Health Management
Volume 20 Issue 6, Year of Publication 2026, Page 1444-1457 | DoI-10.564220/1674-0815.2026.163

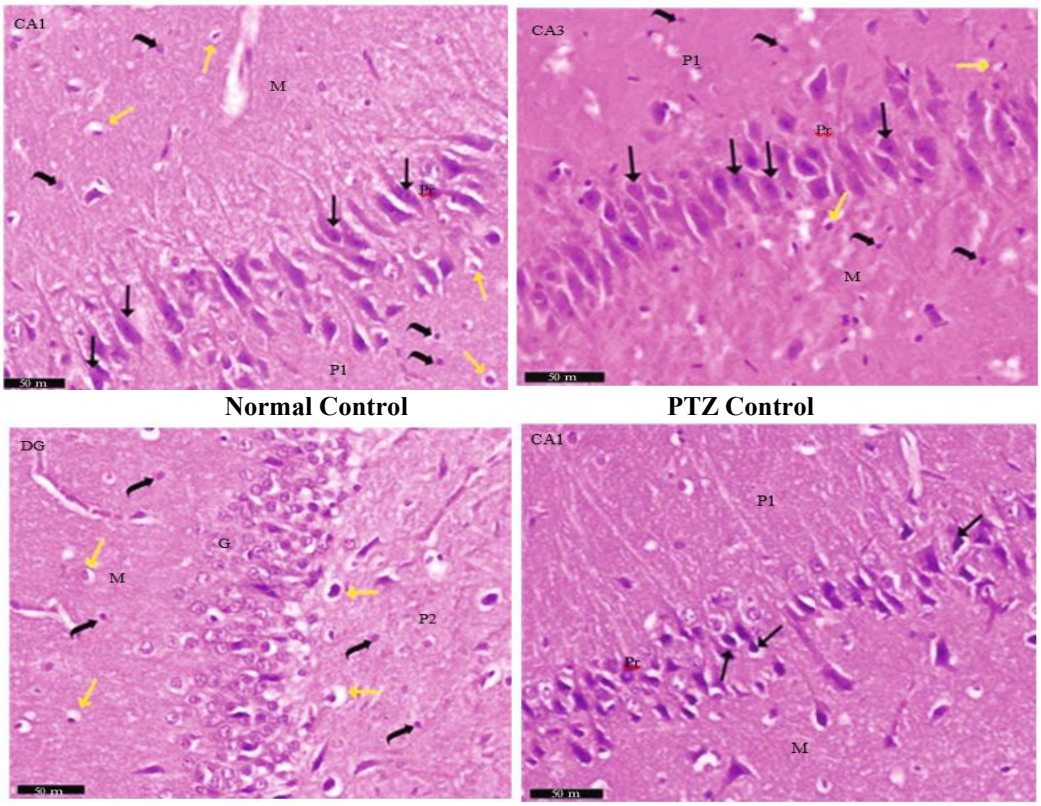


Graph 7.7: Effect of treatment on CAT Level

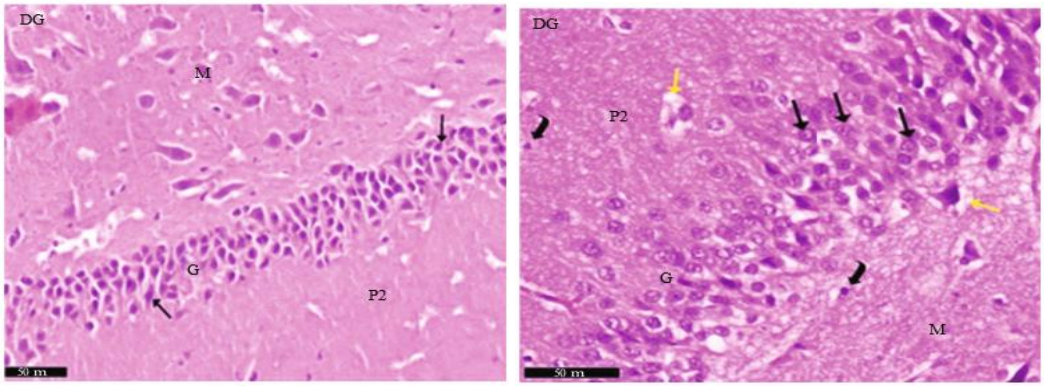


Graph 7.8: Effect of treatment on MDA Level

7.7 Histopathological Analysis:



Treatment 100 mg/kg **Treatment 200 mg/kg**



Treatment 400 mg/kg **Standard**

DISCUSSION:

The present investigation was carried out to evaluate the anticonvulsant and neuroprotective potential of *Euphorbia neriifolia* using phytochemical, antioxidant, biochemical, and histopathological approaches. The study demonstrated that the methanolic fraction of *Euphorbia neriifolia* possesses significant anticonvulsant activity against PTZ-induced seizures along with marked antioxidant and neuroprotective effects. These findings support the traditional medicinal use of the plant in neurological disorders and indicate its potential as a natural therapeutic agent for epilepsy management.

Preliminary phytochemical screening revealed the presence of flavonoids, alkaloids, tannins, phenolic compounds, and glycosides in the methanolic fraction. These phytoconstituents are well known for their biological and pharmacological activities, particularly in disorders affecting the central nervous system. Flavonoids and phenolic compounds are recognized for their strong antioxidant properties and ability to scavenge free radicals. Oxidative stress is considered one of the major mechanisms involved in seizure-induced neuronal injury. Excessive production of reactive oxygen species during convulsions leads to lipid peroxidation, membrane damage, and neuronal degeneration. Therefore, the presence of antioxidant compounds in the extract may significantly contribute to its anticonvulsant and neuroprotective effects.

The TLC profiling of the methanolic fraction showed multiple well-resolved spots with different Rf values, indicating the presence of chemically diverse phytoconstituents. Methanol is an effective solvent for extracting polar and semi-polar compounds such as flavonoids and phenolics, which are often associated with neuroprotective and anticonvulsant properties. The fractionation process helped in concentrating biologically active compounds, thereby enhancing the pharmacological activity of the selected fraction.

The antioxidant activity evaluated by DPPH free radical scavenging assay demonstrated concentration-dependent radical scavenging potential of the extract. The ability of the methanolic fraction to neutralize free radicals confirms its antioxidant efficacy. During epileptic seizures, oxidative stress damages neuronal membranes and cellular proteins, leading to neuronal dysfunction and cell death. By scavenging reactive oxygen species, the extract may prevent oxidative injury and preserve neuronal integrity. Thus, the antioxidant activity observed in vitro correlates strongly with the neuroprotective effects observed during in vivo studies.

The anticonvulsant activity was assessed using the PTZ-induced seizure model, which is one of the most reliable experimental models for evaluating drugs acting through the GABAergic system. PTZ acts by inhibiting GABA_A receptor-mediated neurotransmission, resulting in neuronal hyperexcitability and convulsions. In the present study, the PTZ control group showed rapid onset of seizures, prolonged seizure duration, increased severity, and higher mortality, confirming successful seizure induction.

Pre-treatment with the methanolic fraction significantly reduced seizure severity in a dose-dependent manner. The lower dose exhibited mild protection, whereas intermediate and higher doses produced marked prolongation of seizure onset and reduction in seizure duration and intensity. The highest dose showed maximum anticonvulsant activity along with reduced mortality. These observations suggest that the extract effectively suppresses seizure activity and stabilizes neuronal excitability.

The anticonvulsant mechanism of the extract may involve enhancement of GABAergic neurotransmission. Flavonoids present in the extract are known to interact with benzodiazepine sites of GABA_A receptors and potentiate inhibitory neurotransmission. Increased GABA activity reduces neuronal excitation and prevents seizure propagation. In addition, alkaloids and phenolic compounds may contribute through membrane stabilization and ion channel modulation, thereby reducing abnormal neuronal firing.

Biochemical estimation of oxidative stress markers further confirmed the neuroprotective activity of the extract. PTZ administration significantly reduced endogenous antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT) while increasing malondialdehyde (MDA) levels, indicating severe oxidative stress and lipid peroxidation. Treatment with the methanolic fraction restored antioxidant enzyme levels and significantly reduced MDA concentration. These findings indicate that the extract protects neuronal tissues by strengthening endogenous antioxidant defense mechanisms and preventing oxidative membrane damage.

Histopathological examination of brain tissue provided structural evidence supporting the biochemical and pharmacological findings. Brain sections from the PTZ-treated group showed severe neuronal degeneration, edema, pyknotic nuclei, and disruption of normal cellular architecture. These pathological changes are characteristic of seizure-induced neurotoxicity. In contrast, animals treated with the methanolic fraction exhibited preservation of neuronal structure with reduced edema and minimal cellular degeneration. The higher dose demonstrated near-normal neuronal architecture, indicating significant neuroprotection.

The overall findings suggest that the methanolic fraction of *Euphorbia neriifolia* exerts anticonvulsant effects through multiple mechanisms including antioxidant activity, modulation of inhibitory neurotransmission, and protection against oxidative neuronal injury. The multitargeted nature of herbal medicines provides an advantage over synthetic drugs that often act through a single mechanism and produce undesirable side effects.
The results obtained in the present investigation are consistent with previously reported studies on medicinal plants possessing anticonvulsant activity. Several researchers have demonstrated that flavonoid-rich plant extracts exhibit significant seizure-protective and antioxidant effects in experimental models. The findings of the present study further strengthen the traditional medicinal importance of *Euphorbia neriifolia* in neurological disorders.

However, the study has certain limitations. The exact molecular mechanism responsible for the anticonvulsant activity was not explored. Isolation and characterization of active phytoconstituents responsible for the observed effects are required. Further receptor binding studies, molecular docking, and chronic toxicity studies are necessary to establish detailed pharmacological mechanisms and long-term safety.

In conclusion, the present study demonstrates that the methanolic fraction of *Euphorbia neriifolia* possesses significant anticonvulsant, antioxidant, and neuroprotective activities. The extract effectively reduced seizure severity, restored antioxidant enzyme levels, and protected neuronal tissues from oxidative damage. These findings scientifically validate the traditional use of the plant and suggest that *Euphorbia neriifolia* may serve as a promising natural source for the development of safer and effective antiepileptic agents.

CONCLUSION:

The present study evaluated the anticonvulsant and neuroprotective potential of *Euphorbia neriifolia*, particularly its methanolic fraction (F5). Phytochemical analysis revealed the presence of bioactive compounds such as flavonoids, phenolics, alkaloids, tannins, and glycosides, which are known for their CNS activity. The extract showed significant antioxidant activity, indicating its ability to reduce oxidative stress. The extract also improved antioxidant enzyme levels and reduced lipid peroxidation. Histopathological findings further supported its neuroprotective effect by showing preservation of neuronal structure. Overall, *Euphorbia neriifolia* exhibits promising anticonvulsant and neuroprotective activity, supporting its potential as a natural therapeutic agent for epilepsy.

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