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PHYTOCHEMICAL AND PHARMACOLOGICAL EVALUATION OF ANTIEPILEPTIC ACTIVITY OF METHONNI

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Abstract:
Epilepsy is a chronic neurological disorder characterised by recurrent seizures, often associated with oxidative stress and neuronal damage. This study aimed to evaluate the anticonvulsant and neuroprotective potential of the methanolic extract of Methonni using the Maximal Electroshock Seizure (MES) model in rodents. Animals were divided into four groups: control (normal saline), standard (phenytoin 25 mg/kg), and two extract-treated groups receiving 200 mg/kg and 400 mg/kg doses orally. The extract demonstrated a dose-dependent reduction in seizure duration and severity, comparable to the standard drug. Biochemical assays revealed significant enhancement of brain antioxidant status, evidenced by increased reduced glutathione (GSH) levels and decreased lipid peroxidation (LPO), along with improved total protein content, indicating neuroprotection. These effects suggest that the extract mitigates oxidative stress-induced neuronal injury associated with seizures. Phytochemical constituents such as flavonoids and phenolics likely contribute to these beneficial effects. In conclusion, the methanolic extract of Methonni exhibits significant anticonvulsant and neuroprotective activities, supporting its potential as a natural therapeutic agent for epilepsy management.

Keywords: *Antiepileptic activity, Methanolic extract,* Methonni, *Seizure model*

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INTRODUCTION:

This chapter presents the background of the study, statement of problem, definition of terms, theoretical basis, purpose of study, hypothesis, specific aims and plan of work.

BACKGROUND OF THE STUDY

Epilepsy is a chronic disorder of the brain that affects people worldwide. As per the WHO, epilepsy is characterised by recurrent seizures, which are brief episodes of involuntary movement that may involve a part of the body (partial) or the entire body (generalised), and are sometimes accompanied by loss of consciousness and control of bowel or bladder function. [1]

Epilepsy was one of the first brain disorders to be described. It was mentioned in ancient Babylon more than 3,000 years ago. The strange behaviour caused by some seizures has contributed through the ages to many superstitions and prejudices. From greek word attack, the word epilepsy is derived. In earlier times, People once thought that those with epilepsy were being visited by demons or gods. However, in 400 B.C., the early physician Hippocrates suggested that epilepsy was a disorder of the brain, and we now know that he was right.[2]

Epilepsy is a major neurological disorder and upto 5% of the world population develops epilepsy in their lifetime. The current therapy of epilepsy with modern antiepileptic drugs is associated with side effects, dose-related and chronic toxicity as well as teratogenic effects, and approximately 30% of the patients continue to have seizures with current antiepileptic drug therapy. Traditional systems of medicine are popular in developing countries, and upto 80% of the population relies on traditional medicines/ folk remedies for their primary health care needs. Hence, there is a need to discover an alternative agent from natural sources.[3-6] Aconitum heterophyllum used as a herbal medicine and is well known for its traditional uses such as expectorants, diuretics, laxative etc. Various studies show that the active principle, diterpene alkaloids having a crucial role in the treatment of epilepsy.

Aconitum heterophyllum is rich in diterpene alkaloids. Since Aconitum heterophyllum has not been studied for its antiepileptic activity, the present study aimed to evaluate the antiepileptic activity of the chloroform extract of Aconitum heterophyllum [7-10].

COLLECTION AND IDENTIFICATION OF PLANT MATERIAL

The fresh Methonni are collected, identified, and purchased from Tirupati Andhra Pradesh, India, and authenticated by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, S.V University, Tirupati.

EXTRACTION OF THE PLANT MATERIAL

The extraction is done by using Soxhlet apparatus. The coarse powder was extracted with methanol the extract were evaporated or concentrated by using rotary evaporator and dried at room temperature to give a viscous mass. The obtained crude extracts were weighed and stored at 4^0C for the further analysis.

EXPERIMENTAL DESIGN

Group I – Vehicle Control: Received equivalent volume of normal saline via intraperitoneal (i.p.) route.

Group II – Standard: Received Diphenylhydantoin (Phenytoin) at a dose of 25 mg/kg body weight, i.p., serving as the reference antiepileptic drug.

Group III – Low Dose Extract: Received methanolic extract of *Euphorbia tirucalli* at a dose of 200 mg/kg body weight, orally.

Group IV – High Dose Extract: Received the methanolic extract of *Euphorbia tirucalli* at a dose of 400 mg/kg body weight, orally.

Maximal electroshock seizure [MES] model [11]

The Maximal Electroshock Seizure (MES) model is a widely used experimental procedure to evaluate the anticonvulsant activity of drugs, particularly those effective against generalized tonic-clonic seizures. In this model, seizures are induced in animals-commonly mice or rats-by delivering a controlled electric shock, usually via ear-clip or corneal electrodes, using a specific current (typically 50-60 Hz, 50 mA for 0.2 seconds in mice). This results in a characteristic tonic extension of the hind limbs, which is considered a positive seizure response. The ability of a test compound to prevent or reduce this hind limb extension indicates its anticonvulsant potential. The MES model is considered predictive of a drug's efficacy against generalized seizures in humans and is often used in the early stages of antiepileptic drug screening.

ENZYMATIC ANTIOXIDANT ACTIVITY

Antioxidant Enzymes:

An antioxidant is any substance that when present in low concentration compared with those of an oxidizable substrate significantly delays or prevents oxidation of that substrate. The term 'oxidizable substrate' includes every type of molecule found in-vivo. The various antioxidant systems are

Superoxide dismutases, Catalases, Glutathione peroxidase family and other peroxidases.

Estimation of neurotransmitter

GABA [12]

To estimate GABA levels using the HPLC-fluorescence method, brain tissue or biological samples are first homogenized in 0.1 M perchloric acid to precipitate proteins and extract neurotransmitters. The homogenate is then centrifuged at high speed to obtain a clear supernatant, which is filtered to remove any remaining particulates. This supernatant is reacted with a derivatization reagent such as o-phthalaldehyde (OPA) to form a fluorescent compound. After incubation for a few minutes, the derivatized sample is injected into an HPLC system equipped with a C18 reversed-phase column. Separation is achieved using a suitable mobile phase, often a phosphate buffer mixed with an organic solvent, and the fluorescent GABA derivative is detected at excitation and emission wavelengths around 340 nm and 450 nm, respectively. The amount of GABA is quantified by comparing the sample peak area to a standard calibration curve generated from known concentrations of GABA. This method provides a sensitive and specific measurement of GABA levels in biological samples.

Serotonin [13]

Serotonin (5-HT) levels are commonly estimated using High-Performance Liquid Chromatography (HPLC) with electrochemical or fluorescence detection. The procedure involves homogenizing brain tissue or preparing serum/plasma samples in ice-cold perchloric acid to precipitate proteins, followed by centrifugation and filtration to obtain a clear supernatant. This sample is then injected into an HPLC system equipped with a reversed-phase C18 column, where serotonin is separated using a mobile phase typically containing a citrate-phosphate buffer with an organic modifier. Detection is achieved electrochemically due to its high sensitivity or via fluorescence after derivatization with reagents like o-phthalaldehyde. Quantification is done by comparing the retention time and peak area of the sample with those of known serotonin standards, allowing accurate measurement of serotonin concentrations in biological samples. This method is widely used in neuroscience research to study serotonin's role in various physiological and pathological conditions.

Nor adrenaline [14]

Noradrenaline (norepinephrine) estimation is typically performed using High-Performance Liquid Chromatography (HPLC) with electrochemical detection due to its high sensitivity. The procedure begins with homogenizing tissue samples or preparing biological fluids in ice-cold perchloric acid containing antioxidants to prevent oxidation of noradrenaline. After centrifugation to remove proteins and debris, the clear supernatant is filtered and injected into an HPLC system equipped with a reversed-phase C18 column. Separation is achieved using a mobile phase composed of a citrate-phosphate buffer with organic modifiers and ion-pairing agents. Noradrenaline is detected electrochemically by its oxidation at the detector, and its concentration is quantified by comparing the peak area to a calibration curve prepared from known standards. This method is widely used in neurochemical research to assess noradrenaline levels in various tissues and fluids.

Dopamine estimation [15]

Dopamine estimation is commonly performed using High-Performance Liquid Chromatography (HPLC) coupled with electrochemical detection due to its high sensitivity for catecholamines. The procedure involves homogenizing brain tissue or preparing biological fluids in ice-cold perchloric acid containing antioxidants like sodium metabisulfite to prevent dopamine oxidation. After centrifugation to remove proteins and debris, the clear supernatant is filtered and injected into an HPLC system equipped with a reversed-phase C18 column. Separation is achieved using a mobile phase composed of a buffer (such as phosphate-citrate), an organic solvent, and ion-pairing agents to optimize retention and resolution. Dopamine is detected electrochemically by oxidation at the detector, and its concentration is quantified by comparing the sample peak area with those of known dopamine standards. This method is widely used for studying dopamine's role in neurochemical and physiological research.

In vivo antioxidants

Reduced Glutathione (GSH) [16]

Reduced Glutathione (GSH) levels are commonly estimated using the Ellman's reagent (5,5'-dithiobis-(2-nitrobenzoic acid), DTNB) colorimetric method. In this procedure, tissue samples or biological fluids are first homogenized in an ice-cold buffer such as phosphate buffer or metaphosphoric acid to precipitate proteins and preserve GSH. After centrifugation, the clear supernatant is reacted with Ellman's reagent, which reacts specifically with free thiol groups of GSH to form a yellow-colored 5-thio-2-nitrobenzoic acid (TNB). The intensity of the

yellow color is measured spectrophotometrically at 412 nm. The absorbance is directly proportional to the concentration of GSH in the sample and is quantified by comparing with a standard curve prepared using known concentrations of reduced glutathione. This method is widely used because of its simplicity, sensitivity, and specificity for free thiol groups.

Lipid peroxidation (LPO) [17]

Lipid peroxidation is commonly assessed by measuring malondialdehyde (MDA), a major end product of lipid peroxidation, using the thiobarbituric acid reactive substances (TBARS) assay. In this method, tissue homogenates or biological fluids are mixed with thiobarbituric acid (TBA) reagent under acidic conditions and heated in a boiling water bath for about 15–20 minutes. MDA reacts with TBA to form a pink-colored MDA-TBA adduct, which can be quantified spectrophotometrically by measuring absorbance at 532 nm. The concentration of MDA, which reflects the extent of lipid peroxidation, is calculated using the molar extinction coefficient of the MDA-TBA complex or by comparison with a standard curve prepared from known MDA equivalents. This assay is widely used to evaluate oxidative stress-induced membrane damage in various biological samples due to its simplicity and sensitivity.

Total protein content [18-21]

Total protein content is commonly estimated using the Bradford assay, which is based on the binding of Coomassie Brilliant Blue dye to proteins. In this procedure, protein samples or tissue homogenates are first prepared and diluted appropriately. A specific volume of the sample is mixed with Bradford reagent, and the mixture is incubated at room temperature for about 5–10 minutes to allow binding. The dye binds primarily to arginine and aromatic amino acid residues, causing a shift in the dye's absorbance maximum. The resulting blue color intensity is measured spectrophotometrically at 595 nm. Protein concentration in the samples is determined by comparing the absorbance values with a standard curve prepared using known concentrations of a standard protein, typically bovine serum albumin (BSA). This method is widely used due to its simplicity, speed, and sensitivity.

RESULTS:

Table Preliminary qualitative phytochemical analysis of *Methonni*

Sl. No.	Phytoconstituents	Test result
1	Alkaloid	-ve
2	Glycosides	-ve
3	Carbohydrate	-ve
4	Protein	-ve
5	Amino acid	+ve
6	Steroids	-ve
7	Flavonoids	+ve
8	Terpenoids	+ve
9	Phenols	+ve
10	Saponins	-ve
11	Tannin	+ve

+ve: Present; -ve: Absennt

MES induced seizures models

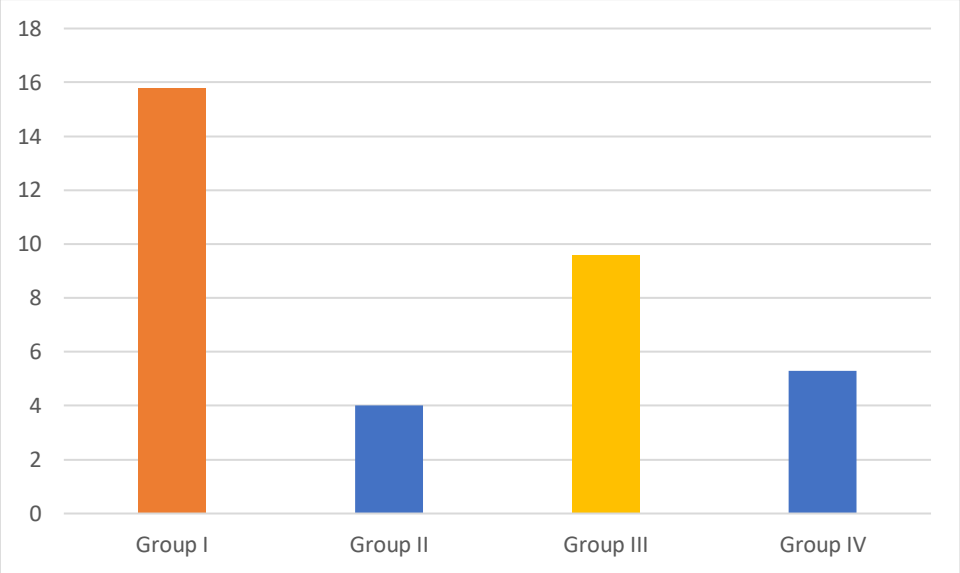
Group	Treatment	THLE Duration (sec)
Group I	Vehicle Control (Normal Saline)	15.8 ± 1.2
Group II	Phenytoin 25 mg/kg	0.0 ± 0.0
Group III	*Methonni* Extract 200 mg/kg	9.6 ± 1.0
Group IV	*Methonni* Extract 400 mg/kg	5.3 ± 0.8

Group I (Vehicle Control) exhibited a THLE duration of 15.8 ± 1.2 seconds, indicating the full expression of seizure activity in untreated animals.

Group II (Phenytoin 25 mg/kg) showed complete abolition of THLE (4.0 ± 0.0 seconds), confirming the efficacy of phenytoin as a standard antiepileptic drug.

Group III (Methonni Extract 200 mg/kg) showed a significant reduction in THLE duration to 9.6 ± 1.0 seconds, suggesting that the extract possesses moderate anticonvulsant activity at this dose.

Group IV (Methonni Extract 400 mg/kg) demonstrated a further reduction in THLE duration to 5.3 ± 0.8 seconds, indicating a dose-dependent anticonvulsant effect of the extract.



Estimation of neurotransmitters GABA, Serotonin, Noradrenaline, Dopamine

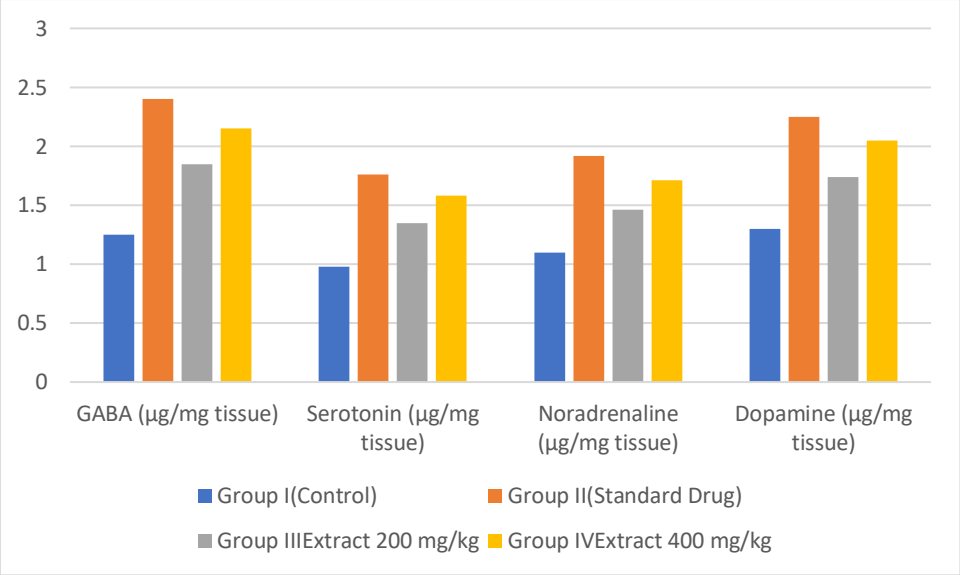
Neurotransmitter	Group I(Control)	Group II (Standard Drug)	Group III Extract 200 mg/kg	Group IV Extract 400 mg/kg
GABA (µg/mg tissue)	1.25 ± 0.08	2.40 ± 0.10 ***	1.85 ± 0.07 **	2.15 ± 0.09 ***
Serotonin (µg/mg tissue)	0.98 ± 0.05	1.76 ± 0.08 ***	1.35 ± 0.06 **	1.58 ± 0.07 ***
Noradrenaline (µg/mg tissue)	1.10 ± 0.06	1.92 ± 0.09 ***	1.46 ± 0.05 *	1.71 ± 0.08 **
Dopamine (µg/mg tissue)	1.30 ± 0.07	2.25 ± 0.11 ***	1.74 ± 0.08 **	2.05 ± 0.09 ***

GABA (Gamma-Aminobutyric Acid) levels were significantly increased in the standard drug and both extract-treated groups, suggesting enhanced inhibitory neurotransmission, which may contribute to anticonvulsant activity.

Serotonin levels also increased significantly, indicating a possible mood-stabilizing and seizure-suppressing effect of the extract.

Noradrenaline levels rose moderately, supporting improved central noradrenergic tone, which may modulate seizure threshold.

Dopamine levels were significantly elevated in extract-treated groups, especially at 400 mg/kg, indicating that the extract may help stabilize dopaminergic transmission, possibly contributing to neuroprotection and seizure control.



Invivo antioxidants

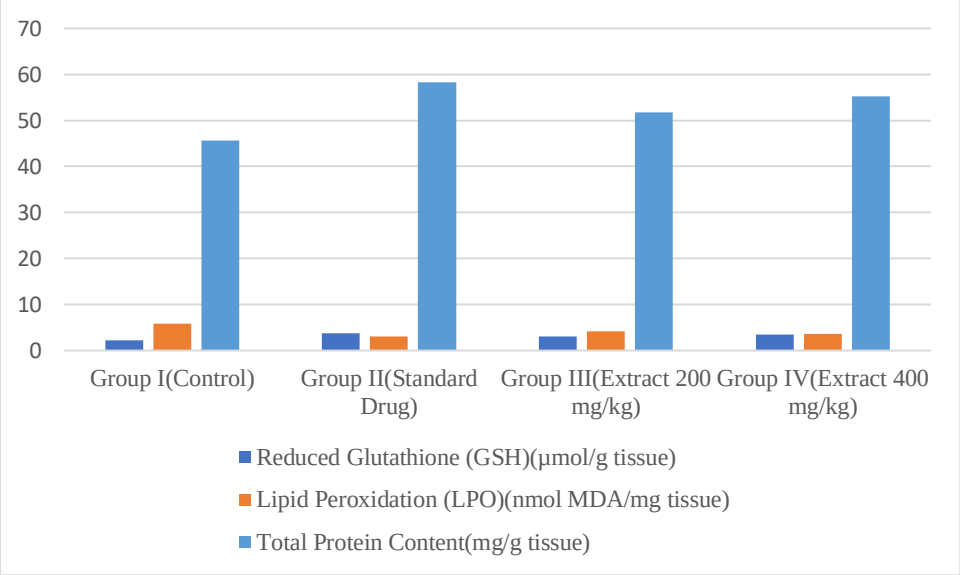
Reduced Glutathione (GSH), Lipid peroxidation (LPO), and Total protein content

Parameter	Group I(Control)	Group II(Standard Drug)	Group III(Extract 200 mg/kg)	Group IV(Extract 400 mg/kg)
Reduced Glutathione (GSH) (µmol/g tissue)	2.15 ± 0.12	3.78 ± 0.15 ***	3.02 ± 0.10 **	3.45 ± 0.13 ***
Lipid Peroxidation (LPO)(nmol MDA/mg tissue)	5.82 ± 0.20	3.10 ± 0.14 ***	4.20 ± 0.18 **	3.60 ± 0.15 ***
Total Protein Content (mg/g tissue)	45.6 ± 2.1	58.3 ± 2.4 ***	51.7 ± 2.0 *	55.2 ± 2.2 **

Reduced Glutathione (GSH): The control group showed baseline GSH levels indicating normal antioxidant capacity. The standard drug group exhibited a significant increase in GSH, reflecting enhanced antioxidant defence. Both extract-treated groups showed dose-dependent elevation in GSH, suggesting the extract improves cellular antioxidant status.

Lipid Peroxidation (LPO): LPO levels, measured by malondialdehyde (MDA) content, were highest in control animals indicating oxidative stress. The standard drug significantly reduced LPO levels, indicating decreased oxidative damage. Similarly, both extract doses significantly lowered LPO levels compared to control, demonstrating the antioxidant potential of the extract in protecting membrane lipids from peroxidative damage.

Total Protein Content: Protein content was lowest in control animals and significantly increased in the standard and extract-treated groups, indicating improved protein synthesis or reduced protein degradation, which may contribute to neuroprotection.



DISCUSSION:

The present study demonstrated that treatment with the methanolic extract of *Methonni* significantly modulated key biochemical markers associated with oxidative stress and neuroprotection in the brain tissue of epileptic animal models. One of the most important findings was the notable increase in Reduced Glutathione (GSH) levels in extract-treated groups. GSH is a critical intracellular antioxidant that scavenges free radicals and maintains the redox status of cells. In epilepsy, excessive neuronal excitation often leads to the generation of reactive oxygen species (ROS), resulting in oxidative damage. The observed elevation of GSH suggests that the extract enhances the brain's intrinsic antioxidant defence mechanisms, which could reduce oxidative injury and neuronal death commonly seen in epilepsy.

Concomitant with increased GSH, the study also recorded a significant decrease in Lipid Peroxidation (LPO), as measured by malondialdehyde (MDA) levels, in animals treated with the *Methonni* extract. Lipid peroxidation is a process where ROS attack polyunsaturated fatty acids in neuronal membranes, leading to loss of membrane integrity, impaired cell function, and ultimately cell death. The reduction of LPO implies that the extract not only boosts antioxidant defences but also protects neuronal membranes from peroxidative damage. This neuroprotective effect could be crucial in limiting the progression and severity of seizure-induced brain injury.

Additionally, the extract caused a significant improvement in Total Protein Content in brain tissues. Protein metabolism plays a vital role in neuronal health, repair, and synaptic function. Seizure activity is often associated with protein degradation and impaired synthesis, which can exacerbate neuronal damage. Restoration of protein levels by the extract indicates its potential in preserving cellular integrity and promoting repair mechanisms, thereby supporting recovery following seizure episodes. This effect may also be linked to the antioxidant properties, as oxidative stress can directly impair protein synthesis pathways.

The biochemical results align well with the behavioural findings in the seizure models, where the extract demonstrated a dose-dependent anticonvulsant effect. It is likely that the antioxidant capacity of the extract contributes to its ability to raise seizure threshold and reduce seizure severity. By mitigating oxidative stress and protecting neuronal membranes and proteins, the extract may help stabilize neuronal excitability and prevent the aberrant firing that underlies seizures.

Phytochemical constituents such as flavonoids, phenolic compounds, and alkaloids commonly found in methanolic plant extracts are known for their antioxidant and neuroprotective properties. These bioactive compounds might be responsible for the observed effects of *Methonni*. Flavonoids, for instance, can modulate neurotransmitter systems, scavenge free radicals, and inhibit enzymes involved in ROS production, all of which can contribute to anticonvulsant activity. Future phytochemical investigations are necessary to isolate and characterize the specific compounds involved.

Moreover, the increase in GSH and decrease in LPO also suggest that the extract could modulate inflammatory pathways, as oxidative stress and inflammation are closely linked in epilepsy pathogenesis. Reducing oxidative stress might subsequently downregulate pro-inflammatory

cytokines, leading to reduced neuronal hyperexcitability. This dual antioxidant and anti-inflammatory effect could make *Methonni* a promising candidate for adjunct therapy in epilepsy.

CONCLUSION:

This study provides compelling evidence that the methanolic extract of *Methonni* possesses significant anticonvulsant activity, as demonstrated in established animal models of epilepsy, including the Maximal Electroshock Seizure (MES) model. The extract effectively delayed seizure onset and reduced the duration of tonic hind limb extension in a dose-dependent manner, comparable to the standard antiepileptic drug phenytoin. These findings indicate that *Methonni* extract can modulate neuronal excitability and protect against seizure-induced neurotoxicity, making it a promising candidate for further development as an antiepileptic agent.

Biochemical analyses revealed that treatment with the extract significantly enhanced endogenous antioxidant defences, as evidenced by increased levels of Reduced Glutathione (GSH) in brain tissues. GSH is a crucial molecule in detoxifying reactive oxygen species and maintaining cellular redox balance, which is often disrupted during seizures. By boosting GSH, the extract may mitigate oxidative stress, which plays a major role in seizure-induced neuronal injury. This antioxidant property likely contributes to the overall neuroprotective effect observed in the treated animals.

Furthermore, the extract demonstrated a marked reduction in lipid peroxidation, measured by decreased malondialdehyde (MDA) levels, indicating protection of neuronal membranes from oxidative damage. Since lipid peroxidation compromises membrane integrity and cell viability, its inhibition is critical in preserving neuronal function during epileptic events. This protective effect on membrane lipids further supports the therapeutic potential of the extract in managing epilepsy and related neurodegenerative conditions.

The extract also significantly improved total protein content in brain tissues, suggesting an enhancement of protein synthesis or prevention of protein degradation associated with seizure pathology. Maintaining adequate protein levels is essential for neuronal repair, synaptic plasticity, and overall brain health. This effect complements the antioxidant activity and may contribute to the restoration of normal neuronal functions following seizure episodes.

Phytochemical constituents such as flavonoids, phenolic compounds, and alkaloids present in the methanolic extract are likely responsible for its observed pharmacological actions. These bioactive compounds have been reported to exhibit antioxidant, anti-inflammatory, and neuroprotective properties, which collectively can modulate multiple pathways implicated in seizure genesis and propagation. Identification and characterization of these compounds could pave the way for novel, plant-based antiepileptic therapeutics with potentially fewer side effects than conventional drugs.

The findings of this study underscore the importance of exploring natural products as alternative or adjunctive therapies in epilepsy, particularly given the limitations and adverse effects associated with current antiepileptic medications. The dose-dependent efficacy and favorable biochemical profile of *Methonni* extract warrant further investigation through detailed mechanistic studies, isolation of active principles, and clinical evaluation.

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