Biolife 2(3):738-746
AN INTERNATIONAL QUARTERLY JOURNAL OF BIOLOGY & LIFE SCIENCES
ISSN (online): 2320-4257
www.biolifejournal.com
BIOLIFE RESEARCH ARTICLE

EVALUATION OF ANTI EPILEPTIC ACTIVITY OF ETHANOLIC EXTRACT OF *MURRAYA KAENIGII* ON MES, PTZ INDUCED CONVULSIONS IN RATS

M. Naveen Kumar[1*], Sravani Samala[2], E. Madhan Mohan[3], Ch Krishna Mohan[4]

[1-4] *Department of Pharmacology, St. John College Of Pharmacy,Yellapur,Warangal*

E-mail: mnkumar007@gmail.com

ABSTRACT

The aim of the study was to evaluate anti epileptic activity of ethanolic extract of *M. kaenigii* on MES, PTZ Induced convulsions in Rats. After the collection of the plant it was extracted with using ethanol solvent by maceration method. Preliminary Phytochemical analysis was done. Acute toxicity was studied. Assessments of antioxidant activity by assay of superoxide dismutase were done and also different enzyme analysis was studied. Effect of EEMK on ME induced convlusions was done in PHT treated animals. Secondary metabolites including alkaloids, saponins, cardiac glycosides and flavonoids were found to be present in the extract and these could have a wide range of compounds with pharmacological activities including those found in this study. Effect of antioxidant enzymes on MES and PTZ induced convolutions were studied. EEMK significantly inhibited the PTZ-induced convulsion, prolonged the latency for convulsion and decreased mortality. The PTZ kindling model is a widely used screening model for testing anticonvulsive compounds. Results based on the PTZ induced clonic seizures and MES induced generalized clonic-tonic seizures in rats model demonstrated that the ethanol extract of *Murraya koenigii* - Linn. is effective in reducing the severity of clonic seizures and generalized clonic-tonic seizures. This, therefore, supports the traditional use of the plant in the treatment of epilepsy.

Key Words: anti epileptic activity, *M. kaenigii,* PTZ, EEMK, PHT.

INTRODUCTION

The term epilepsy refers to disorders of brain function characterized by the periodic and unproductive occurrence of seizures (Vogal *et al.*, 1997). Although some 50 million people worldwide suffer from epilepsy, most of them in developing countries, an overwhelming majority of patients do not receive drugs to control the seizures. Certain relaxation techniques (namely meditation) have been reported to be potentially dangerous for people with epilepsy. Lowered blood pressure and brain electrophysiological arousal can be triggered which are associated with triggering seizures in some people (Miller, 1994).

When investigating the causes of seizures, it is important to understand physiological conditions that may predispose the individual to a seizure occurrence. Several clinical and experimental data have implicated the failure of blood–brain barrier (BBB) function in triggering chronic or acute seizures, some studies implicate the interactions between a common blood protein—albumin and astrocytes. These findings suggest that acute seizures are a predictable consequence of disruption of the BBB by either artificial or inflammatory mechanisms. In addition, expression of drug resistance molecules and transporters at the BBB are a significant

mechanism of resistance to commonly used anti-epileptic drugs.

The plant *Murraya koenigii* is an aromatic and more or less deciduous shrub or a small tree found throughout India up to an altitude of 1 500 m commonly in forests often as gregarious undergrowths. The species is native to India. It commonly occurs in the foothills of Himalaya, Assam, Sikkim, Kerala, Tamil Nadu, Andhra Pradesh and Maharashtra (Lal RK *et al.,* 2003). It is also found in evergreen and deciduous forests of peninsular India, often as underwood (Bhattacharjee SK 2004). An aromatic and more or less deciduous shrub or a small tree up to 6 m in height and 15 to 40 cm in diameter (Satyavati GV *et al.*, 1987). The main stem is dark green to brownish with numerous dots on it. Its bark can be peeled off longitudinally, exposing the white wood underneath.

Several compounds have been isolated from different morphological parts of the plant. The alkaloids (Nayak A 2010) and essential oils (Chowdhury JU *et al.,* 2008) are the most studied phytoconstituents of the plant. Apart from them, terpenoids, phenolics, minerals, protein, fat, carbohydrate, fibre, carotene, nicotinic acid, vitamin C etc. are also present in *M. koenigii* leaves. The leaves are fragrant, strongly aromatic, spicy, bitter, acrid, cooling and weakly acidic in taste. The essential oils impart intense characteristic flavor to the plant parts. Fresh leaves, dried leaf powder and essential oils are extensively used for flavoring soups, curries, chutneys, sausages, fish and meat dishes, pickles, butter milk preparations, egg preparations, curry powder blends, seasonings, ready-to-eat and many modern and other food preparations (Purthi JS 1976). Therefore the objective of this study to evaluate the anti epileptic activity of ethanolic extract of M. kaenigii on MES, PTZ induced convulsions in Rats.

MATERIALS AND METHODS

Collection and authentication of plant :
The leaves *Murraya koenigii* - Linn.belonging to family Rutaceae were collected surroundings of Warangal and authenticated by Dr Vatsavaya S . Raju, Professor, Botany department, Kakatiya University, Warangal, Andhra Pradesh. A voucher specimen was submitted at Botany department, Kakatiya University, Warangal.

Preparation of extract
The powdered dried leaves material was extracted with ethanol as a solvent by maceration, after completion of extraction, it was filtered and the solvent was removed. After complete extraction, the extract was dried. The yield was about 5% w/w and it was stored at 4^oC in desiccators. The extract was suspended in distilled water, for oral administration to animals.

Experimental Animal:
Male Albino rats, weighing 150-200g were procured from Sanzyme labd Hyd, AP. Animals were housed at CPCSEA (Reg No.1278/ac/09/CPCSEA) approved animal house of St. John College of Pharmacy, Warangal. The animals were kept in polypropylene cages (6 in each cages) under standard laboratory condition (12 hr light and 12 hr dark cycle) and had free access to commercial pellet diet (Hindustan lever L.td, Bombay, India) with water *ad libitum.* The animal house temperature was maintained at 25 ± 2^0C with relative humidity at (50 ±15%). The study was approved by the Institutional Animal Ethical Committee, (IAEC numbe of St. John College of Pharmacy. Ethical norms were strictly followed during all experiments.

Preliminary photochemical analysis
The freshly prepared ethanolic extract was analysed for phytochemical constituents as described by Trease and Evans (1989) for the detection of alkaloids, saponins, cardiac glycosides, sterols, terpenoids, proteins and amino acids, flavonoids and tannins (Kokate, 1994). These are described as follows:

Test for alkaloids
A sample of the freeze-dried extract (0.5 g) was boiled with 10 ml of dilute hydrochloric acid in a test tube for 5 minutes. The supernatant liquid was filtered into another test tube and 1 ml of the

filtrate was taken, into which 3 drops of Dragendorffs reagent (potassium bismuth iodide solution) was added. The mixture was shaken and observed for the appearance of an orange-red spot and a precipitate formation.

Test for Tannins

*T*wenty mg of extract was dissolved in 2 ml distilled water and filtered. Two ml $FeCl_3$ was added to the filtrate, blue-black precipitate indicated the presence of tannins.

Test for saponins

A small amount (0.2 g) of the extract was shaken with a few ml of water in a test tube and the mixture observed for the presence of a froth which does not break readily upon standing.

Test for Terpenoids

Knollar's test: 5 mg of extract is treated with 2ml of 0.1% anhydrous stannic chloride in pure thionyl chloride. A deep purple color that changes to red indicates the presence of terpenoids.

Test for *Protein and Amino Acids*:

Small quantity of the extract was dissolved in few ml of water and filtered. Filtrate was subjected to Millons test and Biuret test.

Test for cardiac glycosides

A sample of the extract (500 mg) was boiled in 5 ml of 70% ethyl alcohol for 2 minutes. The mixture was filtered and 10 ml of water and 5 ml of chloroform added to the filtrate and shaken. The lower chloroform layer was separated off and evaporated to dryness in a water bath. The cooled chloroform residue was dissolved in 3 ml of glacial acetic acid containing 0.1 ml of ferric chloride. The solution was carefully transferred to the surface of 2 ml of sulphiric acid and observed for a reddish-brown layer formed at the interface and whether the upper layer gradually acquired a bluish-green colour.

Test for flavonoids

Shinoda test: The extracts were dissolved in alcohol. One piece of magnesium followed by concentrated hydrochloric acid was added drop wise and heated. Appearance of magenta color demonstrated the presence of flavonoids.

Test for *Sterols*

Salkowaski test:

10 mg of extract was dissolved in 2 ml of chloroform and 2ml of concentrated sulphuric acid was added from the side of the test tube. Test tube was shaken for few minutes. The development of red color in chloroform layer indicated the presence of sterols.

Liebermann–Burchard Test:

1 ml of concentrated sulphuric acid was added to 10 mg of extract in 1ml of chloroform. A reddish–blue color exhibited by chloroform layer and green fluorescence by the acid layer suggests the presence of sterols.

Acute toxicity study according to OECD 423

The procedure was followed by using OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic class method). This Guideline is a stepwise procedure with the use of 3 animals of a single sex per step. Depending on the mortality and/or the moribund status of the animals, on average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex (normally females) (OECD, 2000). Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.

- No further testing is needed,
- Dosing of three additional animals, with the same dose,
- Dosing of three additional animals at the next higher or the next lower dose level.

Animals should be fasted prior to dosing (e.g. with the rat, food but not water should be withheld over-night, with the mouse, food but not water should be withheld for 3-4 hours). Following the period of fasting, the animals should be weighed and the test substance

administered. After the substance has been administered, food may be withheld for a further 3-4 hours in rats or 1-2 hours in mice. Where a dose is administered in fractions over a period it may be necessary to provide the animals with food and water depending on the length of the period.

Six rats weighing between 170±200gms were used for study. The starting dose level of ethanol extract of *Murraya koenigii* leaves extract was 2000mg/kg *(p.o)* body weight, as most of the crude extracts possess LD_{50} value more than 2000mg/kg. Dose was administered to the rats orally, which were fasted over night with water *ad libitum*, food were with held for a further 3-4 hrs after administration of drugs & observed for 14 days.

Body weight of the rats before and after treatment were noted and any changes in skin and fur, eyes and mucous membranes and also autonomic, central nervous systems, somatomotor activity and behavior pattern were observed and also signs of tremors, convulsions, salivation, diarrhea, lethargy sleep and coma were noted. The onset of toxicity and signs of toxicity was also to be noted. (Chan *et al.*, 1994).

Pharmacological studies

Method I

Maximal electroshock seizures (MES) induced convulsions

Group-1: Served as control
Group-2: Standard drug Phenytion (25mg/kg/i.p)
Group-3: Plant extract (200mg/kg/ P.O)
Group-4: Plant extract (400mg/kg/P.O)

Procedure

The Albino rats of 150-200 g of either sex animals (24 numbers) were used for the study. Seizures are induced to all the groups by using an electro convulsiometer. Maximal electroshock seizures were elicited by a 60Hz current of 150mA for 0.2sec. A drop of electrolyte solution (0.9% NaCl) was applied to the corneal electrodes prior to application to the rats. This increases the contact and reduces the incidence of fatalities. The METO (200mg/kg, 400mg/kg) were administrated for 14 days before induction of seizures. The duration of various phases (Flexion, Extension, Clonus, Stupor, and Recovery) of epilepsy were observed. The percentage protection was estimated by observing the number of animals showing abolition and duration of Hind Limb Tonic Extension (HLTE).

Method II

Pentylenetetrazole (PTZ) Induced convulsions

Group-1: Served as control
Group-2: Standard drug Diazepam(25mg/kg/i.p)
Group-3: Plant extract (200mg/kg/ P.O)
Group-4: Plant extract (400mg/kg/P.O)

Procedure

The Albino rats of 150-200 g of either sex animals (24 numbers) were used for the study. The test groups received two doses (200mg/kg, 400mg/kg) of METO orally for 14 days and test conducted for antiepileptic 1 h after the last doses of extract. PTZ (60mg/kg/i.p) is used as the inducing agent. After the administration of each extract, PTZ the each animal was placed in an individual plastic cage for observation lasting 1h seizures and tonic clonic convulsions were recorded. The control group animals were received saline while standard groups animals were received diazepam (4.0mg/kg/i,p.) and observed for onset of convulsions up to 1h after PTZ administration

Assessment of Antioxidant Activity:

The free radical scavenging activity of the plant extract was assessed by using DPPH against the standard Ascorbic acid.

Statistical analysis:

Data was analyzed by One-way ANOVA followed by Dunnet's multiple comparison test. P value <0.05 was considered significant

RESULTS

Preliminary Phytochemical Investigation:

The revealed results of the preliminary phytochemical screening of the ethanolic extract

of dried leaves of *Murraya koenigii* Linn. were shown below. The ethanolic extract given positive results for Alkaloids, Flavonoids, Glycosides, Phenols and cabohydrates.

Table-1: Preliminary phytochemical tests for ethanolic extract of *Murraya koenigii* (EEMK)

SL.NO	Phytochemical Tests	Results
1	Test for Alkaloids	+Ve
2	Test for Tannins	+Ve
3	Test for Proteins	+Ve
4	Test for Steroids	-Ve
5	Test for Saponins	-Ve
6	Test for Phenols	+Ve
7	Test for Flavonoids	+Ve
8	Test for Glycosides	+Ve
9	Test for carbohydrates	+Ve
10	Test for Gums and mucilage	+Ve
11	Test for Terpens	-Ve

+Ve: indicates the presence of compounds

-Ve: indicates the absence of compounds

Acute Oral Toxicity Study:

The acute oral toxicity study was done according to the OECD guidelines 423. A starting dose of 2000 mg/kg b.w/p.o of EEMK was administered to 3 either sex of rats, observed for three days. There was no considerable change in body weight before and after treatment of the experiment and no signs of toxicity were observed. When the experiments were repeated again with the same dose level, 2000 mg/kg b.w/p.o of EEMK for 3 days more, and observed for 14days, no changes were observed from the first set of experiment.

Effect Of Eemk On Mes Induced Convulsions

Phenytoin (PHT) treated animals have shown 100% protectin against MES induced convulsions where as EEMK 200 mg/kg and 400 mg/kg have shown 79.66% and 95.83% protection respectively against MES induced convulsions. The EEMK at both doses 200 and 400 mg/kg exhibited significant ($p<0.05$ and $p<0.01$) antiepileptic activity when compared with control. The results wee shown in Fig 1.

Figure-1: Effect of EEMK on MES induced convulsions

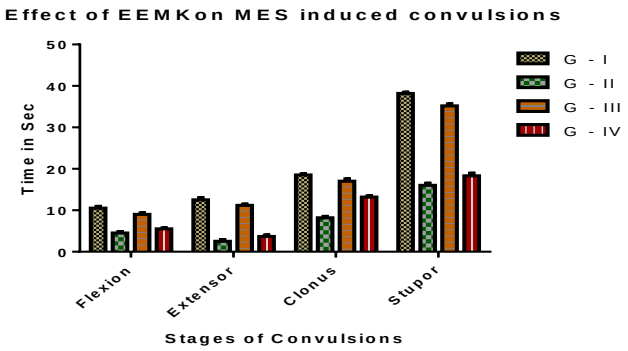


Figure-2: Effect of EEMK on neurotransmitter levels in MES induced convulsions

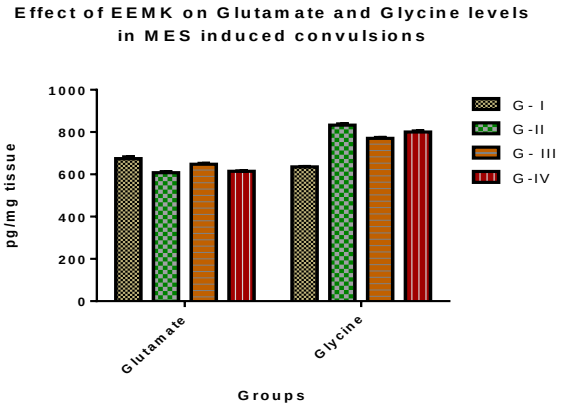
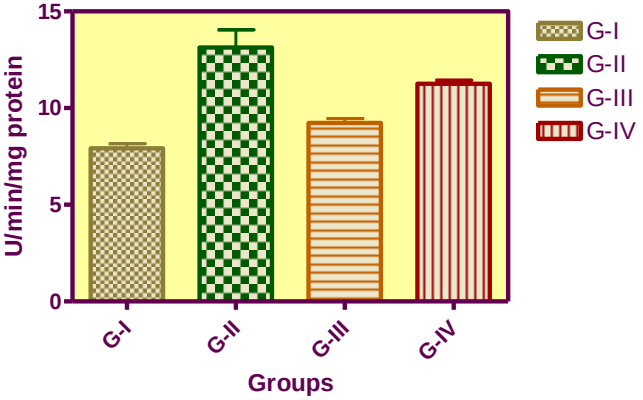


Figure-3: Effect of EEMK on Superoxide dismutase levels in MES induced convulsions



Effect of EEMK on neurotransmitter levels in MES induced convulsions GABA

A significant ($p<0.05$ and $p<0.01$) increase in GABA levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.05$) increase in GABA levels when compared with the control group animals. The results were shown in table 6 and fig-2.

Glutamate

A significant ($p<0.05$ and $p<0.01$) decrease in Glutamate levels was observed in brain on EEMK 20mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.05$) decrease in Glutamate levels when compared with the control group animals. The results were shown in table 6 and fig-2.

Glycine

A significant ($p<0.05$ and $p<0.01$) increase in Glycine levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.05$) increase in GABA levels when compared with the control group animals. The results were shown in table 6 and fig-2

Effect of EEMK on super oxide dismutase levels in MES induced convulsions

A significant ($p<0.01$ and $p<0.01$) increase in SOD levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.01$) increase in SOD levels when compared with the control group animals. The results were shown in fig 3

Effect of EEMK on Catalase levels in MES induced convulsions

A significant ($p<0.05$ and $p<0.01$) increase in catalase levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.01$) increase in catalase levels when compared with the control group animals. The results were shown in table 2.

Table 2: Effect of EEAS on Catalase levels in MES induced convulsions

Groups	Treatment	Catalase(µ mol H_2O_2 decomposed/mg protein/min)
I	Vehicle control	12.45±0.2760
II	PHT 25mg/kg.p.o	23.43±0.2130**
III	EEAS 200mg/kg.p.o	14.23±0.2146*
IV	EEAS 400mg/kg.p.o	20.123±0.1230**

Effect of EEMK on lipid peroxidation levels in MES induced convulsions

A significant ($p<0.01$ and $p<0.01$) decrease in MDA levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, PHT treated animals showed a significant ($p<0.01$) decrease in MDA levels when compared with the control group animals. The results were shown in table 3.

Table 3: Effect of EEAS on lipid peroxidation in MES induced convulsions

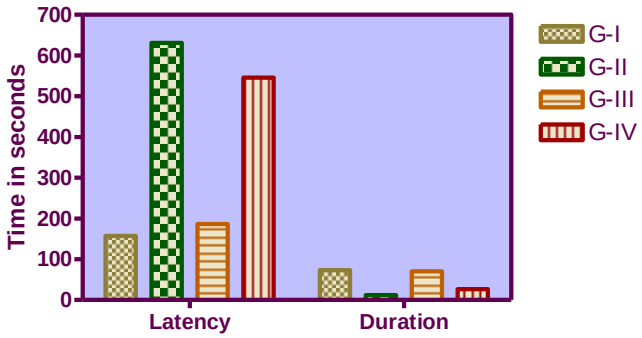
Groups	Treatment	Lipid peroxidation(n mol MDA/mg protein)
I	Vehicle control	4.23±0.167
II	PHT 25mg/kg.p.o	1.916±0.234**
III	EEAS 200mg/kg.p.o	3.618±0.2236**
IV	EEAS 400mg/kg.p.o	2.169±0.234**

Effect Of Eemk On Ptz Induced Convulsions

Diazepam treated animals have shown 100% protection against PTZ induced convulsions where as EEMK 200 mg/kg and 400 mg/kg have shown 70.8% and 100% protection respectively against PTZ induced convulsions. The EEMK at

both doses 200 and 400 mg/kg exhibited significant ($p<0.05$ and $p<0.01$) antiepileptic activity when compared with control. The results were shown in Fig 4.

Figure-4. Effect of EEMK on PTZ induced convulsions



Effect of EEMK on neurotransmitter levels in PTZ induced convulsions

GABA

A significant ($p<0.05$ and $p<0.01$) increase in GABA levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) increase in GABA levels when compared with the control group animals. The results were shown in table-4.

Table-4: Effect of EEMK on neurotransmitter levels in PTZ induced convulsions

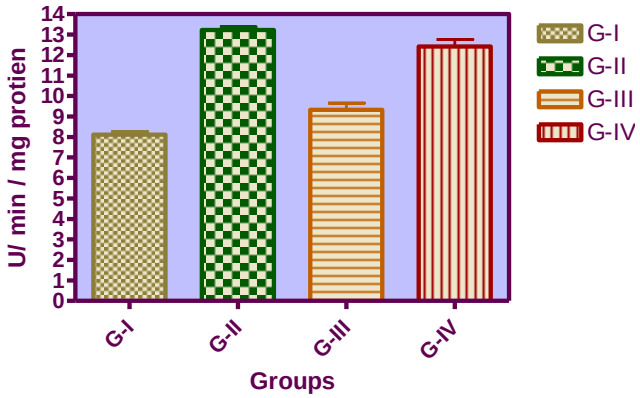
Group	Treatment	Glutamate	Glycine
I	Control	684.2±9.167	749.2±3.516
II	Diazepam	605.8±3.516**	837.5±6.292**
III	200mg	651.72±4.773*	772.5±3.594*
IV	400mg	614.2±2.002**	801.7±6.412**

Comparisons were made between: Group I with Group II, III and IV
Statistical significant test for comparison was done by ANOVA, followed by
*Dun net's-'t' test. **p< 0.01 and *p<0.05*

Glutamate

A significant ($p<0.05$ and $p<0.01$) decrease in Glutamate levels was observed in brain on EEMK 20mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) decrease in Glutamate levels when compared with the control group animals. The results were shown in fig-5.

Fig-5: Effect of EEMK on Superoxide dismutase levels in PTZ induced convulsions



Glycine

A significant ($p<0.05$ and $p<0.01$) increase in Glycine levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) increase in GABA levels when compared with the control group animals. The results were shown in table 4.

Effect of EEMK on super oxide dismutase levels in PTZ induced convulsions

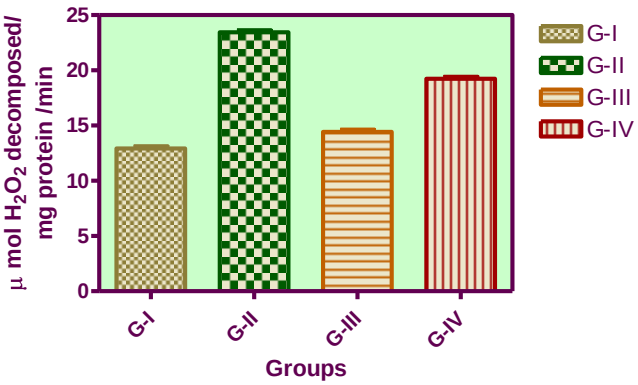
A significant ($p<0.01$ and $p<0.01$) increase in SOD levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) increase in SOD levels when compared with the control group animals. The results were shown in fig-5.

Effect of EEMK on Catalase levels in PTZ induced convulsions

A significant ($p<0.01$ and $p<0.01$) increase in catalase levels was observed in brain on EEMK

200mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) increase in catalase levels when compared with the control group animals. The results were shown in table 13 and fig 16

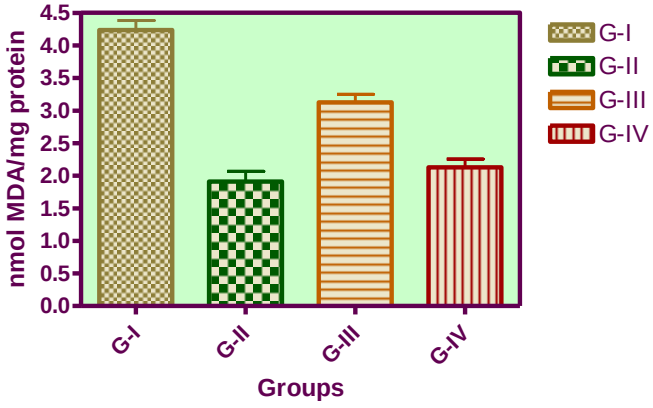
Figure-6: Effect of EEMK on Catalase levels in PTZ induced convulsions



Effect of EEMK on lipid peroxidation levels in PTZ induced convulsions

A significant ($p<0.05$ and $p<0.01$) decrease in MDA levels was observed in brain on EEMK 200mg/kg and 400mg/kg treated animals when compared with the control group animals, diazepam treated animals showed a significant ($p<0.01$) decrease in MDA levels when compared with the control group animals. The results were shown in fig 7.

Figure-7. Effect of EEMKon lipid peroxidation in MES induced convulsions



DISCUSSION

Epilepsy is a common chronic neurological disorder which imposes a burden on health care systems. It is a manifestation of a variety of diseases with variable mortality. Deaths could be due to the causative etiology itself, such as tumors, degenerative conditions or cerebrovascular diseases. Evidence indicates that the imbalance between excitatory and inhibitory neurotransmission in the brain is the main cause of seizure development in both experimental and clinical situations. This present study demonstrates that EEMK has antiepileptic activity

The extract inhibited seizures induced by PTZ and MES. Inhibition of seizures induced by PTZ and maximal electroshock in laboratory animals is the most common predictive screening tests used for characterizing potential anticonvulsant drugs. The maximal electroshock-induced seizure test is considered to be a predictor of likely therapeutic efficacy against generalised tonic-clonic seizures (Danesi and Adetunji, 1994).

EEMK significantly inhibited the PTZ-induced convulsion, prolonged the latency for convulsion and decreased mortality. The PTZ kindling model is a widely used screening model for testing anticonvulsive compounds. It mostly exerts its action via the t-butyl-bicycl phosphorothionate/picrotoxin site of the GABAA receptor (Huang et al., 2001).

The convulsion in MES method is due to the disturbed activity of GABA in the brain. MES seizures resembles to grandmal epilepsy. MES induced seizure can be prevented either by drugs that inhibit voltage gated sodium channel such as phenytoin or by drugs that inhibit glutaminergic excitation mediated by NMDA receptors such as felbmate. In addition, drugs that are effective in protecting animals against the tonic clonic extensor spasm induced by MES are effective in the management of and/or protecting against grand mal epilepsy. This implies that *Murraya koenigii* - Linn. may be effective as an anticonvulsant medicinal plant and its anticonvulsant effect may involve Gabergic inhibitory and Glutaminergic excitatory

mechanisms or inhibition of the voltage gated sodium channel.

Secondary metabolites including alkaloids, saponins, cardiac glycosides and flavonoids were found to be present in the extract and these could have a wide range of compounds with pharmacological activities including those found in this study.

CONCLUSION

Results based on the PTZ induced clonic seizures and MES induced generalized clonic-tonic seizures in rats model demonstrated that the ethanol extract of *Murraya koenigii* - Linn. is effective in reducing the severity of clonic seizures and generalized clonic-tonic seizures. This, therefore, supports the traditional use of the plant in the treatment of epilepsy. However, further bioassay guided phytochemical and pharmacological studies are required to identify the active principles and exact mechanism(s) of action.

REFERENCES

1. Chatterji S, Singh RK, Shukla S, Yadav DK, Watal G. Glycemic effect of freeze dried Murraya koenigii — an evidence based study[J]. Int J Pharma Bio Sci, 2010, 1(2) : 1-9.
2. Khuntia TK, Panda DS. Evaluation of antibacterial, antifungal and anthelmintic activity of Murraya koenigii Spreng[J]. Pharma Sci Monit, 2011, 2(2) : 105-110.
3. Ningappaa MB, Dhananjayaa BL, Dineshaa R, Harshaa R, Srinivas L. Potent antibacterial property of APC protein from curry leaves (Murraya koenigii L.)[J]. Food Chem, 2010, 118(3) : 747-750.

a. Rajeshwar Y and Lalitha R. 2013. Prelimiminary Phytochemical Screening And *In Vitro* Anthelmintic Effects Of *Acmella Paniculata* Plant Extracts. Biolife. 1(3), 106-112.

4. Pande MS, Gupta SPBN, Pathak A. Hepatoprotective activity of Murraya koenigii Linn bark[J]. J Herb Med Toxicol, 2009, 3(1) : 69-71.
5. Parmar C, Kaushal MK. Murraya koenigii. In: Parmar C, Kaushal MK. Wild fruits[M]. New Delhi: Kalyani Publishers, 1982. 45-48.
6. Wang YS, He HP, Shen YM, Hong X, Hao XJ. Two new carbazole alkaloids from Murraya koenigii[J]. J Nat Prod, 2003, 66(3) : 416-418

DOI: http://dx.doi.org/10.17812/blj2312
Received: 5 July 2014;
Accepted; 13 August 2014;
Available online : 5 September 2014