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IPOMEA RENIFORMIS HERB WATER AND METHANOLIC EXTRACT REVEALED PROTECTIVE ACTION AGAINST MES, PTZ AND LITHIUM PILOCARPINE INDUCED CONVULSIONS

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ABSTRACT

The aim of study was to assess the anticonvulsant potential of aqueous and methanolic extract of *Ipomea reniformis* in experimental animals. The anticonvulsant activity of aqueous and methanolic extract was tested at 100, 200 and 400 mg/kg orally for 7 days in mice using maximum electroshock seizure and PTZ induced seizure. The Lithium pilocarpine model of status epilepticus was also used for evaluation of anticonvulsant activity in rats. Methanolic extract showed dose dependant prevention of hind limb extension whereas aqueous extract showed prevention of hind limb extension except at dose of 100 mg/kg orally. In PTZ method, survival rate of mice was not remarkable but methanolic extract showed dose dependant delayed in onset and duration of convulsion. The Methanolic extract at dose 400 mg/kg orally significantly reduced the intensity of seizure in rats induced by Li-pilocarpine. The result revealed that aqueous extract at 400 mg/kg p.o. possess significant anticonvulsant activity in MES and Lithium pilocarpine while methanolic extract at 400 mg/kg p.o. showed anticonvulsant activity in all studied experimental models. Methanolic extract showed more effective action against all anticonvulsant models as compare to aqueous extract. In future, there is need of investigation for identification of the active compounds and their exact mechanism of actions for anticonvulsant activity in experimental animals.

Keywords: Anticonvulsant, *Ipomeareniformis*, PTZ, Lithium-pilocarpine

INTRODUCTION

Epilepsy is a chronic disorder of central nervous system affecting about 1% of the world population and its overall occurrence is around 20 to 70 per 100000 populations per year¹. The prevalence rate is higher in developing countries and it is increasing each year. A large part of the problem is that epilepsy is very difficult to diagnose and treat effectively. The aim of drug is to control and totally prevent all seizure activity at an acceptable level of side effects but only 40-60% of patients become totally seizure free with available drugs and another 20-30% gets partial control, while rest remain resistant¹.

Many new antiepileptic drugs have been developed in the past 15-20 years; one of the most active area for drug development is to improve their efficacy and side effect profile. Improvements in antiepileptic drug have been steady rather than spectacular, and epilepsy remains a difficult problem. The alternative drug therapy for the management of this disease can be by the use of medicinal plants². Hence there is need to focus on the scientific exploration of herbal drugs having fewer side effects. According to WHO statistics, around 25% of the preserved human medicine are derived from plants³. The knowledge of traditional medicine is essential for the discovery of new and potent medicine but scientific data is lacking for many medicinal plants in India. Hence in the present study plants from India with a traditional claim of antiepileptic activity was selected and screened for antiepileptic activity.

Ipomea reniformis (*I. reniformis*) Roxb.Choisy Family-convolvulaceae is a perennial, much branched herb having therapeutic and potential use. *I. reniformis* is also known as *Merremia marginata* Burm. It is widely distributed all over the India, especially in damp places in upper genetic plain, Gujarat,

Bihar, West Bengal, Western-Ghats, ascending up to 900 m in the hills, Goa, Karnataka in India, Ceylon and Tropical Africa⁴.

A decoction of plant is said to act as deobstruent, diuretic; useful in rheumatism, neuralgia, headache, anthelmintic; diseases of the kidney, the lungs, the uterus; good in pains, fevers urethral discharges, anaemia, lucoderma⁵. Leaf juice is given in rat bites and snake bites⁶. Powder of leaves is sniffed up in epilepsy^{7, 8}. Paste of the root used in swelling⁹.

I. reniformis studied for its traditionally claimed activity. The present study was planned to explore the possible antiepileptic potential of aqueous and methanolic extract of *I. reniformis* in experimental animals.

MATERIALS AND METHODS

Plant material

I. reniformis whole plant was collected from Nilanga city, Dist. Latur, Maharashtra, India in between the month of October to December 2014. The plant specimen was authenticated by A. Benniamin, Scientist 'D', Botanical Survey of India, Western regional centre, Pune-411001. The whole plant was washed using water and shade dried at room temperature to retain its vital phytoconstituents and then subjected to size reduction for further extraction process. About one kilogram of the dried plant material was powdered with mechanical grinder and sieved to get uniform particle size.

Preparation of Extract

The powder material was defatted with petroleum ether and then successively extracted with solvent methanol and water by maceration method for 24 hrs. Mark was dried at room

temperature during solvent changing. The extract was concentrated for further studies on water bath at 40°C. The extract was finally air dried then stored in an air tight container till used.

Preliminary Phytochemical investigation of extracts¹⁰

A preliminary phytochemical analysis of the methanolic and aqueous extract was carried out for the presence of various phytoconstituents like flavonoids, saponin glycosides, and tannins, carbohydrates, reducing sugars, steroids and Amino acids.

Experimental Animals

Young albino Wister male rats (180–220 g) and Swiss albino mice of either sex (25–30 g) were used in anticonvulsant testing and maintained under standard laboratory conditions, which include a temperature of 20–24°C, relative humidity of about 50–60% and a 12-h light cycle beginning early in the morning. The animals allowed free access to food (Golden Feed, New Delhi) and water *ad libitum* except during the short time before testing. All the animals were acclimatized for a week before the study and randomized into different groups. Aqueous and methanolic extract and standard prototype drugs (diazepam, phenytoin) were administered once daily (0900) in the morning for a period of 07 days. Food, but not water was withdrawn 3–4 h before the experiment. Protocol of the study approved from the Institutional Animal Ethics Committee (No. IAEC/ABCP/09/2014-15 Date 17/10/2014) and conducted according to CPCSEA guidelines, Govt. of India.

Acute Oral Toxicity Study¹¹

Albino mice of either sex weighing 25–30 g selected by random sampling technique was performed as per OECD-423 guidelines. The animals were fasted for 3–4 hr, provided only water, after which the extract was administered to the respective groups orally at the dose level of 5 mg/kg body weight by stomach intubation. If mortality was observed in 2 or 3 animals, then the same dose was repeated then the dose administered was as a toxic dose. If mortality was observed in one animal, then the same dose was repeated again to confirm the toxic dose. If mortality was not observed, the procedure was repeated for further higher doses such as 5, 50, 300 and 2000 mg/kg orally and the animals were observed for 72 h.

ANTICONVULSANT METHODS

Maximum Electroshock Induced Seizures (MES)¹²

The maximal electroshock (MES) method was performed to induce the seizures in order to screen for antiepileptic activity. Albino Mice were randomly distributed in to nine groups of six animals each. Vehicle, standard drug and extract dose pretreated once daily for 7 days. Group I served as control (vehicle treated not MES induced), Group II served as disease control group (Vehicle treated MES induced), Group III served as reference standard (received Phenytoin sodium 25 mg/kg i.p. body weight); Group IV to IX were treated with Aqueous and methanolic extract Dose 100, 200, 400mg/kg orally. The test extract were administered orally, 1hr prior to induce the convulsion and standard drug (Phenytoin sodium 25 mg/kg) was administered i.p.30 min before to induce the convulsion. Electroconvulsive shock (50 mA for 0.2 sec) was delivered through corneal electrode to induce convulsions to eight groups of mice (n=6) except control group. The various phases of convulsion which were produced are flexion, extension, clonus and stupor. Prior to delivery, current output was checked by

multimeter. After the electric stimulation occurrence, the duration of phases were noted.

Pentylenetetrazol-induced seizures (PTZ)¹³

Albino Mice were randomly distributed in to nine groups of six animals each. Vehicle, standard drug and extract dose pretreated once daily for 7 days. Epilepsy was induced by subcutaneous injection of PTZ (80 mg/kg s.c.) and the animals were observed for onset of myoclonic spasm and clonic convulsions. Group I served as control (vehicle treated not PTZ induced), Group II served as disease control group (Vehicle treated PTZ induced), Group III served as reference standard (received diazepam 2.0 mg/kg, i.p. body weight); Group IV to IX were treated with aqueous and methanolic extract Dose 100, 200, 400mg/kg orally. The test extract were administered orally, 1hr prior to induce the convulsion and standard drug was administered i.p.30 min before to induce the convulsion. The animals were observed for onset of convulsion up to 30 min after PTZ.

Lithium-Pilocarpine Induced Status Epilepticus¹³ (Li-Pilocarpine)

Albino Wistar rats were randomly distributed in to nine groups of six animals each. Vehicle, standard drug and extract dose pretreated once daily for 7 days. Status epilepticus was induced by administration of pilocarpine (30 mg/kg, i.p.) 24 h after lithium sulphate (3 meq/kg, i.p.). Group I served as control (vehicle treated not PTZ induced), Group II served as diseased control group (Vehicle treated PTZ induced), Group III served as reference standard (received diazepam 2.0 mg/kg, i.p. body weight); Group IV to IX were treated with aqueous and methanolic extract Dose 100, 200, 400mg/kg orally. The test extract were administered orally, 1hr prior to induce the convulsion and standard drug was administered i.p.30 min before to induce the convulsion. The severity of status epilepticus was observed every 15 min till 90 min and then every 30 min for next 90 min using the scoring system as described: stage 0- no response, stage 1- fictive scratching, stage 2- tremors, stage 3- head nodding, stage 4- forelimb clonus, stage 5- rearing and falling back.

Statistical analysis

The data were expressed as mean±SD. Difference between the groups was statistically determined by one way ANOVA followed by Bonferroni's test. Level of significance was set at p< 0.05 and p<0.001.

RESULTS

Acute Toxicity

Aqueous and Methanolic extract of *I. reniformis* was exposed to acute toxicity study. For the LD50 dose determination, extract was administered up to dose 2000 mg/kg orally and extract did not produce any mortality, thus 1/5th, 1/10th, 1/20th of maximum dose tested were selected for the present study.

Phytochemical Screening

In preliminary investigation, methanolic extract revealed the presence of tannins, cardiac glycoside, anthraquinone, saponin, steroids, carbohydrate, proteins and amino acids, while aqueous extract contain tannins, cardiac glycoside, saponin, steroids, carbohydrate, proteins.

MES Induced Seizure

MES produced various phases of convulsion i.e. hind limb extension, flexion, clonus and stupor. All the phases of MES induced convulsions were significantly reduced by aqueous and methanolic extract are shown in Table 1. Decrease in the duration of hind limb extension was considered as protective action. The duration of hind limb extension in disease control group was compared with all extract treated groups. Methanolic extract at dose 100, 200 and 400 mg/kg b.w. p.o. were significantly reduced duration of hind limb extension as compared with disease control. Aqueous extract at dose 200 and 400 mg/kg b.w. p.o. decreases duration of hind limb extension which is compared with disease control. However aqueous extract at dose 100 mg/kg b.w. p.o. did not show significant effect on duration of hind limb extension when compared with disease control. Standard drug phenytoin at a dose 25 mg/kg i.p. reduced all the phases of convulsions significantly ($p < 0.05$) as compared with disease control. The aqueous and methanolic extract was able to decrease the duration of hind limb extension (extensor phase), but the extract at 400 mg/kg p.o. dose possesses potent anticonvulsant activity as compared with disease control.

PTZ Induced Seizure

The results of anticonvulsant effect of methanolic and aqueous extract against PTZ induced seizures are shown in Table 2. Administration of PTZ to mice produced myoclonic convulsion in all animals. Methanolic extract at dose 100, 200 and 400 mg/kg b.w. p.o. were significantly delayed in onset of convulsion as compared to disease control group. However duration of convulsion was significantly reduced at dose 200, 400 mg/kg p.o. Aqueous extract dose at 200 and 400 mg/kg p.o. were significantly reduced in onset of convulsion, while duration of convulsion was significantly reduced at dose 400 mg/kg p.o. as compared with disease control group. Standard drug diazepam 5 mg/kg i.p. was completely abolishes the convulsion and offered 100% protection. The statistical data obtained from the anticonvulsant effect of methanolic and aqueous extract against PTZ-induced seizure revealed that methanolic extract at 200 and 400 mg/kg p.o. and aqueous extract at 400 mg/kg p.o. showed significant activity when compared with the effect produced by disease control. There was incidence of mortality in the group of animals treated with extracts except in methanolic extract at 400 mg/kg p.o. Overall the methanolic extract at dose 400 mg/kg p.o. was able to show potent activity against PTZ-induced seizures.

Table 1: Effect of methanolic and aqueous extract of *I. reniformis* against MES induced convulsions

S No	Treatment	Duration of HLE (Sec)	Flexion (Sec)	Clonus (Sec)	Stupor (Sec)	Recovery (Sec)
1	Vehicle Control	Nil	Nil	Nil	Nil	
2	MES control	16.00±3.347	13.67±3.077	17.00±3.742	86.50±7.918	130.83±10.778
3	Standard Phenytoin (25mg/kg)	1.83±1.472 ^{a***}	1.33±0.516 ^{a**}	3.33±2.066 ^{a**}	17.83±3.656 ^{a**}	68.33±3.933 ^{a**}
4	Methanolic 100	12.67±1.966 ^{a***}	12.67±2.160 ^{aNSb**}	14.83±3.061 ^{aNSb**}	79.00±7.266 ^{a***}	121.00±6.261 ^{a***b**}
5	Methanolic 200	10.33±1.211 ^{a***b**}	9.17±1.472 ^{a***b**}	12.33±2.066 ^{a***b**}	71.17±6.080 ^{a***b**}	112.33±5.241 ^{a***b**}
6	Methanolic 400	8.00±1.414 ^{a***b**}	6.83±1.472 ^{a***b**}	9.33±2.805 ^{a***b**}	59.00±4.775 ^{a***b**}	98.33±5.610 ^{a***b**}
7	Aqueous 100	14.17±3.312 ^{aNSb**}	13.00±1.789 ^{aNSb**}	16.67±2.582 ^{aNSb**}	81.50±8.167 ^{a***b**}	129.00±7.694 ^{aNSb**}
8	Aqueous 200	11.83±2.483 ^{a***b**}	10.17±1.722 ^{a***b**}	14.50±2.168 ^{aNSb**}	76.67±7.005 ^{a***b**}	116.50±7.423 ^{a***b**}
9	Aqueous 400	9.83±1.472 ^{a***b**}	8.33±1.506 ^{a***b**}	11.33±2.066 ^{a***b**}	65.33±4.179 ^{a***b**}	104.83±5.037 ^{a***b**}

Values are Mean± SD n= 6 in each group. Plant extract groups are compared against Disease control- a and against standard- b
^{*}<0.05, ^{**}<0.001 NS- Non-significant

Table 2: Effect of methanolic and aqueous extract of *I. reniformis* against PTZ induced convulsions

S No	Treatment	Onset Of Myclonic spasms After PTZ (sec)	Onset Of Clonic Convulsion After PTZ (sec)	Duration Of Convulsion (sec)	Mortality	% survival
1	Control	Nil	Nil	Nil	0/6	100
2	PTZ Control	98.50±8.264	116.67±11.130	83.17±6.242	6/6	0
3	Standard Diazepam (5 mg/kg)	0±0 ^{a**}	0±0 ^{a**}	0±0 ^{a**}	0/6	100
4	Methanolic 100	116.00±6.603 ^{a***b**}	133.83±10.108 ^{a***b**}	78.33±4.761 ^{aNSb**}	6/6	0
5	Methanolic 200	126.00±8.075 ^{a***b**}	143.50±9.006 ^{a***b**}	73.67±5.785 ^{a***b**}	6/6	0
6	Methanolic 400	150.50±11.327 ^{a***b**}	167.17±8.424 ^{a***b**}	57.67±5.574 ^{a***b**}	4/6	33.34
7	Aqueous 100	103.00±.187 ^{aNSb**}	123.83±10.629 ^{aNSb**}	84.00±7.043 ^{aNSb**}	6/6	0
8	Aqueous 200	119.00±9.757 ^{a***b**}	134.67±9.309 ^{a***b**}	77.67±6.022 ^{aNSb**}	6/6	0
9	Aqueous 400	135.83±8.750 ^{a***b**}	152.67±8.501 ^{a***b**}	69.17±5.193 ^{a***b**}	6/6	0

Values are Mean± SD n= 6 in each group. Plant extract groups are compared against Disease control- a and against standard- b
^{*}<0.05, ^{**}<0.001 NS- Non-significant

Table 3: Effect of methanolic and aqueous extract of *I. reniformis* against Li-pilocarpine induced status epilepticus

S No	Time after Pilocarpine (Min)	Vehicle control	Disease Control	Standard Diazepam (5 mg/kg)	Methanolic 100	Methanolic 200	Methanolic 400	Aqueous 100	Aqueous 200	Aqueous 400
1	15	Nil	1.67±0.516	0.50±0.548 ^{a**}	1.50±0.548 ^{aNSb**}	1.33±0.516 ^{aNSb*}	0.83±0.408 ^{a*}	1.67±0.516 ^{aNSb**}	1.50±0.548 ^{aNSb*}	1.17±0.753 ^{aNSb*}
2	30	Nil	2.83±0.408	0.50±0.548 ^{a**}	2.17±0.408 ^{a**}	2.17±0.408 ^{a**}	1.50±0.837 ^{a**}	2.83±0.408 ^{aNSb**}	2.50±0.548 ^{aNSb*}	1.83±0.753 ^{a**}
3	45	Nil	3.33±0.516	0.83±0.408 ^{a**}	3.00±0.632 ^{aNSb**}	2.83±0.753 ^{aNSb**}	2.00±0.632 ^{a**}	3.33±0.516 ^{aNSb**}	3.00±0.632 ^{aNSb*}	2.33±0.516 ^{a**}
4	60	Nil	3.67±0.516	1.33±0.816 ^{a**}	3.33±0.516 ^{aNSb**}	3.17±0.753 ^{aNSb**}	2.67±0.516 ^{a**}	3.67±0.516 ^{aNSb**}	3.33±0.516 ^{aNSb*}	3.17±0.753 ^{aNSb**}
5	75	Nil	4.17±0.408	1.50±0.837 ^{a**}	3.67±0.516 ^{aNSb**}	3.67±0.516 ^{aNSb**}	3.17±0.753 ^{a**}	4.00±0.000 ^{aNSb**}	3.83±0.408 ^{aNSb*}	3.50±0.548 ^{a**}
6	90	Nil	4.50±0.548	1.67±0.816 ^{a**}	4.17±0.408 ^{aNSb**}	3.83±0.753 ^{a**}	3.33±0.816 ^{a**}	4.33±0.516 ^{aNSb**}	4.00±0.632 ^{aNSb*}	3.67±0.516 ^{a**}
7	120	Nil	4.67±0.516	1.17±0.753 ^{a**}	3.50±0.548 ^{a**}	3.33±0.516 ^{a**}	3.00±0.632 ^{a**}	4.00±0.632 ^{aNSb**}	3.67±0.516 ^{a**}	3.33±0.516 ^{a**}
8	150	Nil	3.83±0.408	0.50±0.548 ^{a**}	2.83±0.408 ^{a**}	2.50±0.548 ^{a**}	2.00±0.632 ^{a**}	3.00±0.632 ^{a**}	2.67±0.516 ^{a**}	2.33±0.516 ^{a**}
9	180	Nil	2.67±0.516	0.33±0.516 ^{a**}	1.83±0.753 ^{a**}	1.50±0.837 ^{a**}	1.17±0.753 ^{a**}	2.17±0.753 ^{aNSb**}	2.00±0.632 ^{a**}	1.33±0.516 ^{a**}

Values are Mean± SD n= 6 in each group. Plant extract groups are compared against Disease control- a and against standard- b
^{*}<0.05, ^{**}<0.001 NS- Non-significant

Li-Pilocarpine Induced Seizure

Rats treated with lithium and pilocarpine showed stage 4 seizures in all animals 75 min after pilocarpine. The methanolic extract at 400mg/kg p.o. significantly reduced the intensity of seizures as compared with disease control. The aqueous extract in a dose of 400 mg/kg p.o. was reduced the intensity of seizure but it is not as significant as methanolic extract dose 400mg/kg p.o.. The standard drug diazepam 5mg/kg i.p. significantly diminished the intensity of seizures as compared with disease control and none of the animals exhibited stage 4 seizures and the animals were normal in behaviour after 180 min. The observations are given in Table 3.

DISCUSSION

Epilepsy is chronic disorder of CNS, imbalances of neurotransmitters in brain are responsible for seizure¹⁴. Current therapy for epilepsy have efficacy along with side effects. Medicinal plants are the alternative therapy for management of epilepsy but scientific data is lacking for traditionally claimed plants. Hence traditionally claimed plant was selected and screened for antiepileptic activity.

Attempts to find out a common neurochemical basis for human or experimental epilepsy have been disappointing. Various animal models of convulsions have been developed to evaluate antiepileptic activity. In-vitro systems have limited utility in antiepileptic drug discovery and always necessary to validate activity by using animal models for prediction¹⁵. Therefore the aim of study was assessment of the possible anticonvulsant effect of *I. reniformis* in MES, PTZ and lithium-pilocarpine animal models.

The MES method is probably the best validated method for assessment of antiepileptic drugs in generalised tonic-clonic

seizure¹⁶. In MES test, mice received electrical stimulus to induce seizure of their hind limbs extension as the end point of test. The MES model is used to identify a compound which prevents hind limb extension in mice induced by electrical shock¹⁷. Clinically effective drugs like phenytoin, carbamazepine were effective in generalised seizure and these compounds have the ability to inactivate voltage dependant Na⁺ channels or by drugs that inhibit glutaminergic excitation mediated by NMDA receptors in a dose dependant fashion¹⁸.

The PTZ model is thought to represent a valid model for generalized absence seizures¹⁷. PTZ is believed to be an antagonist of GABA-A receptor and produces convulsion by inhibiting the GABA pathway in the CNS responsible for hyperexcitation of neurons leads to convulsion¹⁹. The PTZ induced seizures are similar to the symptoms observed in the absence seizures and drugs useful in treatment of absence seizures suppress PTZ induced seizures¹⁷. Drugs effective in PTZ induced seizure are drugs that reduces the T-type of Calcium channels or drugs that mimicking GABA mediated neurotransmission in brain²⁰. Status epilepticus is a medical emergency, resulting from continuous or repetitive grand mal seizure. The status epilepticus induced by lithium-pilocarpine increases brain contents of acetylcholine. Cholinomimetic convulsant pilocarpine is used to induce a status epilepticus, which is followed by hippocampal damage and development of spontaneous recurrent seizures²¹. Diazepam drug is choice of drug for status epilepticus treatment. Moreover, MES and PTZ induced seizure models are also associated with oxidative damage²².

The methanolic and aqueous extracts were found no mortality up to the dose of 2000 mg/kg orally. Hence 1/5th, 1/10th and 1/20th of maximum dose tested were selected for the study. Since aqueous and methanolic extract abolishes MES, PTZ and lithium-pilocarpine seizure. It might possess sodium channel

blocker, NMDA blockage, Calcium channel blockage or may be due to mimicking action of neurotransmitter GABA. Anticonvulsant activity may also be due to antioxidant activity²³. Methanolic and aqueous extracts showed anticonvulsant activity in studied anticonvulsant models but methanolic extract at 400mg/kg orally dose exhibited more potent activity.

Various chemical constituents of plant origin particularly alkaloids, flavonoids, terpenoids, saponins and coumarins are reported to have anticonvulsant activity in experimental animal models²⁴. Since anticonvulsant activity can be due to presence of various phytochemicals like tannins, flavonoids, cardiac glycoside, anthraquinone, saponin, steroids in *I. reniformis*²⁵.

In further investigation, there is need of identification of the active compounds and their exact mechanism of actions responsible for anticonvulsant activity of *I. reniformis*. World over, the general opinion is tilting towards use of herbal drugs. Hence anticonvulsant potential of *I. reniformis* will help the researchers to develop herbal medicine.

CONCLUSION

On the basis of results obtained in this study we conclude that *I. reniformis* extracts shows anticonvulsant activity in MES, PTZ and Li-pilocarpine models. However, the exact mechanism and the active compound involved in these effects need to be clarified in future studies. In future, the development of formulation by these plant constituents may give rational drug against convulsion.

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