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Research Article

INVESTIGATION OF ANTIEPILEPTIC PROPERTY OF *STERCULIA GUTTATA* LEAVES

Vikas Jogpal *, Jyoti Gupta, Tarun Virmani
School of Pharmaceutical Sciences, MVN University, Palwal
Author for Correspondence *Email: rxvikasjogpal@gmail.com

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ABSTRACT

The present study was done to investigate the antiepileptic activity of different extracts of leaves of *Sterculia guttata.* Methanolic and aqueous extracts of the leaves of S*terculia guttata* were evaluated for antiepileptic activity using PTZ (Phenyltetrazole 100 mg/kg), MES (Maximal Electroshock Convulsions 75 mA), Strychnine (4 mg/kg) and Picrotoxin (3.5 mg/kg) induced seizures. Swiss albino mice were randomly selected and subjected to dosages of 100, 300 and 500 mg/kg of body weight of each extract used in study. Diazepam and phenytoin were used as standard drugs for all experimental methods in the dose of 4 mg/kg and 25 mg/kg respectively. The results were analyzed by applying one-way ANOVA (Analysis of variance) followed by Dunnett's t- Test and compared with the control group. The software used for calculating the significance of result was Graph Pad (All values expressed as mean ± SEM; n = 6 mice in each group, significance values are *p < 0.05, **p < 0.01 and ***p < 0.001.) The methanol extracts of leaves of *Sterculia guttata* showed significant anticonvulsant activity by increasing onset of seizure, the time of latency and reducing duration of extensor phase of seizure in MES and PTZ induced seizures. Picrotoxin and strychnine induced seizure model also showed significant results but up to less extent as compare to MES and PTZ animal models. The ideal dose from the methanol extract was 300 and 500 mg/kg. The aqueous extract showed significant result but only at the dose of 500 mg/kg.

Keywords: *Sterculia guttata*, Epilepsy, Seizures, MES, PTZ, Treatment Gap, ANOVA

INTRODUCTION

Epilepsy is one of the most complex neurological disorders whose cure is very challenging. Its prevalence and progression are increasing day by day. Total number of patients worldwide estimated by a study was 50 million, out of which 20-40% are drug resistant[1]. Nearly one-sixth of the global burden (about 12 million) is bore by India[2]. Out of several types of epilepsies only, few have been reported for their partially known pathophysiology e.g. epilepsies caused by disorders of neuronal migration and monogenic epilepsies[3]. The complexity of this disorder is one of major cause of less available drugs, treatment gap and frequent resistance of AEDs (anti-epileptic drugs). Even the present AEDs are costly and 20-30% patients could not receive proper medications[4]. In order to cumulate precautionary, primitive, remedial, and rehabilitative services for mankind and to understanding the need, risk factors, and determinants of epilepsy, it is required to investigate more aspects and tools to treat epilepsy. This investigation work is a step towards reducing the treatment gap and to improve the quality of epilepsy treatment. In view of above said problem many plants have already been investigated, to add to this *Sterculia guttata* have been selected for present study. *Sterculia guttata* is deciduous tree growing up to 20 meters tall. The bole is straight and cylindrical, and the leaves are hairless above, velvety beneath, oblong-ovate acute or acuminate. *Sterculia guttata* (Malvaceae) is a tree commonly known as 'Hirik' tree. *Sterculia guttata* seeds has been already reported for its CNS depressant[5] and for their mosquito larvicidal[6] activity. *Sterculia foeteda* (a plant with same genus) has been reported for its antiepileptic activity[7]. *Sterculia guttata* is found in local regions of Maharashtra, Assam and Andaman in India and also found in Sri Lanka.

METERIALS AND METHODS

Drugs and chemicals

All the standard drugs were received as drug sample. Phenytoin (Sun Pharmaceuticals India Ltd, Halol, Gujarat, India). Diazepam Ranbaxy Laboratories Ltd, HMTD Textile, India). Pentyletetrazole (Sigma Aldrich, St. Louis, MO63103. USA). All the solvents used for the extraction were of laboratory grade and they were purchased from local firm.

Experimental animals

Swiss albino mice of either sex (18-25 g) were obtained and acclimatized in the animal house of Advanced Institute of Pharmacy, Palwal, Haryana. Standard conditions were provided to all the animals, that is room temperature 26 ± 1˚C relative humidity 45-50% and 12-12 hours light dark cycle. The experimental protocols were approved by the institutional animal ethical committee (IAEC) of Advanced Institute of Pharmacy, Palwal (Approval No IAEC/AIP/2015/438, dated 18/12/2015) and all the experiments were conducted according to the guidelines of CPCSEA.

Preparation of Extract

The leaves of *Sterculia guttata* were collected from local region of Pune, Maharashtra. Plant was authenticated at department of Botany, Pt. Jawahar Lal Nehru government college (Approval No: JLNG/2016/1213) Faridabad, Haryana. A sample of which was kept in the department. (Herbarium voucher specimen number JLNG/13/2016)

Leaves of *Sterculia guttata* were dried at room temperature and pulverized to form coarse powder extracts were prepared by the method described by Rosenthaler[8]. 500 g of powdered leaves were placed in Soxhlet apparatus, (Perfit, India) and defatted with petroleum ether (60°-80°C), methanol and water. This extraction process was continued for about 48 h or until methanol coming down the siphoning became colourless. Aqueous extract was obtained after macerating the powdered leaves (500 g for 72 h) with occasional stirring, the extracts so formed were filtered and the filtrate was evaporated using vacuum evaporator (Perfit India) under reduced pressure at ≥ 50˚C. The crude extract obtained after evaporation were stored in desiccators and weighed. Physical Nature and % yield of extract was recorded. The % yield was evaluated using the following formula:

% Yield= Weight of extract (g)/ Weight of dry powder (g) ×100

The detail of the prepared extracts is mentioned below:
SGME: *S. guttata* leaves methanol extract
SGAE: *S. guttata* leaves aqueous extract

Preliminary phytochemical screening

The extracts were dissolved in methanol and were further diluted with distilled water so as to give a concentration of 10 % w/v. The solution so prepared was subjected to various tests. Preliminary phytochemical screening was done according to standard procedures[9].

Acute Toxicity Studies

Oral toxicity of various extract of *S. guttata* were estimated by using mice of weight 18 - 25 g. Prior to experiment, animals were kept fasting for 5 h. Animals were administered with single dose of extract and observed for their mortality during 48 h study period (short term) toxicity[10]. Gradually increased the dose of *S. guttata* extract was administered to mice. LD_{50} was calculated as per OECD guidelines 425[11].

Pentylenetetrazole (PTZ) Induced Seizure Test

Swiss albino mice were divided into 5 groups with 6 animal in each, either group served as control (NS 0.1%), standard (diazepam 4 mg/kg) and plant extracts (*S. guttata* 100, 300 and 500 mg/kg) all group received PTZ (100 mg/kg) after 30 min of standard/extracts respectively except control group which receive NS before the administration of PTZ. All groups receive PTZ through intra peritonial route of administration. Each animal was individually investigated for following parameter i.e. onset of seizure, duration of seizure and recovery or death. Parameters were initially recorded for 30 min at the end of 24 h[12]

Maximal Electroshock Induced Convulsion

Maximal electroshock induced convulsion was done by using swiss albino mice with body weight 18 to 25 g. Animal were divided into 5 groups with 6 animals in each, as discussed in PTZ method. Control group received normal saline, standard group received phenytoin (25 mg/kg) orally and test group received single dose of 100, 300, 500 mg/kg of *S. guttata* respectively, 30 min after administration of extract in control group, experiment was started. Maximal electroshock (MES, inco) of 50 mA current for 0-2 sec was given to each animal individually through ear electrode to induce convulsion to drug treated and control group[13].

Strychnine Induced Convulsions

Mice were randomly selected and named as control, test and standard with same criteria as that of PTZ model. The control groups receive strychnine (2 mg/kg) after 30 min of administration of normal saline. Standard group was administered with diazepam (5 mg/kg) and then with strychnine (2 mg/kg) after 30 min. test group received strychnine (2 mg/kg) 30 min after the administration of 100, 300 and 500 mg/kg of *S. guttata* extract respectively. Any mouse did nit convulse within 30 min after strychnine administration was considered protected[14]

Picrotoxin Induced Convulsions

Mice were randomly allotted as described in strychnine induced method. Picrotoxin (3.5 mg/kg) was used to induce seizure using same protocol as discussed in above method[15].

RESULTS

Percentage Yield of Extracts

% of SGME (*Sterculia guttata* methanolic extract) = Weight of extract / Weight of powder × 100
107 / 500 × 100 = 21.4

% of SGME (*Sterculia guttata* aqueous extract) = Weight of extract / Weight of powder × 100
107 / 500 × 100 = 13.1

Preliminary Phytochemical Screening

Preliminary phytochemical screening of SGME shows the presence of Glycoside, Saponin glycoside, Flavonoids, Carbohydrates, Tannin and Phenolic compounds. Presence of flavonoids in the extract may be responsible for anticonvulsant effect[16]. While the aqueous extract shows presence of Alkaloid, Glycoside, Saponin glycoside, Flavonoids, Amino acid, Carbohydrates and Tannin and Phenolic compounds. Phytochemical screening of *S.guttata* shows the presence of following constituents in methanolic as well as aqueous extract. (Table 1)

Acute Toxicity Study

Even at the dose of 5000 mg/kg no mice were killed so the different extracts of *Sterculia guttata* were found to be non toxic.

Anticonvulsant activity of SGME on PTZ-induced seizures

At the dose 500 mg/kg the SGME shows highly significant result by delaying the onset of convulsion by much margin. 300 mg/kg SGME also shows good significant results but less than 500 mg/kg. (Table 4)

Anticonvulsant activity of SGAE on PTZ-induced seizures

Aqueous extract shows significant anti-convulsant effect at 500 mg/kg. At 300 mg/kg the reduction in duration of convulsion and increase in onset convulsion was less. (Table 5)

Anticonvulsant activity of SGME on MES-induced seizures

All the models i.e. 100, 300, and 500 mg/kg were observed with reduction in all phases of convulsion in all doses (Table 2). All values expressed as mean ± SEM; n = 6 rats in each group, by one-way ANOVA followed by Dunnett'st- Test (compared with

control group) *$p < 0.05$ and **$p < 0.01$, i. p-intra peritoneally, p.o per oral.

Anticonvulsant activity of SGAE on MES-induced seizures

All the models i.e. 100, 300, and 500 mg/kg were observed with reduction in all phases of convulsion in all doses. As compare to methanolic extract, aqueous extract shows less significance. (Table 3)

Anticonvulsant activity of SGME on Strychnine induced seizures

At the dose 500 mg/kg there was significant delay in onset of convulsion while reduction in duration of convulsion was less significant. At the dose 300 mg/kg effect on onset of convulsion was less as compare to 500 mg/kg and reduction in duration of convulsion was almost same. (Table 6)

Anticonvulsant activity of SGAE on Strychnine induced seizures

At the dose of 300 mg/kg of SGAE, the significance of result was little less while at 500 mg/kg the significance was high. (Table 7)

Anticonvulsant activity of SGME on Picrotoxin induced seizures

The significance find was only at dose 500 mg/kg of SGME. It was less significant as compare to other models. (Table 8)

Anticonvulsant activity of SGAE on Picrotoxin induced seizures

The significance find was only at dose 500 mg/kg of SGAE. No significance was found at the dose 100 and 300 mg/kg. (Table 9)

Table 1: Different phytoconstituents of methanolic and aqueous extraction of *Sterculia guttata*

S. No	Tests	Methanol extract	Aqueous Extract
1.	Alkaloid	Absent	Present
2.	Glycoside	Present	Present
3.	Saponin glycoside	Present	Present
4.	Steroidal triterpenoid (Liberman and Salkowski)	Absent	Absent
5.	Flavonoids	Present	Present
6.	Amino acid	Absent	Present
7.	Carbohydrates	Present	Present
8.	Tannin and Phenolic compounds	Present	Present
9.	Fixed oils	Absent	Absent

Table 2: Anticonvulsant activity of SGME on MES-Induced Seizures

Group	Treatment	Number of Animals (n)	Duration of Convulsion (Time in Sec)			Death percentage
			Flexion	Extensor	Stupor	
Group-I	Control (0.9 % Normal saline p.o)	6	39 ± 0.32	64 ± 0.15	212 ± 0.95	4/6 (66.66)
Group-II	Standard Drug (Phenytoin 25 mg/kg i.p)	6	23 ± 0.44**	_	_	0/6 (0.00)
Group-III	SGME (100 mg/kg p.o)	6	32 ± 0.84	39 ± 0.85	142 ± 0.40	2/6 (33.33)
Group-IV	SGME (300 mg/kg p.o)	6	27 ± 0.46**	37 ± 0.84*	128 ± 0.97*	2/6 (33.33)
Group-V	SGME (500 mg/kg p.o)	6	26 ± 0.46**	31 ± 0.15**	124 ± 0.38**	1/6 (16.66)

Table 3: Anticonvulsant activity of SGAE on MES-Induced Seizures

Group	Treatment	Number of Animals (n)	Duration of Convulsion (Time in Sec)			Death percentage
			Flexion	Extensor	Stupor	
Group-I	Control (0.9 % Normal saline p.o)	6	39 ± 0.32	64 ± 0.15	212 ± 0.39	3/6 (50)
Group-II	Standard Drug(Phenytoin 25 mg/kg i.p)	6	23 ± 0.44**	_	_	0/6 (0.00)
Group-III	SGAE (100 mg/kg p.o)	6	33 ± 0.84	31 ± 0.84	147 ± 0.40	2/6 (33.33)
Group-IV	SGAE (300 mg/kg p.o)	6	32 ± 0.46*	32 ± 0.84	159 ± 0.58	1/6 (16.66)
Group-V	SGAE (500 mg/kg p.o)	6	29 ± 0.46*	34 ± 0.15*	170 ± 0.38	1/6 (16.66)

Table 4: Anticonvulsant activity of SGME on PTZ-Induced Seizures

Group	Treatment	Number of Animals	Onset of convulsion (sec)	Duration of Convulsion (sec)	Death percentage
Group-I	Control (Saline 0.9%, p.o)	6	5 ± 0.10	287 ± 0.03	5/6 (83.33)
Group-II	Standard Drug (Diazepam 4 mg/kg i.p)	6	236**	27***	0/6 (0.00)
Group-III	SGME (100 mg/kg p.o)	6	42 ± 0.48	237 ± 0.67	2/6 (33.33)
Group-IV	SGME (300 mg/kg p.o)	6	87 ± 0.50*	196 ± 0.54*	1/6 (16.66)
Group-V	SGME (500 mg/kg p.o)	6	159 ± 0.76***	127 ± 0.65**	1/6 (16.66)

Table 5: Anticonvulsant activity of SGAE on PTZ-Induced Seizures

Group	Treatment	Number of Animals	Onset of convulsion (sec)	Duration of Convulsion (sec)	Death percentage
Group-I	Control (Saline 0.9%, p.o)	6	5 ± 0.10	287 ± 0.03	5/6 (83.33)
Group-II	Standard (Diazepam 4 mg/kg i.p	6	236**	27**	0/6 (0.00)
Group-III	SGAE (100 mg/kg p.o)	6	91 ± 0.48	180 ± 0.67	3/6 (50.00)
Group IV	SGAE (300 mg/kg p.o)	6	145 ± 0.50*	128 ± 0.54*	2/6 (33.33)
Group V	SGAE (500 mg/kg p.o)	6	189 ± 0.76**	88 ± 0.65**	1/6 (16.66)

Table 6: Anticonvulsant activity of SGME on Strychnine Induced Seizures

S. No.	Group/Treatment	Onset of clonic seizures in min ± S. E.M.	Onset of tonic seizures in min ± S. E.M.	Death percentage
1	Control group (Normal Saline)	3.12 ± 0.2	3.13 ± 0.2	6/6 (100)
2	Standard group (Phenytoin 25mg/kg)	6.4 ± 0.5	8.4 ± 0.8	3/6 (50)
3	Test Group-I (S. guttata 100mg/kg)	2.1±0.3	3.5 ± 0.4	6/6 (100)
4	Test Group-II (S. guttata 300mg/kg)	4.5 ± 0.3*	4.6 ± 0.4*	5/6 (83.33)
5	Test Group-III (S. guttata 500mg/kg)	5.1 ± 0.4**	6.8 ± 0.7*	4/6 (66.66)

Table-7: Anticonvulsant activity of SGAE on Strychnine Induced Seizures

S. No.	Group/Treatment	Onset of clonic seizures in min ± S. E.M.	Onset of tonic seizures in min ± S. E.M.	Death percentage
1	Control group (Normal Saline)	3.2 ± 0.4	3.7 ± 0.4	6/6 (100)
2	Standard group (Phenytoin 25mg/kg)	8.5 ± 0.7	10.1 ± 0.9	2/6 (33.33)
3	Test Group-I (S. guttata 100mg/kg)	3.7±0.5	4.5 ± 0.6	6/6 (100)
4	Test Group-II (S. guttata 300mg/kg)	6.5 ± 0.4*	6.9 ± 0.7*	5/6 (83.33)
5	Test Group-III (S. guttata 500mg/kg)	7.3 ± 0.4**	8.01 ± 0.9**	4/6 (66.66)

Table 8: Anticonvulsant activity of SGME on Picrotoxin Induced Seizures

S. No.	Group/Treatment	Onset of clonic seizures in min ± S. E.M.	Onset of tonic seizures in min ± S. E.M.	Death percentage
1	Control group (Normal Saline)	6.7 ± 0.2	7.5 ±0.3	6/6 (100)
2	Standard group (Phenytoin 25mg/kg)	17.2 ± 1.7**	20.4 ± 2.8**	2/6 (33.33)
3	Test Group-I (S. guttata 100mg/kg)	10.6 ± 1.4	10.9 ± 1.7	6/6 (100)
4	Test Group-II (S. guttata 300mg/kg)	11.9 ± 1.4	11.6 ± 1.7	4/6 (66.66)
5	Test Group-III (S. guttata 500mg/kg)	13.4 ± 1.2*	14.6 ± 1.7*	4/6 (66.66)

Table 9: Anticonvulsant activity of SGAE on Picrotoxin Induced Seizures

S. No.	Group/Treatment	Onset of clonic seizures in min ± S. E.M.	Onset of tonic seizures in min ± S. E.M.	Death percentage
1	Control group (Normal Saline)	10.5 ± 0.4	11.5 ±0.7	5/6 (83.33)
2	Standard group (Phenytoin 25mg/kg)	51.3 ± 1.9**	54.4 ± 3.2**	1/6 (16.66)
3	Test Group-I (S. guttata 100mg/kg)	14.6 ± 1.7	15.5 ± 2.1	6/6 (100)
4	Test Group-II (S. guttata 300mg/kg)	15.9 ± 1.8	17.9 ± 2.1	5/6 (83.33)
5	Test Group-III (S. guttata 500mg/kg)	27.4 ± 1.6*	28.6 ± 2.1*	5/6 (83.33)

DISCUSSION

MES

In MES induced seizure model the methanolic extract of *S. guttata* in doses of 300 mg/kg and 500 mg/kg body weight reduced all the phases of convulsion significantly ($p < 0.01$, $p < 0.05$), Aqueous extract of *S. guttata* in dose100 mg shows no significance while at dose 300 mg/kg and 500 mg/kg of body weight showed little significant activity (Table 3).

MES test is every now and then utilized model for studying anticonvulsant property of drugs for generalized tonic-clonic seizures "grand mal"epilepsy[17]. This model depends on perception of the stimulation by rehashed electrical pulses. The pharmacology of acute maximal electroshock dose not differs from the pharmacology of generalized tonic-clonic seizures in genetic models with chronic epilepsy[18]. It has often been stated that antiepileptic drugs that block MES-induced tonic extension act by blocking seizure spread. In addition, MES-induced tonic augmentation can be averted either by drugs that repress voltage-mediated Na + channels, for example, phenytoin, valproate, felbamate and lamotrigine or by drugs that prevent glutamatergic excitation by the N-methyl-D-aspartate (NMDA) receptor, such as phenytoin, valproate, felbamate and lamotrigine or and glutamatergic excitation through NMDA receptor[19]. Currently used anticonvulsant drugs (e.g. phenytoin, carbamazepine) are effective in therapy of generalized tonic-clonic and partial seizures have been found to show strong anticonvulsant action in MES test[20]. Thus the anticonvulsant activity exhibited by the extract shows that it could have blocked the seizure spread by inhibiting Na + channels and glutamatergic excitation through NMDA receptor.

The investigation likewise proposes that the extract would be compelling against generalized tonic-clonic and partial seizures. In the present study, it is found that treatment with SGME on mice significantly reduces in tonic hind-leg extensor stage in MES

induced epilepsy; the MES model – to identify compounds which prevent seizure spread, corresponding to generalized tonic-clonic seizures in humans. Since, SGME significantly inhibited generalized tonic-clonic seizures in MES test (*p < 0.05 and **p < 0.01). It may be suggested that level of significant i.e. may be increased by using more studies on it. A few medications are thought to restrain the seizures by directing GABA mediated synaptic restraint through an activity at particular locales of the neurotransmitter. Researchers are gaining new insight in to the traditional medicine in assisting the body to maintain its own self-healing systems while preventing debilitating effects of chronic diseases, like epilepsy. The results suggest that the methanol extract of the plant is useful in suppressing generalized tonic clonic seizures. Thus, methanolic extracts of the *S. guttata* possess anticonvulsant property against the MES in mice. However, the further research is in progress to isolate the compound responsible for activity.

PTZ

The present study investigated the anti-convulsant effect of methanol and aqueous extracts of leaves of the plant *S. guttata*. The extracts increase the onset of clonic seizures in PTZ models and it seems that this effect increased dose dependently.

In PTZ induced seizures the administration of methanol extract at the dose of 300 mg/kg and 500 mg/kg 30 min prior to injection of PTZ (significantly p < 0.01) delayed the onset of clonic seizure. Aqueous extract shows little significant activity. Standard drug diazepam at a dose (4 mg/kg) shows much delayed onset of clonic seizure. The number of mortalities was less at the dose 300 mg/kg and 500 mg/kg body weight as compared to the dose 100 mg/kg in both the extracts.

The traditional medicine in helping the body to keep up its very own self mending frameworks while counteracting weakening impacts of chronic seizure is practically equivalent to petitmal seizure and human generalized seizure[21]. The oral administration of *S. guttata* at the dose of 300 mg/kg, 500 mg/kg, 30 min. before the injection of PTZ, delayed the onset of seizures, and decreased the duration of seizures (**p < 0.01 and ***p < 0.001, Table 4). Drugs that are effective against petit mal seizures reduce T- type calcium currents and these types of seizures can also be prevented by drugs that enhance GABA – BZD receptor mediated neurotransmission such as benzodiazepines and phenobarbitone intervened neurotransmission, for example, benzodiazepines and phenobarbitone. Studies have demonstrated that activation of N-methyl D-aspartate receptor (NMDA) is likewise engaged with the inception and speculation of PTZ induced seizures. Drugs that prevent glutametargic excitation via NMDA receptors, for example, felbamate, have anticonvulsant property against PTZ induced seizures. Drugs that block glutametargic excitation mediated by NMDA receptors, such as felbamate, have anticonvulsant property against PTZ induced seizures. Phytochemicals such as ursolic acid, beta-amyrin and friedelin are the active principles responsible for the anticonvulsant activity of *S. guttata*; it is likely that triterpenoidal compounds, present in this plant may be involved in this action. Hence this drug may able to modulate the function of GABA or glutamate receptors.

Strychnine is a drug that induces seizures by inhibiting the glycine receptor[23]. Glycine acts as inhibitory neurotransmitter, reducing its activity increase the excitation of neurons thus induce convulsions. SGME at dose 300 and 500 mg/kg delayed the onset of tonic and clonic seizures both in strychnine induced seizures (*p < 0.05, **p < 0.01). SGAE shows little significance at the dose 500 mg/kg only.

Picrotoxin is a $GABA_A$ receptor antagonist and can mediate by both pre and post synaptic inhibition[24]. In picrotoxin induced model only 500 mg/kg shows significant effect but at very less extent (*p < 0.05).

CONCLUSION

It was concluded from the above investigation that the leaves of *Sterculia guttata* may be used as antiepileptic agent as the alcoholic extracts shows more significant result as compare to aqueous extract. The similar work was also done with alcoholic and aqueous extract of leaves of *Shorea robusta* that reported for its anticonvulsant activity[25]. This suggests the significance of *Sterculia guttata* as an antiepileptic agent.

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