JOURNAL OF NATURAL REMEDIES

Anticonvulsant Activity of *Bauhinia Purpurea Linn.* Leaves

Vishal D.Joshi[1]*, Tekeshwar Verma[2], Jayaraman R.[1], Prajwal R.Shetty[3], Sanjay R.Patel[1], Hitesh V. Hirapara[1]

1. K.N.V. Pharmacy College - Metoda, Rajkot, Gujarat, India.

2. Rungta College of Pharmaceutical Sciences, Bhilai, Chattisgrah

3. N.G.S.M. Institute of Pharmaceutical Sciences, Paneer, Deralakatte, Mangalore, India

Abstract

The Ethanolic extract of the leaves of *Bauhinia Purpurea Linn.* studied for anticonvulsant effect on maximum electroshock (MES) and Pentylenetetrazole (PTZ) induced seizure in albino male mice at 3 different dose levels (100 mg/kg , 250 mg/kg and 500 mg/kg i.p). The ethanolic leaves extract showed significant action ($p<0.001$) against both Maximal Electroshock (MES) and Pentylenetetrazole (PTZ) induced seizure.

Key words: *Bauhinia purpurea Linn*, Anticonvulsant activity, MES and PTZ

1. Introduction

Epilepsy is a major neurological disorder and up to 5% of the world population develops epilepsy in their lifetime.[1] Anepidemiological study suggests a prevalence of 6.8/1000 in the U.S.A.[2] It is probable that the prevalence is higher in less developed countries because of higher incidence of factors such as brain infections, cranial and prenatal traumas and parasitic Infection.[3] Although the prognosis for controlling seizures in most patients in terms of seizure control, remission and withdrawal of medication is good.[4] Many patients (20 - 30%) however, have seizures that are not adequately managed by the established antiepileptic drugs (AEDs).[5] Traditional systems of medicine are popular in developing countries and up to 80% of the population relies on traditional medicines or folk remedies for their primary health care need.[6] Several plants used for the treatment of epilepsy in different systems of traditional medicine have shown activity when tested in modern bioassays for the detection of anticonvulsant activity[7] and many such plants are yet to be scientifically investigated.

Bauhinia purpurea Linn (Leguminosae) is a medium-sized deciduous tree, sparingly grown in India. Traditionally this plant is used in the treatment of drowsy, pain, rheumatism,

* Corresponding author
Email: vishaljoshi646@gmail.com

convulsions, delirium, septicemia, etc.[8] *B. purpurea* has also been used in the treatment and control of hyperglycemia[9] and seems to possess laxative and astringent effects[10,11] Several types of bioactive compounds have been isolated from different parts of *B. purpurea*[12,13,14] particularly flavonoids, such as kaempferol, quercetin and isorhamnetin and pacharin and four new dibenz [b,f]oxepins (2a, 3–5), named bauhiniastatins 1–4 from the plant leaves. These active constituents have been attributed the therapeutic activity of the plant. Therefore, the present study was undertaken to evaluate their anticonvulsant activity.

2. Materials and Methods

2.1 Collection of Bauhinia Purpurea and preparation of the extract

Fresh leaves of *Bauhinia purpurea* were collected from Paneer, Deralakatte, Karnataka, India during Feb-March 2008 and its botanical identity was confirmed by Dr. Noeline J. Pinto, Head of Botany Department, St. Agnes College, Mangalore. A Voucher specimen was deposited in NGSM Institute of Pharmaceutical Sciences.

Collected leaves were shade dried. Dried leaves were then crushed in a coffee grinder. The powdered plant material was then soaked in ethanol (95%) and kept aside for four days. After four days the ethanol layer was decanted off, this process was repeated for four times. The solvent was distilled and concentrated on a water bath to a syrupy consistency and then evaporated to dryness to give a sticky greenish mass. The obtained Ethanolic extract of *Bauhinia purpurea* was then tested for anticonvulsant activity.

2.2 Phytochemical screening

The preliminary Phytochemical screening of crude extract of *Bauhinia purpurea* was carried out in order to ascertain the presence of its constituents utilizing standard conventional protocols.[15]

2.3 Experimental Animals

Albino male Swiss mice [22-25 gm] were obtained from the animal house of the NGSM Institute of Pharmaceutical Sciences, Paneer, Deralakatte, Karnataka, India. Animals had free access to food and water; food was withdrawn 8 hour before the experiments. All experimental protocols were reviewed and accepted by the Institutional Animal Ethical Committee (IAEC) prior to the initiation of the experiment.

2.4 Drugs and Chemicals

Pentylenetetrazol (Sigma, USA), Diazepam (Ranbaxy, India) and Phenytoin (Sun pharmaceuticals, India) were used in this study.

2.5 Toxicity evaluation test

The adult albino male mice of 6-8 week old with an average weight of 120-180g were used for the study. To determine the toxicity, a single oral administration of Ethanolic extract of *Bauhinia purpurea* in different doses (100,250 and 500 mg/kg) were administered to different groups of animals. Mortality and general behavior of the animals were observed periodically for next 48 hrs.

2.6 Assessment of Anticonvulsant activity

2.6.1 Electrically-induced Seizures

A total of 30 male albino mice (25-30gm) were used in this experiment. The mice were divided into Five group (n=6).

Group I received Vehicle

Group II received Phenytoin 25 mg/kg

Group III received 100 mg/kg p.o of EEBP suspended in Tween 80

Group IV received 250 mg/kg p.o of EEBP suspended in Tween 80

Group V received 500 mg/kg p.o of EEBP suspended in Tween 80

The maximal seizure pattern was induced by using electro Convulsiometer (Techno, India) with an alternating current (0.2s stimulus durations and 50 mA) delivered via ear-clip electrodes and the duration of tonic hind limb extensor was noted.[16,17,18,19,20] A drop of 0.9% saline solution was poured into each ear prior to placing the electrodes. The test compound EEBP were administered orally 45 min before being subjected to an electroshock. The animals were observed closely. The ability of the test drug to abolish or reduce the duration of tonic hind limb extensor phase was noted, with relative to control were recorded and calculated.

2.6.2 Pentylenetetrazol-induced Seizures

A total of 30 male mice were used in this experiment. The mice were divided into five groups (n=6)

Group I received Vehicle

Group II received Diazepam 2 mg/kg

Group III received 100 mg/kg p.o of EEBP suspended in Tween 80

Group IV received 250 mg/kg p.o of EEBP suspended in Tween 80

Group V received 500 mg/kg p.o of EEBP suspended in Tween 80

Mice were injected intraperitoneally (i.p) with PTZ at the dose of 105 mg/kg which was its CD97 (97% convulsive dose for the clonic phase) for the induction of chemo convulsions[21,22]. The test drug EEBP were administered orally 45min prior to each PTZ treatment. The animals were observed for next 30min for the development of Clonic seizures. The ability of EEBP to abolish or reduce such movements was selected as criteria to establish anticonvulsant activity of the drug.

2.6.3 Statistical analysis

Results was expressed as mean ± S.D the significance of difference in the response between treatment group and control was determined by one way analysis of variance (ANOVA) followed by Dunnett test $p<0.05$ was considered as statistically significant.

3.0 Results

3.1 Phytochemical screening

Phytochemical screening of the ethanolic extract showed the presence of flavonoids, saponins, steroids, cardiac glycosides, triterpenoids, phenolic compounds and tannins.

3.2 Acute toxicity

The orally administered ethanolic extract of EEBP does not cause any mortality among the entire group at various doses (100-1000 mg/kg). There is no significant alteration in behavior was observed.

3.3 Assessment of anticonvulsant activity

3.3.1 Maximal Electroshock Seizures

In MES, the duration of hind limb extension for control mice was 10.87 ± 0.77s whereas 100% mortality was observed. EEBP (100 mg/kg p.o) administered before electroshock lowered the convulsive threshold ($P<0.05$) when compared to control groups. But at a dose of (250, 500 mg/kg p.o) EEBP exhibited more significant protection ($P<0.001$) when compared with control groups (Table 1).

3.3.2 Pentylenetetrazole induced seizures

The onset of action, nature and severity of convulsion by means of duration of action was after treatment with PTZ. In animals treated with vehicle, clonic convulsions appeared were 7.92 ± 0.20s and mortality were observed in all the animals. The ethanolic extract of

Bauhinia Purpurea Linn. Leaves significantly and on a dose depended manner inhibited the duration of convulsions. At a dose of 500 mg/kg, EEBP exhibited significantly increased protection against PTZ induced convulsions. (Table 2).

Table 1. Effect of EEBP on MES induced Seizures in Mice

Treatment	Dose (mg/kg p.o)	Duration of Tonic Hind Limb Extensor Phase (Sec)		Mortality	% of protection
		Before treatment	After treatment		
Normal Saline	0.5 ml	10.75±0.84	10.87 ±0.77	6	0
Phenytoin	25	10.01±0.45	6.80±0.43	0	37.44%
EEBP	100	10.03 ±0.45	9.98±0.38*	2	8.18%
EEBP	250	10.19± 0.47	8.24±0.44**	2	24.19%
EEBP	500	10.14± 0.43	7.46±0.48**	0	31.37%

Values are expressed as mean ±S.D (n=6) * P<0.05, ** P<0.01 when compared to control groups

Table 2. Effect of EEBP on PTZ induced Seizures in Mice

Treatment	Dose(mg/kg p.o)	Clonic Convulsions		Mortality	% of protection
		Onset(Sec)	Duration(Min)		
Vehicle +PTZ	105 (i.p)	85.03±0.88	7.92±0.20	6	0
Diazepam	2	97.62±1.60	3.52±0.16	0	55.55%
EEBP	100	88.48±2.17	6.84±0.22**	3	13.63%
EEBP	250	93.56±2.32	5.86±0.15**	1	26.01%
EEBP	500	95.66±1.23	4.91±0.15**	0	38.00%

Values are expressed as mean ±S.D (n=6) ** P<0.01 when compared to control groups.

4. Discussion

The present results demonstrated that the EEBP administered orally, revealed anticonvulsant activity against MES or PTZ seizure models, in a dose dependent manner in mice. At higher doses, there was more significant protection against both the seizure model in mice were observed. Electroshock causes the inhibition of GABA release and this, in turn, may inhibit GABA synthesis[23]. It is therefore, possible that the ethanolic extract of *Bauhinia Purpurea* Leaves increases the release of GABA. It has been observed that an increase in catecholamines, will enhance anticonvulsant activity[24], also PTZ have been reported to induce seizures by inhibiting g-amino butyric acid (GABA) neurotransmission. So it is probable that the ethanolic extract of *Bauhinia Purpurea* may have some connection with in the cascade of events in neurohumoral transmission.

Summing up, Obtained results from our study demonstrated that the ethanolic extract of *Bauhinia Purpurea* Leaves possess apparent pharmacological activity towards experimental convulsions produced by MES and PTZ methods which deserves further investigations in order to elucidate the exact mechanism of the anticonvulsant activity of ethanolic extract of *Bauhinia Purpurea* Linn.

References

1. Sander JWAS, Shorvon SD. (1996) J Neurol Neurosurg Psychiatry.61: 433-443.
2. Hauser WA, Annegers JF, Kurland LT. (1991) Epilepsia.32: 429-445.
3. Sander JWA, Shorvon SD. (1987) A review journal of neurology, neurosurgery and psychiatry. 50: 829-839.
4. Goodridge DM, Shorvon SD. (1983) Brit Med J. 287: 645-647.
5. Richens A, Perucca E. (1993) Textbook of epilepsy Churchill Livingstone, Edinburgh: 495 – 559.
6. Akerele O. (1988) Fitoterapia. 355-363.
7. Raza M, Choudary MI, Atta-ur-Rahman. (1999) Studies in Natural Product Chemistry, Elsevier Science Publishers : Netherlands; 507-553.
8. Asolker LV,Kakkar KK, Chakre OJ. (2000) Supplement to Glossary of Indian Medicinal Plants, New Delhi: 116-7.
9. Salatino A, Blatt CTT, Dos Santos DYAC, Vaz AMSF.(1999) Rev Bras Botan. 22: 17–20.
10. Jones DT. (1993) Flora of Malaysia (Illustrated).Oxford, Oxford University Press: 49–50.
11. Lanka Chronicle: Kobo-leela (Bauhinia purpurea).
12. Salatino A, Blatt CTT, Dos Santos DYAC, Vaz AMSF. (1999) Rev Bras Botan. 22: 17–20.
13. Ramadana MF, Sharanabasappab G, Seetharamb YN, Seshagiric M, Moerseld JT. (2006) Food Chem. 98: 359–365.
14. Yadava RN, Tripathi P. (2000) Fitoterapia. 71: 88–90.
15. Kokate CK, Purohit AP, Gokhale SB. (2006) Pharmacognosy, Niraliprakasan; 593-597.
16. Eadie MJ, Tyrer JH. (1974) Pharmacological basis and practice, Churchill Livingston: London; 101-107.
17. Losher W, Schmidt D.(1988) J epilepsy Res. 2 : 145-181.
18. Perucca E, Richens A. (1981) Antiepileptic drugs: Clinical aspects. In: Therapeutic drug monitoring, Churchill Livingston: London ; 320-348.
19. Swinyard EA, Brown WC, Goodman LS. (1952) J Pharmacol Exp Ther. 106: 319-330.
20. Toman JEP, Guy M, Laurence DR, Bacharach AL. (1964) Pharmacometics.1:290.
21. Sanjay NM, Keerthikumar A, Rajamannar T. (2007) Seizure. 16 : 636-644.
22. Turner RA. (1965) Anticonvulsants. In: screening methods in pharmacology, Academics Press: 64-65.
23. Serme E, Gregoire MC, Galy G, Lavenne F, Pierre C, Veyre L, Lebras D, Cinotti L, Comar D, Dalery J, Bobilier P. (1998) Neurosci Lett. 254: 41 – 44.
24. Jobe PC, Stull RE, Geige PF.(1974) Neuropharmacol. 13: 961-968.