PHYTOCHEMICAL SCREENING ANTIEPILEPTIC& ANALGESIC ACTIVITY OF LEAF EXTRACT OF PASSIFLORA FOETIDA

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Summary

Antiepileptic activity of passiflora foetida was evaluated in mice against maximum electroshock induced convulsions and pentylenetetrazole induced covulsions.Screening of analgesic activity was evaluated using Eddy's hot plate method using aspirin as standard.Antispamodic activity was evaluated in mice using Diazepam as standard. Hypnotic activity was evaluated using thiopentone as standard.

Key words: Passiflora foetida, Antiepileptic activity, Analgesic activity

Introduction

Passiflora foetida is a perennial creeping vine which has an edible fruit and leaves that have a mild rank aroma. Literature study reveals it has been used traditionally in various countries for treatment of stress, anxiety, depression, insomnia, cardiac arrhythmia and tension related asthma (1-2). Passiflora edulis sims possesses antibacterial, cytotoxic and anti-oxidant activity (3-4).Leshmanicidal activity of passifloricin A and derivatives were reported from the resin extract of passiflora foetida (5).Benzoflavone moiety present in passiflora incarnate were responsible for antiaxiety activity(6).Several HPLC methods has been employed for qualitative and quantitative analysis of C-glycosylflavones in P.incarnata(7).The active constituents of Passiflora foetida are hydrocyanic acid, groups of flavone alkaloids, harman alkaloids ,alkaloids, phenols, cyanogenic compounds , passifloricins,polyketides and alpha-pyrones(8-10).The present study was undertaken to investigate antiepileptic and analgesic activity of methanol extract of passiflora foetida.

Materials and methods

Dried leaves of Passiflora foetida were obtained from Kotla vijaybhaskar reddy botanical garden, Hyderabad Andhra Pradesh, India and authenticated by Pranali Pandit, pharmacognist, CM College of pharmacy, Hyderabad. A voucher specimen has been deposited at the museum of our college. The leaves of passiflora foetida were air-dried. The leaves were subjected to soxhlet extraction using different methanol for 48 hrs.The extract was concentrated using rotary vacuum to get the solid mass. The phytochemical investigation of various extracts are represented in Table-1, yield color and consistency of various extracts are represented in Table-2

TABLE-1

Extract	Constituents investigated
Petroleum ether	Carbohydrates , flavanoids
Chloroform	Carbohydrates,alkaloids,flavanoids,glycosides
Methanol	Carbohydrates,tannins,triterpenes,flavanoids,glycosides
Hydroalcoholic	Saponins,glycosides,alkaloids,carbohydrates,triterpenes

TABLE-2

Extract	Yield(% W /W)	Co lour	consistency
Petroleum ether	5.0	Reddish brown	Hard waxy
Chloroform	3.0	Green	Waxy
Methanol	20	Dark brown	Resinous
Hydro alcoholic	13.2	Reddish brown	Resinous

Antiepileptic activity

Swiss albino mice male weighing 20-25 g were housed in groups of six under standard lab conditions (temperature 25^0C $\pm 1^0$C, relative humidity 55 ± 5% , 12h:12h dark: light cycle) with standard pellet food and water ad libitum.The animals were transferred to the laboratory atleast 1 hr before the start of the experiment. The experiments were performed during the day and as per the guidelines of the Committee for the purpose of supervision and control of Experiments on Animals (CPSCEA), Government of India.

Maximum electroshock (MES) –induced convulsions

Mice were divided into 5 groups (n=6).Group I served as control ad received only vehicle. Groups II, III and IV were treated orally with PG at a dose 30, 100 and 300 mg/kg body weight. Group V received 20 mg/Kg i.p phenytoin.The mice received a current of 50mA for 0.2 sec duration through electroconvulsiometer using ear electrodes after 60 min of oral administration of PG and 30 min after i.p administration of phenytoin.The incidence and duration of extensor tonus was noted. A complete abolition of hind limb tonic extension was considered as 100% protection (9). The results were reported in Table-2.

Pentylenetetrazol (PTZ)-induced convulsions

Five groups of mice (n=6) were treated with 80mg/kg s.c PTZ, 60 min after oral administration of either PG (30, 100 and 300 mg/kg) or vehicle or 30 min after i.p administration of diazepam (4mg/kg).The animals were observed for 30 min for onset, presence or absence of clonic convulsions and mortality(9). The results were reported in Table-3.

TABLE-2
Effect of methanol extract and phenytoin against MES-induced convulsions

Groups	Duration of hind limb Extension (Sec) Mean±SEM	n=6	Death P (Dunn's test)
Control	13.3±0.2	5/6	
Passiflora methanol extract (30mg/kg.b.wt)	12.5±0.6	3/6	>0.05
Passiflora methanol extract (100mg/kg.b.wt)	5.0±0.4	0/6	<0.01
Passiflora methanol extract(300mg/kg.b.wt)	5.2±0.2	0/6	<0.01
Phenytoin	4.0±1.0	0/6	<0.01

The data were analyzed by kruskal-Wallis test KW =22.26 (non parametric ANOVA, P=0.0002, from chi square distribution) followed by Dunn's multiple comparison test.

TABLE-3
Effect of methanol extract and Diazepam against PTZ-Induced convulsions

Groups	Onset of myoclonic spasm(sec) Mean±SEM	Onset of clonic convulsions(sec) Mean±SEM	Death P n=6
Control	68.25 ±0.2	105±0.6	5/6
Passiflora methanol extract (30mg/kg.b.wt)	119±12.8	164±20.0	4/6
Passiflora methanol extract (100mg/kg.b.wt)	254±1.2	220±22.0	5/6
Passiflora methanol extract(300mg/kg.b.wt)	A	A	0/6
Diazepam	A	A	0/6

The data were analyzed by krushkal-Wallis test KW= 14.126(non parametric ANOVA, P= 0.0005, from chi square distribution) followed by Dunn's multiple comparison test P. 0.05 (Not significant) p< 0.001(Highly significant) A-Absence of any seizure activity.

Analgesic activity

Screening of analgesic activity was carried in animal models which showed reaction time 3-5 seconds (10). By using Eddy's hot plate method screening of analgesic activity is carried, the animals used are albino mice of either sex weighing 25-30 gm. Mice were divided into 4 groups of sex and tested for 4 hours. Group I received Tween-80 1% i.p and served as control. Group II received Analgin and served as standard. Group III & IV received the methanol extract of passiflora foetida 50 mg/kg and 100mg/kg (i.p).The observations were made at 30 min interval upto 4 hours. The results are shown in Table-4

TABLE-4
Analgesic activity of methanol extract of passiflora foetida

Group	Reaction time in seconds					
	0 min	**30 min**	**60 min**	**120 min**	**180 min**	**240 min**
I	5.96±0.1	5.56±0.0	3.51±0.21	3.24±0.10	3.62±0.24	3.82±0.30
II	5.48±0.1	14.25±0.2	15.11±0.5	16.50±0.2	17.20±0.2	5.5±0.92
III	5.10±0.1	11.40±0.1	12.26. ±0.1	14.87±2.6	16.87±1.0	7.9±0.2
IV	5.50±0.1	5.84±0.6	5.12±0.2	3.2±0.1	5.62±0.2	4.93±1.2

P<0.001 vs. control student t test

Results and Discussion

Antiepileptic activity was evaluated using Maximum electricshock induced convulsions and pentylenetetrazol-induced convulsions .Doses at 30 mg/kg body wt did not show significant anticonvulsant activity. Doses at 100 and 300 mg/kg significantly reduced the severity of seizures when compared to standard drug phenytoin, in MES induced convulsions, but in the case of pentylenetetrazole induced convulsions, the convulsions were completely abolished at doses 100mg/kg & 300 mg/kg b.wt when compared to standard drug diazepam. The methanol extract of leaves of passiflora foetida showed antiepileptic activity in a dose dependant manner. The detailed pharmacological activity have to be investigated whether it is due to inhibiting Na+ channels/Ca++ channels, GABA/Beznzodiazepine, NMDA receptor complex. The extract of passiflora foetida may be effective in generalized tonic seizures and absence seizures. Detailed EEG (Electroencephalogram) discharge studies for different convulsions whether it due to imbalance between which excitatory and inhibitory influences in the brain should be studied for the extracts of passiflora foetida.

Analgesic activity was evaluated using Eddy's hot plate method. The methanol extract of passiflora foetida exhibited maximum analgesic activity at 120 min.In the present study the methanol extract showed good analgesic activity at 100mg/kg when compared to 50mg/kg. The mode of action of analgesics is on peripheral or central nervous system. Any damage to tissue or injury leads to pain and inflammation .opiod analgesics produce analgesia by binding to specific G protein coupled receptors that are located in brain and spinal cord regions involved in the transmission and modulation of pain.

Peripherally acting analgesics act by blocking the generation of impulses at chemoreceptor pain site, but centrally acting analgesics not only raise the threshold for pain but also alter the physiologic response to pain and suppress the patient's anxiety and apprehension. The pharmacological action of extract may be peripherally. The detailed investigation of mechanism of action of drug should be studied. The isolation of active principle from the crude drug responsible for anticonvulsant and antiepileptic activity should be studied.

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