

**Research Article****EVALUATION OF ANTI CATALEPTIC ACTIVITY FOR METHANOLIC EXTRACT OF *AERVA LANATA* WHOLE PLANT**Sneha Nawale^{1*}, P. Pranusha¹, K. Padma Priya¹, M. Ganga Raju²¹ Department of Pharmacognosy, Gokaraju Rangaraju College of Pharmacy, Bachupally, Hyderabad-500090, Telangana, INDIA.² Department of Pharmacology, Gokaraju Rangaraju College of Pharmacy, Bachupally, Hyderabad-500090, Telangana, INDIA.

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ABSTRACT

The present study was done to evaluate the anti-cataleptic activity for methanolic extract of *Aerva lanata* whole plant (MEAL) in haloperidol induced catalepsy (HIC) and clonidine induced catalepsy (CIC) in mice. Catalepsy was induced by administering haloperidol (1.0 mg/kg bd.wt, i.p.) and clonidine (0.5 mg/kg, bd.wt, s.c.) daily for 21 days. The anti-cataleptic effect of *Aerva lanata* was evaluated by standard bar test, locomotor activity, rota rod test, and cook's pole climbing test in mice models. Results were analyzed by one-way ANOVA followed by Dunnett's test. MEAL (200 and 400 mg/kg bd.wt, p.o.) was found to decrease the duration of catalepsy significantly ($P < 0.01$) in standard bar test, significantly increases ($P < 0.01$) basal activity score in locomotor activity and fall-off time in rota rod test respectively, when results were compared to clonidine group in CIC. MEAL (200 mg/kg bd.wt, p.o.) showed more potent anti-cataleptic activity than MEAL (400 mg/kg bd.wt, p.o.). Anti-cataleptic activity of MEAL might be due to presence of flavonoids, alkaloids and other secondary metabolites. Further studies are proposed to prove the anti-cataleptic activity with isolated secondary metabolites at receptor levels.

KEYWORDS: Catalepsy; *Aerva lanata*; Amaranthaceae; Haloperidol; Clonidine; Levodopa; Carbidopa.**INTRODUCTION**

Central nervous system (CNS) is mainly associated with various diseases which are appearing as a major threat due to increasing mental stress, strain and work load, in developing countries may leads to CNS disorders. The present anti-parkinson's drugs encounter many side effects so there is need for prolonged treatment including efficacy in the treatment. So, these reasons force the area of research to find new and improved treatments which will encounter the adverse effects and drawbacks of the existing treatments.

Various pathological conditions like cancer, rheumatoid arthritis, Alzheimer's disease, diabetes mellitus, ischemia, atherosclerosis and parkinson's disease have etiological relation to reactive oxygen species (ROS) induced and free radical mediated oxidation of biomolecules, which take place in conditions with inadequate antioxidant defense stress. Inhibition of the initial cellular damage caused by ROS species has been the focused on intense investigation and resulted in

the discovery of several naturally occurring drugs, which have been attributed as potent antioxidants^[1].

Catalepsy is a sign of extrapyramidal effect of drugs that inhibit dopaminergic transmission or increase histamine release in brain and a condition where the animal maintains imposed posture for long time before regaining the normal posture^[2]. Catalepsy induced by neuroleptics may be due to a blockade of dopaminergic neurotransmission in the striatum. Neuroleptic-induced catalepsy is another animal model for screening drugs for Parkinsonism. The catalepsy test is generally used to evaluate motor effects of drugs which act on the extrapyramidal system. Evidence suggests enormous oxidative stress, free radical formation, genetic susceptibility, and programmed cell death that causes neurodegeneration associated with parkinson's and other associated diseases^[3].

Haloperidol induced catalepsy occurs due to the blockade of dopamine (D₂) receptors and decreased dopaminergic transmission. The cataleptic immobility induced in rodents by typical neuroleptics (e.g. haloperidol) is a strong behavioural model to study nigrostriatal function and its modulation by cholinergic, serotonergic, nitrenergic and other neurotransmitter systems^[4].

The present study was done to evaluate the anti-cataleptic activity of *Aerva lanata* whole plant in haloperidol induced catalepsy and clonidine induced catalepsy models in mice.

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MATERIALS AND METHODS

Plant collection & drying:

The whole plant of *Aerva lanata* Linn. was collected from Rangareddy district, Telangana in the month of December. The plant specimen was authenticated from Government Degree college, Kukatpally, Hyderabad by Dr. R. Lalitha (Associate Prof.) with Voucher specimen no., ALP-4). The crude plant material was dried and size reduced to coarsely powdered in pulverizer. The powdered crude material was stored for further extraction process.

Preparation of methanolic extract of *Aerva lanata* (MEAL):

The powdered plant material was extracted by simple distillation process with methanol. The plant material was suspended in the round bottomed flask containing the extraction solvent. This was subsequently equipped with a condenser. The flask was then heated, and the extract gets into the solvent. At the end of the extraction process, extract was filtered, and the filtrate was evaporated to solid mass. The solid extract was kept in desiccators to remove the moisture, if present, and finally stored in air tight container for further use.

***In vitro* antioxidant assay:**

H₂O₂ radical scavenging assay and reducing power assay was performed based on method given by Saumya SM *et al.*, 2010 and Manmohan Singhal *et al.*, 2011 [5,6].

Animals:

Swiss albino mice (20–25 g) were used for pharmacological activities. They were kept in polypropylene cages at 25 ± 2°C, with relative humidity 45-55% under 12h light and dark cycles. All the animals were acclimatized to the laboratory conditions for a week before use. They were feed with standard animal feed and water *ad libitum*. The animal studies were approved by the Institutional Animal Ethics Committee (Reg. No.1175/ac/08/CPCSEA), New Delhi, India.

Drugs and chemicals:

Levodopa and carbidopa was obtained from Sun pharma laboratories, haloperidol was procured from RPG life sciences Ltd and clonidine was procured from Unichem laboratories.

Acute toxicity studies:

Acute toxicity study was performed as per organization for economic cooperation and development (OECD) guidelines no. 425. The mice were fasted overnight, providing only water after which the extract was administered to the respective groups orally at the dose level of 2000 mg/kg body weight through gastric intubation and all groups were observed for 24h for behavioral, neurological and autonomic profiles, and then at 24h and 72h for any signs of toxicity. The animals were further observed for toxic symptoms for 14 days [7].

Methodology:***Grouping of animals for catalepsy induced by haloperidol:***

Haloperidol (1.0 mg/kg bd.wt, *i.p.*) was given to the mice daily for a period of 21 days to induce catalepsy. MEAL and standard drugs were given 30 mins before haloperidol through oral route. The animals were divided into six groups, which contain 6 animals in each group.

Group I: The animals serve as normal group.

Group II: The animals were administered with haloperidol (1.0 mg/kg, *i.p.*) which serves as negative control.

Group III: The animals were administered with haloperidol (1.0 mg/kg, *i.p.*) and then treated with MEAL (200 mg/kg bd.wt., *p.o.*) which is suspended in vehicle.

Group IV: The animals were administered with haloperidol (1.0 mg/kg bd.wt., *i.p.*) and then further treated with MEAL (400 mg/kg bd.wt., *p.o.*) which is suspended in vehicle.

Group V: The animals were administered with haloperidol (1.0 mg/kg bd.wt., *i.p.*) and then treated with L-DOPA + carbidopa (100 + 25 mg/kg, *p.o.*) and MEAL (400 mg/kg, *p.o.*) which is suspended in vehicle.

Group VI: The animals were administered with haloperidol (1.0 mg/kg bd.wt., *i.p.*) and then treated with L-DOPA+ carbidopa (100 + 25 mg/kg, *p.o.*) which are suspended in vehicle.

The animals were allowed to adjust in the box for 2 mins. A cataleptic behaviour was measured with a "standard bar test method". The standard (L-dopa + carbidopa) and test drugs were administered orally, 30 mins before to the haloperidol. Catalepsy score was measured for each hour upto 4h after haloperidol administration, by gently placing both the forepaws of the mouse over a wooden bar which is in 1 cm diameter and 4 cm height above the table top. The intensity of catalepsy was assessed by counting time in seconds until the mouse steps down to the table top, with a maximum cut-off time of 180s [8]. On last day of the experiment, and the animals were sacrificed for biochemical studies.

Grouping of animals for catalepsy induced by clonidine:

Clonidine (0.5 mg/kg bd. wt, *s.c.*) was given to the mice daily for a period of 21 days to induce catalepsy. MEAL and standard drugs were given 30 mins before clonidine through oral route. The animals were divided into six groups, which contain 6 animals in each group.

Group I: The animals serve as normal group.

Group II: The animals were administered with clonidine (0.5 mg/kg, *s.c.*) and which serves as negative control.

Group III: The animals were administered with clonidine (0.5 mg/kg, *s.c.*) and then treated with MEAL (200 mg/kg bd.wt, *p.o.*) which is suspended in vehicle.

Group IV: The animals were administered with clonidine (0.5 mg/kg bd.wt, *s.c.*) and then treated with MEAL (400 mg/kg bd.wt, *p.o.*) which is suspended in vehicle.

Group V: The animals were administered with clonidine (0.5 mg/kg bd.wt, *s.c.*) and then treated with L-DOPA+ carbidopa (100 + 25 mg/kg, *p.o.*) and MEAL (400 mg/kg bd.wt, *p.o.*) which is suspended in vehicle.

Group VI: The animals were administered with clonidine (0.5 mg/kg, *s.c.*) and then treated with L-DOPA+ carbidopa (100 + 25 mg/kg, *p.o.*) which is suspended in vehicle.

In vivo pharmacological studies were carried out on 0th day, 7th day, 14th day and 21st day of the experiment, and then the animals were sacrificed for biochemical parameters.

The animals were allowed to adjust in the box for 2 mins. A cataleptic behaviour was measured with a "standard bar test method". The standard (L-dopa + carbidopa) and test drugs were administered orally, 30 mins before to the haloperidol. Catalepsy score was measured for each hour upto 4h after haloperidol administration, by gently placing both the forepaws of the mouse over a wooden bar which is in 1 cm diameter and 4 cm height above the table top. The intensity of catalepsy was assessed by counting time in seconds until the mouse steps down to the table top, with a maximum cut-off time of 180s [8]. On last day of the experiment, and the animals were sacrificed for biochemical studies

***In vivo* models:**

Effect of MEAL on haloperidol induced catalepsy in mice (standard bar test):

The effect of test and standard drugs on haloperidol induced catalepsy was studied by the following method.

Albino mice of either sex weighing 20-25 g was divided into six groups of six animals each (n=6). The animals were allowed to adjust in the box for 2 mins. A cataleptic behaviour was measured with a "standard bar test method". The standard (L-dopa + carbidopa) and test drugs were administered orally, 30 mins before to the haloperidol. Catalepsy score was measured for each hour upto 4h after haloperidol administration, by gently placing both the forepaws of the mouse over a wooden bar which is in 1 cm diameter and 4 cm height above the table top. The intensity of catalepsy was assessed by counting time in seconds until the mouse steps down to the table top, with a maximum cut-off time of 180s [8].

Effect of MEAL and standard drugs (L-DOPA+ carbidopa) on locomotor activity:

The effect of drugs on locomotor activity was measured for 10 mins at every 30 mins upto 3 h with actophotometer. Actophotometer operates on photoelectric cells that are collected in circuit with a counter. When the beam of light falls on the photocell the cut-off by animal is counted and recorded. An actophotometer will be either in circular or square arena in which animal moves [1].

Effect of MEAL and standard drugs (L-DOPA + carbidopa) on rota rod apparatus:

All the groups were previously trained to stay on the rod rotating at the speed of 20 rpm for about 5 mins and in the next day all the groups were treated with particular doses and were challenged with clonidine and levodopa + carbidopa according to the protocol except the normal group and the time taken to fall from the rotating rod was noted for each animal [9].

Effect of the MEAL and standard drugs (L-DOPA + carbidopa) on cook's pole climbing apparatus:

All the groups are trained for about 10 days to accomplish attainment change (95-99%), by placing them inside the perspex chamber of the apparatus. After an adapted period of 5 mins to the chamber, a buzzer was given followed by a

shock through the grid floor. The mice will jump on the pole (shock free zone) to avoid foot shock, jumping on pole terminates the shock and this was classified as an escape while such jumping prior to onset of the shock was considered as avoidance. All the groups were treated with particular doses of MEAL & all the groups were challenged with clonidine and levodopa + carbidopa according to the protocol except the normal group and conditioned avoidance response was observed [10].

Statistical analysis:

The statistical analysis was carried out through analysis of variance (ANOVA), followed by Dunnett's test. P values < 0.05 were considered as significant [11].

Histopathological Studies:

One animal of each group was taken and euthanized at the end of the experiment. Brain was isolated from these animals by opening the cranium carefully and the isolated brains were placed in the containers which contain 4% formalin and then sent for histopathological studies.

RESULTS

Extractive value of MEAL was found to be 6.15% w/w. Phytochemical screening of MEAL revealed the presence of flavonoids, steroids, terpenoids, tannins and alkaloids.

***In vitro* antioxidant assay:**

MEAL had shown good H₂O₂ radical scavenging activity with IC₅₀ 15 µg/mL as compared with standard (ascorbic acid) IC₅₀ 13.5µg/mL and also shown good reducing capability by reducing power assay with IC₅₀ 28 µg/mL as compared with standard (ascorbic acid) IC₅₀ 16.5 µg/mL.

Acute oral toxicity study:

MEAL was tested on female mice at the dose of 2000 mg/kg *bd. wt. p.o.* and did not exhibit any signs of toxicity and mortality even upto 2000 mg/kg. *bd. wt.* All animals were safe even after 14 days of observation. The result revealed that the essential organs were not adversely affected throughout the treatment as seen in the gross observation of systemic organ of both control and treated groups. It indicates that MEAL was not lethal to the mice at 2000 mg/kg doses. Hence, 1/10th (200 mg/kg *bd.wt., p.o.*) and 1/5th (400 mg/kg *bd.wt., p.o.*) of the doses were selected for the further study.

***In vivo* models:**

Effect of MEAL on standard bar test:

The cataleptic scores after haloperidol administration were shown in fig 1. MEAL at a dose of 200 mg/kg *bd.wt.* showed prominent anti-cataleptic activity comparable to standard group. There was a significant difference (P < 0.01) between Group (II) and MEAL treated groups (III, IV, V) in catalepsy. MEAL at a dose of 200 mg/kg *bd.wt.*, showed good anti-cataleptic activity at 30, 60, 90, 120, 180 and 240 mins as compare to 400 mg/kg *bd.wt.* after haloperidol challenge. The effect of MEAL on haloperidol induced catalepsy by standard bar test was shown in Table 1.

The effect of MEAL on locomotor activity:

The changes in locomotor activity after haloperidol administration were shown in fig. 2. MEAL at a dose of 200 mg/kg *bd. wt.* showed anti-cataleptic activity comparable to standard. There was a significant difference (P < 0.01) between

negative Group (II) and MEAL treated Groups (III and V) in locomotor activity and MEAL treated Group IV showed significant ($P < 0.01$) anti-cataleptic activity at 30, 60, 90, 120 and 180 mins after haloperidol challenge. The effect of MEAL on haloperidol induced catalepsy by actophotometer (locomotor activity) was shown in Table 2.

Effect of MEAL on rota rod test:

The changes in fall-off time after clonidine administration are shown in fig. 3. MEAL at a dose of 200 mg/kg showed significant anti-cataleptic activity as that of standard drug treatment. There was a significant difference ($P < 0.01$) between negative group (II) and MEAL treated Groups (III, IV) in fall-off time on 7th, 14thday and 21stday and MEAL treated group V showed significant ($P < 0.01$) anti-cataleptic activity on 21stday after clonidine challenge. The effect of MEAL on

clonidine induced catalepsy by rota rod performance test was shown in Table 3.

Effect of MEAL on cook's pole climbing test:

The changes in pole climbing time after clonidine administration are shown in fig 4. MEAL at a dose of 200 mg/kg showed significant anti-cataleptic activity comparable to negative group ($P < 0.01$). MEAL at a dose of 200 mg/kg bd.wt. showed anti-cataleptic activity comparable to standard drug treatment. There was a significant difference ($P < 0.01$) in time between negative group (II) and MEAL treated groups (III, IV, V) in pole climbing time on 7thday, 14thday and 21stday after clonidine challenge. The effect of MEAL on clonidine induced catalepsy by cook's pole climbing test was shown in Table 4 and histopathological study was represented in fig. 1 (Fig-a to Fig-f).

Table No. 1: Effect of MEAL on haloperidol induced catalepsy by standard bar test

Groups	Cataleptic Score (in secs)					
	30 min	60 min	90 min	120 min	180 min	240 min
Group I	16.83 ± 1.64	16.16 ± 1.99	16 ± 1.88	15.33 ± 1.77	16.33 ± 1.23	13.83 ± 1.64
Group II	164.66 ± 2.19 a,*	171.5 ± 1.95 a,*	174 ± 2.10 a,*	181.83 ± 1.81 a,*	198.66 ± 1.36 a,*	167.5 ± 1.34 a,*
Group III	164.33 ± 1.70 a,*	157.66 ± 1.20 a,*,#	155.5 ± 1.11 a,*,#	149.16 ± 1.25 a,*,#	141.83 ± 1.81 a,*,#	161.83 ± 1.25 a,*
Group IV	148.33 ± 1.23 a,*,#	143 ± 1.06 a,*,#	136.16 ± 1.25 b,*,#	125.33 ± 1.36 a,*,#	124.5 ± 1.34 a,*,#	144.33 ± 2.23 a,*,#
Group V	156.33 ± 1.23 a,*,#	160 ± 1.31 a,*,#	168.33 ± 1.14 b,*	176.66 ± 1.05 a,*	175.16 ± 1.30 a,*,#	168.66 ± 1.45 a,*
Group VI	139.83 ± 1.42 *,#	135.66 ± 1.11 *,#	129.33 ± 1.36 *,#	118.16 ± 1.47 *,#	117 ± 1.53 *,#	126.5 ± 1.52 *,#

Values are expressed as Mean ± SEM, (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett's test. Results were compared with control group (* = $p < 0.01$), disease control (# = $p < 0.01$) and standard (a = $p < 0.01$, b = $p < 0.05$).

Table No. 2: Effect of MEAL on haloperidol induced catalepsy by actophotometer

Groups	Basal activity score (no. of movements)					
	30 min	60 min	90 min	120 min	180 min	240 min
Group I	312.66 ± 1.14	318.33 ± 1.23	326 ± 1.29	330.16 ± 1.60	332.66 ± 1.23	335 ± 1.18
Group II	262.16 ± 1.16 a,*	258 ± 1.24 a,*	246.33 ± 1.16 a,*	227.5 ± 1.18 a,*	231.66 ± 1.23 a,*	235.66 ± 1.23 a,*
Group III	270.33 ± 1.28 a,*,#	284.5 ± 1.11 a,*,#	294.66 ± 1.05 a,*,#	312.83 ± 1.56 a,*,#	308 ± 1.41 a,*,#	304.16 ± 1.42 a,*,#
Group IV	285.83 ± 1.07 a,*,#	296.33 ± 1.36 a,*,#	303.5 ± 1.47 a,*,#	315 ± 1.31 a,*,#	312.66 ± 1.36 a,*	310.5 ± 1.61 a,*
Group V	274.16 ± 1.45 a,*,#	267.83 ± 1.07 a,*,#	260.5 ± 0.76 a,*,#	241.66 ± 1.14 a,*,#	247 ± 1.18 a,*,#	252.33 ± 1.70 a,*,#
Group VI	292.33 ± 1.36 *,#	305.83 ± 1.30 *,#	312.16 ± 1.52 *,#	323.33 ± 1.36 *,#	315.16 ± 1.42 *,#	313 ± 1.24 *,#

Values are expressed as Mean ± SEM, (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett's test. Results were compared with control group (* = $p < 0.01$), disease control (# = $p < 0.01$) and standard (a = $p < 0.01$).

Table No. 3: Effect of MEAL on clonidine induced catalepsy by rota rod performance test

Groups	Fall- off time (in secs)			
	0th day	7th day	14th day	21st day
Group I	115.16 ± 1.07	115.3 ± 1.02	115.5 ± 1.23	115.83 ± 1.13
Group II	116.66 ± 1.14	61.83 ± 1.07 a,*	58.66 ± 1.28 a,*	46.83 ± 1.35 a,*
Group III	117.83 ± 1.07	64.16 ± 1.25 a,*	70.5 ± 1.43 a,*,#	85.5 ± 1.26 a,*,#
Group IV	116.33 ± 1.23	64 ± 1.31 a,*	70.33 ± 1.23 a,*,#	85.16 ± 1.25 a,*,#
Group V	114.5 ± 1.26	62.16 ± 1.45 a,*	60.83 ± 1.13 a,*	56.5 ± 1.26 a,*,#
Group VI	113.83 ± 1.07	74.16 ± 1.45 *,#	88.83 ± 1.13 *,#	93.5 ± 1.26 *,#

Values are expressed as Mean ± SEM, (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett's test. Results were compared with control group (* = $p < 0.01$), disease control (# = $p < 0.01$) and standard (a = $p < 0.01$).

Table No. 4: Effect of MEAL on transfer latency (Cook's Pole Climbing test)

Groups	Time taken to climb the pole (in secs)			
	0th day	7th day	14th day	21st day
Group I	59 ± 1.46	61 ± 1.57	63 ± 1.36	60 ± 1.75
Group II	58 ± 1.31	94 ± 1.53 a,*	101 ± 1.41 a,*	108 ± 1.88 a,*
Group III	60 ± 1.18	82 ± 1.24 a,*,#	78 ± 1.06 a,*,#	76 ± 1.24 a,*,#

Group IV	62 ± 1.8	84 ± 1.24 a*,#	85 ± 1.53 a*,#	87 ± 1.41 a*,#
Group V	57 ± 1.53	86 ± 1.24 a*,#	89 ± 1.41 a*,#	91 ± 1.53 a*,#
Group VI	62 ± 1.06	73 ± 1.36 *,#	70 ± 1.46 *,#	68 ± 1.88 *,#

Values are expressed as Mean ± SEM, (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett's test. Results were compared with control group (* = $p < 0.01$), disease control (# = $p < 0.01$) and standard (a = $p < 0.01$).

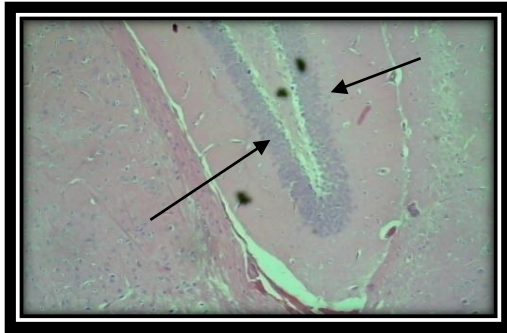


Fig. a: In control, the cerebellum showed the normal neuronal morphology with grey matter content

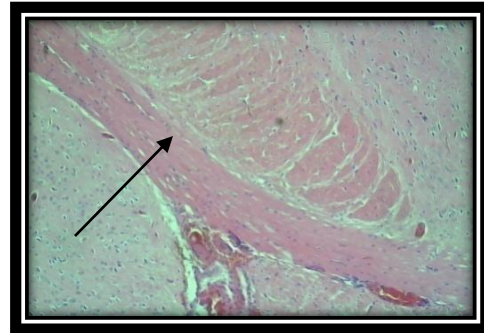


Fig. b: In disease control, the cerebellum showed the severe neuronal damage with no grey matter content



Fig. c: In test extract (200 mg/kg bd.wt., p.o.), the cerebellum showed mild neuronal damage with normal grey matter content



Fig. d: In test extract (400 mg/kg bd.wt., p.o.), the cerebellum showed moderate neuronal damage with minimal grey matter content



Fig. e: In standard + test (400mg/kg bd.wt., p.o.), showed moderate neuronal damage with minimal grey matter content

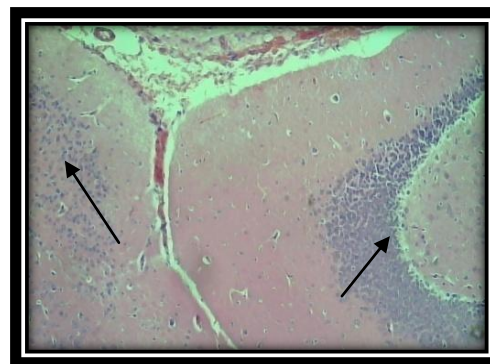


Fig. f: In standard, the cerebellum showed no signs of neuronal damage with optimal grey matter content

Fig. 1: Histopathological studies of mice brain in clonidine induced catalepsy model

DISCUSSION

Catalepsy is a sign of extrapyramidal effect of drugs that inhibit dopaminergic transmission or increase histamine release in brain. Neuroleptics like haloperidol induced catalepsy and clonidine induced catalepsy in mice, are used to evaluate the extract for their anti-cataleptic effects. In present study, the

MEAL was screened for its effect in haloperidol induced catalepsy and clonidine induced catalepsy in mice [2].

MEAL showed radical scavenging activity with H₂O₂ scavenging assay with IC₅₀ value-15 for methanolic extract and IC₅₀ value-13.5 for ascorbic acid respectively and reducing

power assay with IC₅₀ value-28 for methanolic extract and IC₅₀ value-16.5 for ascorbic acid respectively.

Oxidative stress generated as a result of mitochondrial dysfunction particularly mitochondrial complex-1 impairment plays a significant role in the pathogenesis of PD. The oxidative stress was measured through anti-oxidant assays (H₂O₂ scavenging assay and reducing power assay) which reduced the oxidative stress and resulted in anti-cataleptic activity of MEAL.

In the current study behavioural parameters were evaluated with standard bar test and locomotor activity to assess haloperidol induced catalepsy in mice and rota rod performance test and cook's pole climbing test to assess clonidine induced catalepsy in mice.

In haloperidol induced catalepsy model, the behavioural parameters like standard bar test and locomotor activity with MEAL (400 mg/kg bd.wt., p.o.) have shown more significant anti-cataleptic activity (i.e., decrease in cataleptic behaviour and increase in locomotor activity). Standard (L-dopa + carbidopa, 100 + 25 mg/kg bd.wt., p.o.) + MEAL (400 mg/kg bd.wt., p.o.) showed significant effect but did not show synergistic effect in the cataleptic behaviour. Anti-cataleptic activity of MEAL might be due to the action on dopaminergic transmission due to the direct consequence on D₂ receptors and therefore increases the dopamine levels [11].

In clonidine induced catalepsy model, behavioural parameters like rota rod performance test and cook's pole climbing test with MEAL (200 mg/kg bd.wt., p.o.) have shown significant anti-cataleptic activity (i.e., decrease in muscle rigidity and pole climbing time). Standard (L-dopa + carbidopa, 100 + 25 mg/kg bd.wt., p.o.) + MEAL (400 mg/kg bd.wt., p.o.) showed significant effect but did not show synergistic effect in the cataleptic behaviour. MEAL might have anti-histaminic activity due to their mast cell stabilizing property and therefore decreases the histamine levels [2].

The previous evidences have shown beneficial effects of flavonoids on neurodegeneration. Flavonoids can protect the brain by their ability to modulate intracellular signals promoting cellular survival. Alkaloids may act on the CNS, including nerve cells of the brain and spinal cord which control many direct body functions and the behaviour, and may interfere or compete with the action of serotonin in the brain.

The anti-cataleptic activity in haloperidol induced catalepsy and clonidine induced catalepsy models may be due to the presence of flavonoids, steroids, terpenoids, tannins and alkaloids in MEAL [1].

The histopathological results of MEAL (200 mg/kg bd.wt., p.o.) showed mild neuronal damage with normal grey matter content in the cerebellum when compared with MEAL

(400 mg/kg bd.wt., p.o.) in clonidine induced catalepsy model, so MEAL showed good anti-cataleptic activity.

CONCLUSION

In the present study the anti-cataleptic activity of methanolic extract of *Aerva lanata* (MEAL) was evaluated and following conclusions were drawn. MEAL showed the significant anticataleptic activity with haloperidol induced catalepsy and clonidine induced catalepsy, which might be due to the presence of flavonoids, steroids, terpenoids, tannins and alkaloids. Further studies are proposed to prove the anti-cataleptic activity with isolated secondary metabolites.

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