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NEUROPROTECTION BY ETHANOLIC EXTRACT *OF SIDA CORDATA* IN RAT MODEL

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ABSTRACT

In the present study, we evaluated anti-Parkinson's activity of ethanolic extract of *Sida Cordata* leaves and stem in haloperidol induced experimental animal models. In this study, effects of *Sida Cordata* (200, 400mg/kg, p.o) were studied using *in vivo* behavioral parameters like catalepsy, muscle rigidity and locomotor activity and its effects on neurochemical parameters (dopamine, serotonine, and L-Glutamate) in rats. The experiment was designed by giving haloperidol in induce catalepsy to induce Parkinson's disease like symptoms. The increased haloperidol-induced cataleptic scores were significantly ($p<0.01$) found to be reduced, with EESC at dose of 200mg/kg and 400mg/kg (ip). EESC administration showed significant increase in dopamine level and significant reduced in serotonin and L-glutamate level. Daily administration of EESC (400mg/kg) significantly improved motor performance. Thus, the study proved that *Sida Cordata* treatment significantly attenuated the motor defects and also increased the neuro chemical dopamine level.

KEYWORDS

Sida Cordata, Anti-Parkinson's activity, Ethanol extract, L-Dopa, Carbidopa and Haloperidol.

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INTRODUCTON

Parkinson's disease (PD) is a neurodegenerative brain disorder characterized by progressive loss of dopaminergic and other neurons present in the substantia nigra pars compacta resulting in malfunctioning of the cerebral neuronal systems. Clinically the disease may cause slowness of movement, muscle rigidity, and rest tremor[1]. Pathologically, depletion of dopamine in brain due to the presence of intracytoplasmic inclusions known as Lewy bodies is caused. These pathological changes also observed in the locus

coeruleus, pedunculopontine nucleus, raphe nucleus and dorsal motor nucleus of the vagal nerve[2] and sympathetic and parasympathetic postganglionic neurons. Drug management of Parkinson's includes drugs like levodopa, carbidopa, orphenadrine, benztropine, selegiline, pergola which reverses the symptoms of Parkinson's condition but these drugs possess various side effects like respiratory disturbances, hallucinations, discoloration of saliva and urine, dyskinesia convulsions and anxiety, arrhythmia, mydriasis, dry mouth, sore throat, and transient dizziness on long term use[3].

Sida Cordata is a medicinal herb that belongs to the family Malvaceae and is available in moist and semi-evergreen forests of Western Ghats, Tamilnadu, Kerala and Andhra. The stem and leaves of *Sida Cordata* is used to Pulmonary tuberculosis, rheumatism, hematuria, urinary and heart diseases. Also used to cure parkinson's disease and as a food supplement for fat loss. There have been very few scientific studies conducted on *Sida Cordata*. However, a comprehensive and exclusive study of the medicinal properties of *Sida Cordata* is still lacking.

MATERIAL AND METHODS

Collection of Plant Material

Fresh leaves and stems of *Sida Cordata* were collected from natural locations in kollihill region at Namakkal dt, Tamilnadu, and Andhra during July-September. This plant was identified and authenticated by department of Botony, sri venkateswara University. Tirupati. Andhra Pradesh, India. Voucher number 1776.

Animals

Wistar rats (150-200g) of either sex were obtained from Srinivasa Enterprises, Bengaluru, India, housed under standard light/dark cycle and fed with standard pellet diet and water *ad libitum*. The Institutional Animal Ethics Committee, Chennai, India, approved (IAEC, P.Col/11/2017/IAEC/VMCP) experimental protocols and guidelines were followed in conducting the experiments on animals for the purpose of control and supervision of experiments on animals.

Preparation of Leaf Extract

The leaves were collected, shade dried and coarsely grounded. Coarsely powdered plant material (1000g) was weighed and extracted with 5 lit of solvents like Petroleum ether (60-80°C), Chloroform and ethanol by successive extraction in a Soxhlet apparatus for 72hrs. After each extraction, the solvent was distilled off and concentrated extract was transferred to previously weighed petri dish and evaporated to dryness at room temperature (45-50°C) to obtain dried extracts. The extracts were weighed and the percentage yield of the extracts was calculated as follows:

$$\% \text{ of extractive yield (w/w)} = \frac{\text{Weight of dried extract}}{\text{Weight of dried leaves powder}} \times 100$$

The yield of petroleum ether, chloroform and ethanol extract was 18.3, 11.5 and 26.6% (w/w), respectively.

Preliminary Phytochemical Studies

Qualitative chemical tests for establishing the chemical composition profile of given extracts were performed using standard methods/protocols[4] to detect various phyto constituents present.

Detection of Alkaloids

Alkaloids were detected by following in which solvent free extract (50mg) was stirred with few ml of dilute hydrochloric acid and filtered. To few ml of filtrate, a drop or two of Mayer's reagent was added by the sides of the test tube. A white creamy precipitate formed indicates the test as positive.

Detection of Carbohydrates

The extract (100mg) was dissolved in 50ml of distilled water, filtered and subjected to the following test[6].

Fehling's test

One ml of filtrate was boiled on water bath with 1ml each of Fehling's solution I and II. A red precipitate precipitate indicated the presence of sugar.

Benedicts's test

To 2 ml of extract, 0.5ml of Benedicts reagent was added and heated on a boiling water bath for 2min. Characteristic colored precipitate formation indicates the presence of sugar.

Detection of glycosides

50 mg of extract was hydrolyzed with concentrated hydrochloric acid for 2hr on a water bath, filtered and the hydrolysate was subjected to the following test.

Borntrager's test

To 2 ml of filtrate hydrolysate, 3 ml of chloroform was added and shaken. Chloroform layer was separated and 10% ammonia solution was added. Pink colour formation indicates the presence of glycosides.

Detection of saponins

Foam test

50mg of extract was diluted and made up to 20 ml with distilled water and the suspension was shaken in a graduated cylinder for 15 min. A 2cm layer of foam formation indicates the presence of saponins.

Detection of proteins and amino acids

The extract (100mg) was dissolved in 10ml of distilled water and filtered through Whatman No.1 filter paper and the filtrate was subjected to tests of proteins and amino acids.

Millon's test

To 2ml of filtrate, few drops of millon's reagent were added. A white precipitate formation indicates the presence of proteins.

Biuret test

An aliquot of 2ml of filtrated was treated with on drop of 2% copper sulphate solution. To this, 1 ml of ethanol (95%) was added, followed by addition of excess of potassium hydroxide pellets. Pink coloration of the ethanolic layer indicates the presence of proteins.

Detection of Phenolic Compound

Ferric chloride test

The extract (50mg) was dissolved in 5ml of distilled water and few drops of neutral 5% ferric chloride solution were added. A dark green color indicates the presence of phenolic compounds.

Lead acetate test

The extract (50mg) was dissolved in 5ml of distilled water and 3ml of 10% lead acetate were added. A bulky white precipitate indicates the presence of phenolic compounds.

Acute Oral Toxicity of the Extract

Acute oral toxicity study was performed as per OECD 423 guidelines, albino wistar rats (n=6) of either sex selected by random sampling technique were used for acute toxicity study[2]. The animal were kept fasting for overnight providing only water, after which the extract was administered orally at the starting maximum dose level 2000 mg/kg body weight by oral needle and observed for deaths of overt signs of toxicity ½, 1, 2 and 4 h after dosing and subsequently once daily for fourteen days. The signs of changes in skin and fur, eyes, mucous membranes, respiratory, circulatory, autonomic and central nervous system, motor activity and behavior pattern were noted. The toxicity signs of fits, excessive salivation, tremors, diarrhea, lethargy, sleep and coma, as well as the onset of toxicity and signs of toxicity were also noted[5].

Haloperidol Induced Catalepsy (Model by Elliott and Close in 1990)[6]

Haloperidol causes dysfunctioning of various neurotransmitters such as acetyl choline, serotonin and GABA and induced catalepsy due to increased oxidative stress. Haloperidol, an antipsychotic drug, blocks central dopamine receptor in striatum and also produces a behavioral immobility and postural cataleptic state in animals characterized by muscle rigidity and failure to correct an externally imposed posture, thus haloperidol induced catalepsy model was selected. The method described by Elliott and Close in 1990 was followed for the anti cataleptic activity. The animals were divided into five groups (n=6). Group I served as vehicle control, group II served as negative control, group III and IV served as test group treated with EESC (200, 400 mg/kg, i.p.) and group V served as standard L-DOPA + Carbidopa (100+25 mg/kg, p.o.) respectively. Haloperidol (1mg/kg, i.p.) induced catalepsy was examined at every 30min interval for 180min using standard bar test. The duration for which the rat retains the forepaws extended and resting on the scale (block-method 0-3.5scale) was considered as cataleptic score.

Behavioral Assessment

The different behavioral tests assessment were performed by blinded observer at different time points after lesion induction.

Locomotor Activity

The effect on locomotor activity was measured for 10 min at every 30 min upto 3 hours using actophotometer. Actophotometer was used to measure the locomotor activity. It operates on photoelectric cells that are collected in circuit with a counter. When the beam of light falling on the photocell is cutoff by animal, a count is recorded. Actophotometer has either circular or square arena in which animal movements are measured.

Exploratory behaviors

Head dipping[7]

The effect on exploratory behavior (head dipping) was measured for 10 min at every 30 min upto 3 hours using hole board.

The hole board made of plywood has the size (60 cm X 60 cm, 3 mm thick). The mat finished of the upper surface avoids reflections which might alter the behaviour of the animal. The board embodies 9 uniformly distributed holes each of 5 cm in diameter. Each rat was acclimatized for 10 min and number of holes explored through head plunging acts during the total observation time period were noted. Care has to taken to avoid multiple events (two or more head plunging in quicker session). A fresh exploration was considered when the animal neatly plunged its head once and did something else in between like grooming, taking a short walk etc., before plunging its head for the next time. One animal at a time was tested for each activity.

Line crossing[7]

The effect on exploratory behavior (line crossing) was measured for 10 min at every 30 min upto 3 hours using hole board.

The hole board made of plywood has the size (60 cm X 60 cm, 3 mm thick). The mat finished of the upper surface avoids reflections which might alter the behaviour of the animal. The board embodies 9 uniformly distributed lines. Each rat was acclimatized for 10 min and the number of line crossing acts, during the total observation period were counted. Care has to take to avoid multiple events.

Dissection and Homogenization

On the day of experiment rats were sacrificed, whole brain was dissected out and separated the subcortical region (including the striatum). Weighed a specific quantity of tissue and was homogenized in 3 ml HCl- butanol in a cool environment. The sample was then centrifuged for 10 min at 2000 rpm. 0.8 ml of supernatant phase was removed and added to an eppendorf reagent tube containing 2 ml of heptane and 0.25 ml 0.1 M HCl. After 10 min, shake the tube and centrifuged under same conditions to separate two phases. Upper organic phase was discarded and the aqueous phase was used for biochemical estimation.

Biochemical Estimation

Dopamine assay[8]

To 0.02ml of the HCl phase, 0.005 ml 0.4 ml HCl and 0.01ml EDTA/ Sodium Acetate buffer (pH 6.9) were added, followed by 0.01 ml iodine solution for oxidation. The reaction was stopped after 2 min by the addition of 0.1ml sodium thiosulphate in 5 M Sodium hydroxide. 10 M Acetic acid was added 1.5 min later. The solution was then heated to 100^{o} C for 6 min. Excitation and emission spectra were determined using spectrofluorimeter at 330 - 375 nm for the samples at room temperature. Fluorescence (Test - Blank) values, with internal standard (0.005 ml distilled water and 0.1 ml HCl butanol to 20ng of dopamine standard), were measured.

Serotinine Estimation[8]

Serotonin assay

Tissue extract 0.5 ml and 0.625 ml of OPT (Orthopthaldialdehyde) reagent was heated to 100°C for ten min. After the samples reached equilibrium with the ambient temperature, excitation/emission spectra readings at 360-470 nm were taken.

Estimation of brain glutamate levels by UV assay method[9]

In this study animals were decapitated after the last behavioral session. The brains were immediately excised and cerebellum discarded. The cortex, striatum and the sub-cortical parts were separated

and weighed. The sub-cortical region of the brain comprised all the remaining parts of the forebrain after dissection of the cerebral cortex and striatum, including the hippocampus, thalamus, hypothalamus, amygdale and other sub-thalamic structures. Spectrophotometer-suitable for precise measurement at 430 nm.

Deproteinization

Weighed quantity of brain portion were homogenized with 2 parts of perchloric acid by centrifugation for 10 min at 3000 rpm. Pippette out 3.0ml supernatant fluid and make up the pH to 9 with phosphate solution. Allow the solution to stand for 10 min in ice bath and filtered using fluted filter paper. Further, solution allowed to warm to room temperature, diluted and take 1.0 ml for the subsequent assays. Wavelength: 340 nm, Light path: 1cm, Final volume: 3.35ml, Room temperature. For blank measurement, water used as blank instead of sample.

Pipette out 2.00 ml of Glycine-hydrazine buffer, 1.00 ml of Sample, 0.1 ml of ADP solution, 0.2 ml of NAD solution. Mix and read the extinction E_1 at 340 nm. Then added 0.05 ml of GDH (Glutamate dehydrogenase) solution. Sample and Blank solutions were allowed to stand for 45 min and measured the extinction E_2 at 340 nm. Calculated the difference between E_1 and E_2 for sample and blank (ΔE). ΔE_{sample}- ΔE_{blank}= $\Delta E_{glutamate}$ is used for the calculations.

Statistical Analysis

Result values were expressed as average mean ± SEM. The statistical analysis was carried out using analysis of variance ANOVA (between control and drug treatments), followed by Dunnett's 't' test. P values < 0.05 considered as significant.

Histopathological Studies

Histopathological study on the brains from control and experimental groups were done after fixing with 10% formalin, embedded in paraffin wax and sliced into longitudinal sections of 5µm. Further, Histopathology of these sections was observed using stained with hemotoxylin and eosin dye.

RESULTS

Phytochemical Screening:

Acute Toxicity

The EESC dose of 2000 mg/kg was found to be the maximal safe dose with no mortality. Therefore, lower dose of 200 mg/kg and higher dose of 400 mg/kg of EESC were fixed as the study dose.

Effect of EESC on haloperidol induced catalepsy (0-3.5 scale)

The cataleptic scores are depicted in Table No.1. There was a significant difference (P<0.01) between control group (I) and negative control group (II) in catalepsy. The EESC treated groups shows significant anticataleptic action. EESC at dose level of 400 mg/kg particularly, shows anticataleptic action comparable to standard drug treatment. There was a significant difference (P<0.01) between negative control group (II) and EESC in catalepsy.

EESC at dose level of 400 mg/kg, showed good anticataleptic action at 30, 150, 180 min after haloperidol challenge.

Effect of EESC on locomotor activity

The changes in locomotor activity after haloperidol administration are shown in Table No.2. There is a significant (P<0.01) decrease in locomotor activity in negative control group (II) when compared with the control group (I). EESC 400 mg/kg treated animals showed improved locomotor activity when compared with negative control group (P<0.01). EESC at a dose of 400 mg/kg potentiated the locomotor activity of standard drug.

All groups were showing significant difference (P<0.01) when compared with negative control group at all time intervals.

Effect of EESC on exploratory behavior

The exploratory behaviour was expressed by head dippings and line crossings. Head dippings are shown in Table No.3. Negative control group (II) indicated decrease in exploratory behaviour i.e. head dippings and line crossings when compared with control group. The results presented by the EESC treated groups show significant (P<0.01 and P<0.05) increase in head dippings and line crossings when compared with negative control group at 90, 120, 150, 180 min after haloperidol challenge.

Effect of EESC dopamine levels

Dopamine levels are significantly (P<0.01) decreased in negative control group (II) when compared with control group. A significant (P<0.01) increase in dopamine value is observed in EESC treated group animals. The results are showing increased dopamine levels in standard drug treated animals also. The results were shown in Table No.5.

Effect of EESC on serotonin levels

The present study shows significant (P<0.01) increase in serotonin levels in negative control group (II) animals. EESC treated animals at both doses 200 mg/kg and 400 mg/kg showed significant (P<0.05 and P<0.01) reduction in serotonin levels when compared with negative control group. The results are shown in Table No.5.

Effect of EESC on glutamate levels

The present study shows significant (P<0.01) increase in glutamate levels in negative control group animals. EESC treated animals at both doses 200 mg/kg and 400 mg/kg showed significant (P<0.05 and P<0.01) decrease in glutamate level, which is a significant factor in excitotoxicity. The results are shown in Table No.5.

DISCUSSION

Parkinson's disease is a chronic neurodegenerative disorder characterized by loss of dopamine neurons of the SN_{pc}. In the present study, we evaluated the effect of ethanolic extract of *Sida Cordata* in neurotoxin (haloperidol) induced Parkinson disease in experimental animals.

Haloperidol induced catalepsy is a widely accepted animal model of PD. Haloperidol (nonselective D_2 dopamine antagonists) provides a pharmacological model of parkinsonism by interfering with the storage of catecholamine's intracellularly, resulting in dopamine depletion in nerve endings. In the present study, haloperidol (1mg/kg ip) induced significant catalepsy in rats as evidenced by a significant increase in the time spent on the block as compared to vehicle treated animals. Treatment with *Sida Cordata* significantly reduced the catalepsy in haloperidol treated rats in dose dependent manner. The EESC at doses of 200 and 400mg/kg showed protective effect against haloperidol induced catalepsy indicated that this plant has an ability to protect dopaminergic neurotransmission in striatum. The EESC at the doses of 200 and 400mg/kg exhibited significant increase in locomotor activity and increase in muscle activity and thus could be proved with possible action on CNS.

Table No.1: Showed the Phytochemical screening of the ethanolic extract of *H.pentandra*

S.No	Compound	Result
1	Alkaloids	+
2	Steroids	+
3	Glycosides	+
4	Flavonoids	+
5	Terpenoids	+
6	Phenolic compounds and Tannins	+

Table No.1: Effect of EESC on Catalepsy (0-3.5 scale)

Group	Catalepsy (0-3.5 scale)					
	30 min	60 min	90 min	120 min	150 min	180 min
I	0	0	0	0	0	0
II	2.90±0.13 ^{a**}	3.10±0.18 ^{a**}	3.40±0.15 ^{a**}	3.60±0.17 ^{a**}	3.60±0.15 ^{a**}	3.60±0.14 ^{a**}
III	1.27±0.09 ^{b**}	1.99±0.19 ^{b**}	2.17±0.10 ^{b**}	1.85±0.14 ^{b**}	1.42±0.17 ^{b**}	1.09±0.22 ^{b**}
IV	0.95±0.05 ^{b**}	1.82±0.12 ^{b**}	1.94±0.08 ^{b**}	1.72±0.09 ^{b**}	1.28±0.14 ^{b**}	0.74±0.10 ^{b**}
V	0.80±0.07 ^{b**}	1.05±0.10 ^{b**}	1.24±0.09 ^{b**}	1.05±0.05 ^{b**}	0.65±0.10 ^{b**}	0.42±0.12 ^{b**}

The values are expressed as mean ± SEM of 6 animals. Statistical significance test for comparison was done by ANOVA, followed by Dunnett's 't' test. **P<0.01, *P<0.05, ns- Non significant.

Table No.2: Effect of EESC on Locomotor activity

Group	Locomotor					
	30 min	60 min	90 min	120 min	150 min	180 min
I	321.52±7.24	329.22±7.14	325.19±7.62	337.19±5.33	331.28±6.17	334.33±5.29
II	29.19±2.17^{a**}	32.33±2.41^{a**}	30.41±2.64^{a**}	31.22±1.92^{a**}	32.17±2.74^{a**}	34.28±2.52^{b**}
III	82.64±3.29^{b**}	133.29±5.41^{b**}	152.69±4.19^{b**}	175.19±4.52^{b**}	205.48±4.72^{b**}	219.27±4.67^{b**}
IV	95.22±4.67^{b**}	152.87±4.33^{b**}	177.33±4.71^{b**}	195.64±5.37^{b**}	224.52±3.92^{b**}	252.67±4.21^{b**}
V	128.05±6.27^{b**}	228.33±6.47^{b**}	244.17±6.54^{b**}	267.22±6.14^{b**}	272.19±5.21^{b**}	312.64±6.39^{b**}

The values are expressed as mean ± SEM of 6 animals. Statistical significance test for comparison was done by ANOVA, followed by Dunnett's 't' test. **P<0.01, *P<0.05, ns- Non significant.

Table No.3: Effect of EESC on Exploratory Behaviour - Head Dipping

Group	Head Dipping Behaviour					
	30 min	60 min	90 min	120 min	150 min	180 min
I	8.52±0.30	7.84±0.43	8.22±0.54	7.72±0.42	7.52±0.42	7.41±0.21
II	0.49±0.22^{a**}	0.45±0.31^{a**}	0.21±0.17^{a**}	0.52±0.22^{a**}	0.37±0.21^{a**}	0.59±0.22^{a**}
III	3.05±0.22^{b**}	5.09±0.46^{b**}	6.17±0.27^{b**}	6.67±0.49^{b**}	7.07±0.52^{b**}	7.45±0.33^{b**}
IV	4.54±0.33^{b**}	6.05±0.52^{b**}	7.22±0.37^{b**}	7.42±0.39^{b**}	8.02±0.47^{b**}	8.45±0.14^{b**}
V	5.21±0.22^{b**}	6.54±0.41^{b**}	7.69±0.33^{b**}	8.12±0.28^{b**}	8.29±0.51^{b**}	8.69±0.33^{b**}

The values are expressed as mean ± SEM of 6 animals. Statistical significance test for comparison was done by ANOVA, followed by Dunnett's 't' test. **P<0.01, *P<0.05, ns- Non significant.

Table No.4: Effect of EESC on Exploratory Behaviour - Line Crossing

Group	Line Crossing Behaviour					
	30 min	60 min	90 min	120 min	150 min	180 min
I	84.33±3.69	82.67±4.41	85.49±4.57	81.22±3.67	88.42±4.33	89.17±4.17 ^{a**}
II	5.29±0.54 ^{a**}	4.52±0.33 ^{a**}	4.19±0.41 ^{a**}	3.54±0.29 ^{a**}	3.22±0.57 ^{b**}	3.64±0.46 ^{b**}
III	16.51±1.47^{bns}	25.22±2.09 ^{b**}	33.65±3.114^{b**}	47.21±3.33 ^{b**}	55.97±4.24 ^{b**}	61.28±2.17 ^{b**}
IV	32.19±3.29 ^{b**}	52.69±3.41^{b**}	60.28±2.97^{b**}	65.21±4.54^{b**}	71.67±4.28 ^{b**}	74.22±4.54 ^{b**}
V	39.54±3.13 ^{b**}	59.22±4.80 ^{b**}	68.44±4.46 ^{b**}	74.19±4.75^{b**}	77.69±4.49 ^{b**}	79.54±5.28 ^{b**}

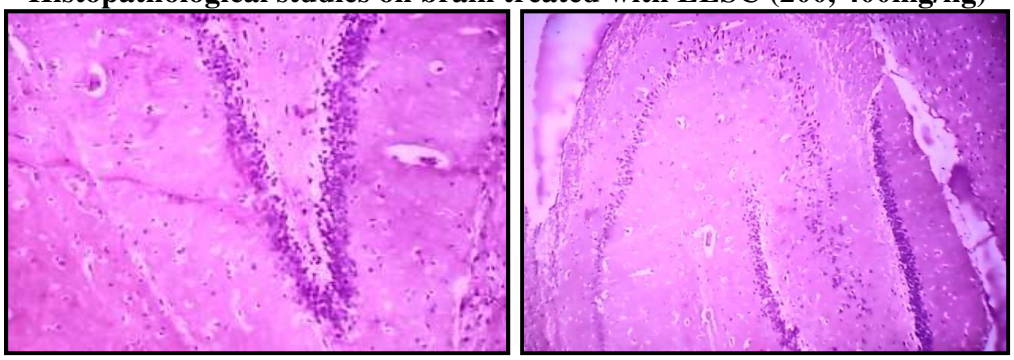
The values are expressed as mean ± SEM of 6 animals. Statistical significance test for comparison was done by ANOVA, followed by Dunnett's 't' test. **P<0.01, *P<0.05, ns- Non significant.

Table No.5: Effect of EESC on brain Neurotransmitter Levels in chronic haloperidol treated rats

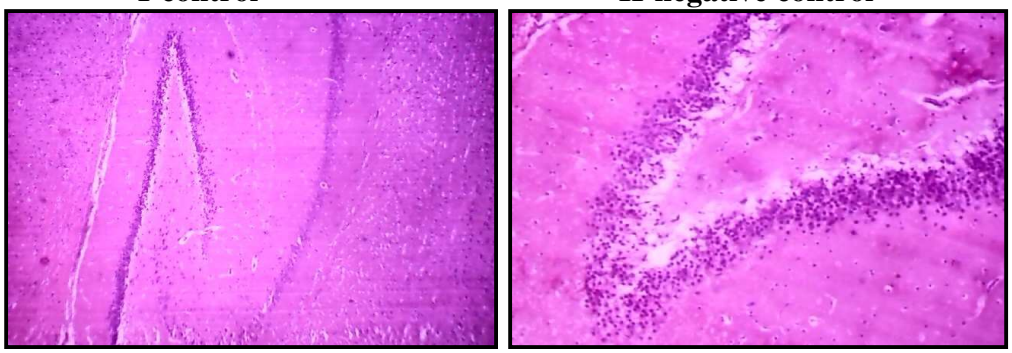
Groups	Treatment	Dopamine (pg/mg tissue)	Serotonin (pg/mg tissue)	L-Glutamate (µ mol/gm tissue)
I	Control	754.69±12.47	274.33±5.17	74.19±3.22
II	Negative control (Haloperidol)	267.33±10.14 ^{a**}	412.27±4.67^{a**}	52.17±2.33^{a**}
III	EESC (200 mg/kg)	554.42±8.12 ^{b**}	345.62±5.33^{b*}	59.21±2.37^{bns}
IV	EESC(400 mg/kg)	617.28±10.44 ^{b**}	325.24±5.12 ^{b**}	64.33±3.11^{b**}
V	L-DOPA (100 mg/kg) + Carbidopa (25 mg/kg) as Standard	682.19±10.22 ^{b**}	307.58±4.69^{b**}	69.22±2.54^{b**}

The values are expressed as mean ± SEM of 6 animals. Statistical significance test for comparison was done by ANOVA, followed by Dunnett's 't' test. **P<0.01, *P<0.05, ns- Non significant.

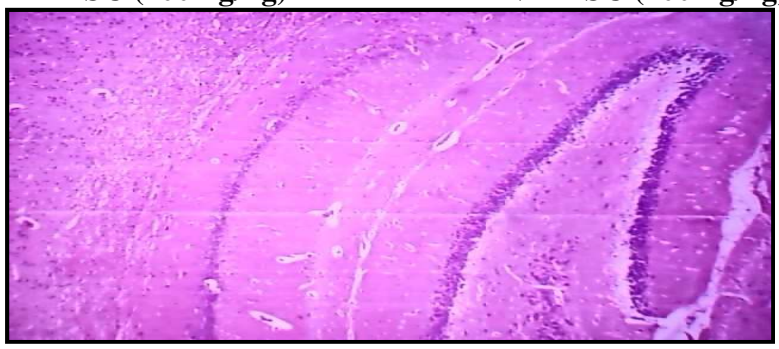
Histopathological studies on brain treated with EESC (200, 400mg/kg)



I-control **II-negative control**



III-EESC (200mg/kg) **IV-EESC (400mg/kg)**



V-STD

CONCLUSION

In view of the above facts, we are concluding that ethanolic extract of *Sida Cordata* plant showed a promising effect in animals with Parkinson's disease. Further detailed molecular studies on anti-Parkinson's pharmacology and toxicology activity and also detailed characterization of active constituents responsible for neuroprotective effect would be appreciated.

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CONFLICT OF INTEREST

We declare that we have no conflict of interest.

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