Evaluation of Anti-Parkinsons And Monoaminooxidase-B(Mao_B) Inhibitory Properties of *Polygonum Cuspidatam* In Male Sprague Dawley Rats

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Abstract
Background: Parkinson's disease (PD) is a neurodegenerative disorder characterized by dopaminergic neuronal loss, oxidative stress, and elevated monoamine oxidase-B (MAO-B) activity. Polygonum cuspidatum, rich in antioxidant phytochemicals, has potential neuroprotective effects.
Objective: To evaluate the anti-Parkinsonian and MAO-B inhibitory properties of Polygonum cuspidatum ethanolic extract (PCEE) in a 6-hydroxydopamine (6-OHDA) induced rat model of PD.
Methods: Rats were divided into five groups: Sham control, 6-OHDA control, 6-OHDA + L-DOPA (6 mg/kg), and 6-OHDA + PCEE (200 and 500 mg/kg). Behavioral assessments (rotarod, catalepsy, and apomorphine-induced rotations) and biochemical analyses (MAO-B activity, dopamine levels via HPLC, lipid peroxidation, catalase, and glutathione levels) were performed.
Results: 6-OHDA induced significant motor deficits, elevated MAO-B activity, reduced dopamine, increased lipid peroxidation, and decreased antioxidant enzyme activities. PCEE treatment produced a dose-dependent improvement in motor behavior and significantly inhibited MAO-B activity. Additionally, PCEE restored dopamine levels and antioxidant status, reducing oxidative damage comparable to L-DOPA.
Conclusion: Polygonum cuspidatum ethanolic extract exhibits potent anti-Parkinsonian effects through MAO-B inhibition and antioxidant mechanisms, suggesting its potential as a natural therapeutic agent for PD management.

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Keywords: Polygonum Cuspidatum, L-DOPA, Oxidative Damage, Antioxidant

Introduction

Herb is a combination of the Latin word herba and the old French word here. "here". Previously, the term "herb" was only used to refer to non-woody plants, such as those found in trees and bushes. Herb now refers to any plant part, such as the fruit, seed, stem, bark, flower, leaf, stigma, or root, as well as a non-woody plant. These medicinal plants are also utilized as food, a flavonoid, medication, or perfume, as well as for spiritual purposes. Initially, these medications were in the form of crude drugs including tinctures, teas, poultices, powders, and other herbal concoctions. An oral tradition passed down the precise plants to be used and the methods of application for specific diseases. Information about medicinal plants was eventually recorded in herbal pharmacopoeias [1, 2]. Treatment with medicinal plants is thought to be quite safe, with no or little side effects. The major advantage of these treatments is that they are in sync with nature.

Medicinal plants are becoming a major source of pharmaceutical ingredients. Traditional practitioners and their armamentarium of medicinal plants are relied on by a considerable part of the population in many developing nations to address health care requirements. Traditional medicine practitioners provide extremely powerful recipes for the treatment of common problems such as diarrhea, constipation, hypertension, low sperm count, dysentery, weak penile erection, piles, coated tongue, menstrual disorders, bronchial asthma, leucorrhea, and fevers. Although modern medicine coexists with such ancient practices, herbal medicine has remained popular for historical and cultural reasons. Herbs are also utilized in natural coloring, pest control, cuisine, perfume, tea, and other applications. Various plants are used in various nations to keep ants, flies, mice, and flees away from houses and businesses.

Long before the prehistoric period, plants were employed for medical purposes. Egyptian papyrus and Chinese scriptures both cited manuscripts from the period of the Unani Herbs. Unani Hakims, Indian Vaids, and European and Mediterranean cultures have all used plants as medicine for over 4000 years, according to evidence. In traditional communities such as Rome, Egypt, Iran, Africa, and America, herbal treatments were routinely used. According to the WHO (World Health Organization), 80 percent of people around the world use herbal medicines for some part of their basic health care needs. Around 21,000 plant species have the potential to be used as medicinal plants, according to the WHO.

According to published data, about three-quarters of the world's population primarily relies on plants and plant extracts for their health care needs. More than 30% of all plant species have been used for medical reasons at one time or another. Plant medications are expected to account for up to 25% of total drugs in established countries like the United States, while they account for up to 80% in quickly emerging countries like India and China. As a result, the economic relevance of medicinal plants is significantly greater in India than in the rest of the globe. These countries contribute two-thirds of the plants utilized in modern medicine, and the rural population's healthcare system is reliant on indigenous medicine [3, 4]. India is one of the most important countries in the world in terms of logistic diversity. Food, ornamental, and medicinal plant crops occupy approximately 54 percent of the country's territory. In India, there are over 45,000 plant species, however, they are scarce in the eastern Himalaya, the western seacoast, and the Andaman and Nicobar Islands. The diverse and varied agro-climatic conditions are responsible for the rich flora legacy. Ayurvedic medical practitioners number around 2,50,000, with a total of nearly 2,91,000 for all traditional systems, compared to 7,00,000 for modern medicine. In rural India, 70 percent of the population relies on the conventional medical system. Herbal medicine practitioners are constantly increasing in the Western world, with roughly 40% of the population using herbs to cure diseases [5, 6]. Rauwolfia serpentina, Asparagus racemosus, Cassia Angustifolia, Sesamum indicum, Holarrhena antidysenterica, Centipedaminima, Withania somnifera, Aconitumnapellus, Piperlongum, and Bauhiniapurpurea are some of the main herbs used as remedies. The utilization of medicinal plants is projected to increase globally as the esteem of herbal treatment and Ayurveda grows. The popularity of herbs has increased because of the side effects of synthetic drugs, the development of resistance to many drugs like antibiotics, public awareness, population explosion, an insufficient supply of drugs, high cost of synthetic drugs.

Plant-based medications can be utilized directly, that is, they can be gathered, dried, and used as therapeutic agents (crude drugs), or their constituents/active principles can be extracted and used as medicines using different chemical procedures. Chemically, the active principle or compounds with comparable structure and activity are synthesized to make synthetic medications utilized in allopathic and contemporary medical systems [6].

Methods and Materials

Animals

Healthy, adult Wistar rats of both sexes (180-220g) were obtained from the animal house facility from our institution. The animals were kept in a well-ventilated room and the animals had exposed to 12 hrs day and night cycle with a temperature between 20±3 °C. The animals were housed in large spacious, hygienic polypropylene cages during the course of the experimental period. The animals were fed with water and rat feed adlibitum. All experiments were performed after obtaining prior approval from CCSEA and IAEC. The animals were housed in suitable environmental conditions.

Collection and authentication of plant material

The fresh Polygonum cuspidatam leafs are collected and identified was purchased from an herbal Market of Hyderabad, Telangana, India, and authenticated by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, S.V University, and Tirupati.

Grouping of animals

Animals were divided into seven groups of either sex; six rats in each group.

Group I: Sham Control
Group II: 6-OHDA Control
Group III: 6-OHDA +L-DOPA (Standard) (6 mg/kg orally)
Group IV: 6-OHDA+ PCEE (200 mg/kg orally)
Group V: 6-OHDA+ PCEE (200 mg/kg orally)
Group VI: 6-OHDA+ PCEE (200 mg/kg orally)
Group VII: 6-OHDA+ PCEE (200 mg/kg orally)

Treatment

were given orally at a dose of 200 mg/kg after 48 hrs of induction for sixty days. L-DOPA was given orally at a dose of 6 mg/kg as standard drug. 38,39 Treatment drugs were suspended in 0.3% CMC solution and control animals were treated with 0.3% CMC in saline.

Parameters Evaluated

The following parameters were evaluated, after the 60th day of treatment.

1. Measurement of Monoamine Oxidase Activity [7]

Procedure

Sample preparation

Rats weighing about 200 g were sacrificed and the brains were quickly removed and homogenized with four volumes of cold 0.9% potassium chloride. Homogenates were kept in small aliquots at -20°C until assayed. The protein concentrations of tissue homogenates were measured by Lowry's method.

Assay

The enzyme incubation mixture contained the following components in a total volume of 0.5 ml: 0.35 ml of 0.2 M sodium phosphate buffer pH 7.2, 0.1 ml of brain homogenate and 0.05 ml of 2 mM benzylamine. The mixture was incubated for 30 min at 37°C except for the study of time course. The reaction was stopped by the addition of 50 µl of 4 M perchloric acid. Protein was removed by centrifugation. A 20 µl aliquot was injected into the liquid chromatogram.

Chromatographic conditions

5 µl Ultrasphere-ODS column (4.5x150 mm); mobile phase, 40% methanol in 50 mM sodium phosphate buffer, pH 3.2, containing 1 mM sodium heptane sulphonic acid; flow-rate, 1.5 ml/min; detection at 254 nm; sensitivity, 0.01 a.u.f.s., injection volume, 20 µl.

Rotarod (Grip strength) [8-9]

The main symptom of the Parkinsonism disease is muscle rigidity. The loss of muscle grip is an indication of muscle rigidity. This effect can be easily studied in animals by using rotarod apparatus. Rotarod has been used to evaluate muscle grip strength by testing the ability of rats to remain on revolving rod. The apparatus has a horizontal rough metal rod of 3 cm diameter attached to a motor with variable speed. This 70 cm long rod was divided into four sections by wooden partitions. First rotarod apparatus was turned on then selected 20 rpm as an appropriate speed. Each rat was given five trials before the actual reading was taken. The animal was placed individually one by one on the rotating rod. The 'fall of time' was noted when animal falls from the rotating rod and then the fall off time of animals were compared in treated group.

Catalepsy test (Fore limb placing test) [11-14]

The major clinical symptom of Parkinson's disease includes difficulty to move and change the posture (akinesia and rigidity) and tremors. So by this parameter we could observe the severity of catatonia as fallowed Stage I- Rat moves normally when placed on table= (Score- 0) Stage II- Rat moves only when touched/pushed= (Score- 0.5) Stage III- Rat placed on the table with front paws set at least on a 3 cm high block fails to correct the posture in 10 sec= (Score- 0.5 for each paw total score-1) Stage IV- Rat fails to remove when front paws are placed alternately on 9 cm block= (score-1 for each paw total Score- 2) Thus for a single rat maximum possible score would be 3.5 revealing total catatonia.

Biochemical Evaluation

Estimation of total protein by Lowry's method [15-16]

Procedure

Extraction of protein from sample- Extraction was carried out with buffer used for the enzyme assay. 500 mg of the brain sample was weighed and homogenized with 5-10 ml of the buffer. Then homogenize was centrifuged and the supernatant was used for the protein estimation.

Estimation of protein

1. Different dilutions of BSA solutions are prepared by mixing stock BSA solution (1 mg/ml) and water in the series of test tubes as given in the table. The final volume in each test tube was 5 ml, and then final BSA concentration was 0.05 to 1 mg/ml.
2. From these different dilutions pipette out 0.2 ml protein solution to different test tubes and added 2 ml of alkaline copper sulphate reagent (analytical reagent). Mixed the solution well.
3. These solutions were incubated at room temperature for 10 minutes.
4. Then added 0.2 ml of reagent Folin-Ciocalteau solution to each tube mixed well and incubated for 30 minutes. Blue colour was developed.
5. Measured the absorbance by colorimeter at 660 nm.
6. Drawn the standard graph and calculated the amount of protein in the sample.

HPLC measurement of dopamine and metabolites [17-19]

Procedure for estimation dopamine by HPLC

Dopamine content was analyzed according to the previously described method with some modifications. Dissected striata were immediately frozen on dry ice and stored at -80°C. Striatal tissues were sonicated in 0.1 M of perchloric acid (about 100 µL/mg tissue) containing paracetamol (100 µg/ml, lemda max-257) as the internal standard. The supernatant fluids were taken for measurements of levels of dopamine by HPLC. Briefly, 20 µL supernatant fluid was isocratically eluted through an 4.6-mm C18 column with a mobile phase containing 50 mM Ammonium phosphate pH 4.6, 25mM Hexane sulphonic acid pH 4.04, 5% acetonitrile and detected by a UV detector at 254 nanometre. The flow rate was 0.5 ml/min. Concentrations of DA was expressed as nanograms per milligram of protein. The protein concentrations of tissue homogenates were measured by Lowry's method.

Lipid peroxidation assay [20]

Procedure

Lipid peroxidation in rat brain homogenate was carried out essentially as described earlier. Rat forebrain (stored at -80ºC for less than 8 days) was homogenized in 20 mM Tris-HCl, pH 7.4 (10 ml) at 4ºC using a Polytron homogenizer. The homogenate was centrifuged at 1000 g for 10 min at 4°C, and the supernatant collected. Then acetic acid 1.5 ml (20%; pH 3.5), 1.5 ml of thiobarbituric acid (0.8%) and 0.2 ml of sodium dodecyl sulphate (8.1%) were added to 0.1 ml of supernatant and heated at 100 ºC for 60 min. Mixture was cooled and 5 ml of n-butanol-pyridine (15:1) mixture, 1 ml of distilled water was added and vortexed vigorously. After centrifugation at 1200×g for 10 min, the organic layer was separated and absorbance was measured at 532 nm using Elisa plate reader. Malonyldialdehyde (MDA) is an end product of lipid peroxidation, which reacts with thiobarbituric acid to form pink chromogen– thiobarbituric acid reactive substance.

Estimation of catalase (CAT) [21-22]

Procedure

Catalase measurement was carried out by the ability of CAT to oxidize hydrogen peroxide (H2O2). 2.25 ml of potassium phosphate buffer (65 mM, pH 7.8) and 100 µl of the brain homogenate were incubated at 25 ºC for 30 min. A 650 µl H2O2 (7.5 mM) was added to the brain homogenate to initiate the reaction. The change in absorption was measured at 240 nm for 2–3 min and the results were expressed as CAT µmol/min mg of protein.

Analysis of GSH/ Glutathione reductase [22-23]

Procedure

GSH was measured enzymatically by the method described by Owen. The striata were homogenized in ice-cold perchloric acid (0.2 M) containing 0.01% EDTA. The homogenate was centrifuged at 10,000 rpm at 4°C for 10 min. The enzymatic reaction was started by adding 200 µl of clear supernatant in a spectrophotometric cuvette containing 500 µl of 0.3 mM reduced nicotinamide adenine dinucleotide phosphate (NADPH), 100 µl of 6 mM 5,5-dithiobis-2-nitrobenzoic acid (DTNB) and 10 µl of 25 units/ml glutathione reductase (all the above three reagents were freshly prepared in phosphate buffer at pH 7.5). The absorbance was measured over a period of 3 min at 412 nm at 30°C. The GSH level was determined by comparing the change of absorbance (ΔA) of test solution with the ΔA of standard GSH.

Isolation of mitochondria [24-26]

Procedure

Tissue was homogenized with a Dounce tissue grinder in mitochondrial isolation buffer (70 mM sucrose, 210 mM mannitol, 5 mM Tris HCl, 1 mM EDTA; pH 7.4) and suspensions were centrifuged at 800 g, 4°C, for 10 min. The supernatant fluids were centrifuged at 13000 g, 4°C, for 10 min, and the pellets were washed with mitochondrial isolation buffer and centrifuged at 13000 g, 4°C, for 10 min to obtain the crude mitochondrial fraction.

Complex I activity assay

Procedure

NADH: ubiquinone oxidoreducase (Complex I) activity was measured in the SN as described in the literature. Brain mitochondria, isolated as above, were lysed by freeze–thawing in hypotonic buffer (25 mM KH2PO4, 5 mM MgCl2, pH 7.4). The reaction was initiated by the addition of 50 µg mitochondria to the assay buffer [hypotonic buffer containing 65 µM ubiquinone, 130 µM NADH, 2 µg/ml antimycin A and 2.5 mg/ml defatted bovine serum albumin (BSA)]. The oxidation of NADH by Complex I was monitored spectrophotometrically at 340 nm for 2 min at 30°C. The activity was monitored for a further 2 min following the addition of rotenone (2µg/ml). The difference between the rate of oxidation before and after the addition of rotenone was used to calculate Complex I activity.

Estimation of mid brain Calcium [27-29]

Procedure

Wistar rats were sacrificed by excess anaesthesia and brain samples were obtained. Homogenates of brain samples were prepared. Tissues (0.4 g) were diced, added to ice-cold PBS solution (40ml) and homogenized with an Omni 5000 homogenizer over ice for 5 min. The homogenate was centrifuged (3000 rpm- 5 min) and the supernatant was separated and stored at -80 0C before AAS analysis. Supernatant (2ml) was diluted with 0.1 M Perchloric acid (0.1ml), mixed well and then centrifuged at 3500 rpm for 10 minutes. The concentration of calcium present in the supernatant was determined by atomic absorption spectroscopy. The standards of different Ca concentrations (i.e., 1, 1.5, 2 and 2.5 µg/ml) were prepared from stock standard. The standards and samples were read against the blank solution. The absorbance of samples, standards and blank were noted. The concentration of calcium in the brain was calculated by reading from the standard curve.

Table 1: Preliminary qualitative phytochemical analysis of Polygonum cuspidatam

Sl. No.	Phytoconstituents	Test result
1	Alkaloid	-ve
2	Glycosides	-ve
3	Carbohydrate	-ve
4	Protein	-ve
5	Amino acid	+ve
6	Steroids	-ve
7	Flavonoids	+ve
8	Terpenoids	+ve
9	Phenols	+ve
10	Saponins	-ve
11	Tannin	+ve

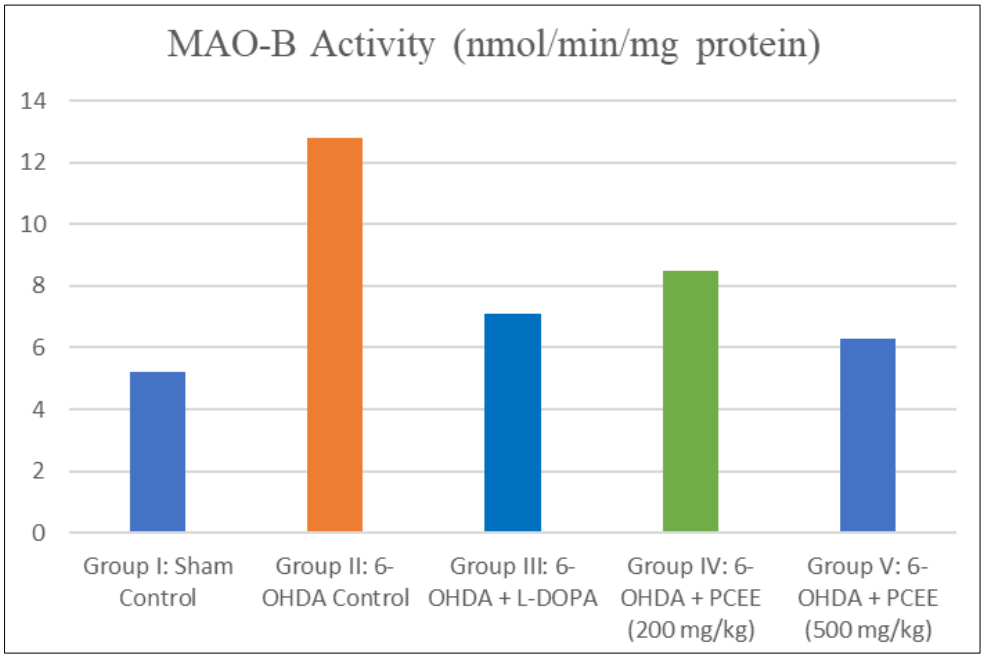
+ve: Present; -ve: Absent

Table 2: Measurement of Monoamine Oxidase Activity

Group	MAO-B Activity (nmol/min/mg protein)
Group I: Sham Control	5.2±0.4
Group II: 6-OHDA Control	12.8±0.6
Group III: 6-OHDA + L-DOPA	7.1±0.5
Group IV: 6-OHDA + PCEE (200 mg/kg)	8.5±0.7
Group V: 6-OHDA + PCEE (500 mg/kg)	6.3±0.4

The study demonstrates that administration of 6-OHDA significantly elevated monoamine oxidase-B (MAO-B) activity in the brain, confirming its role in Parkinson's disease pathology through enhanced oxidative deamination of dopamine and subsequent neuronal damage. Treatment with L-DOPA effectively reduced MAO-B activity compared to the 6-OHDA control group, indicating partial restoration of dopaminergic function. Notably, *Polygonum cuspidatum* ethanolic extract (PCEE) exhibited a dose-dependent reduction in MAO-B activity, with the higher dose (500 mg/kg) showing a more pronounced effect, approaching near-normal enzyme levels. This suggests that PCEE possesses MAO-B inhibitory potential, likely due to its phytoconstituents such as resveratrol and emodin, which may

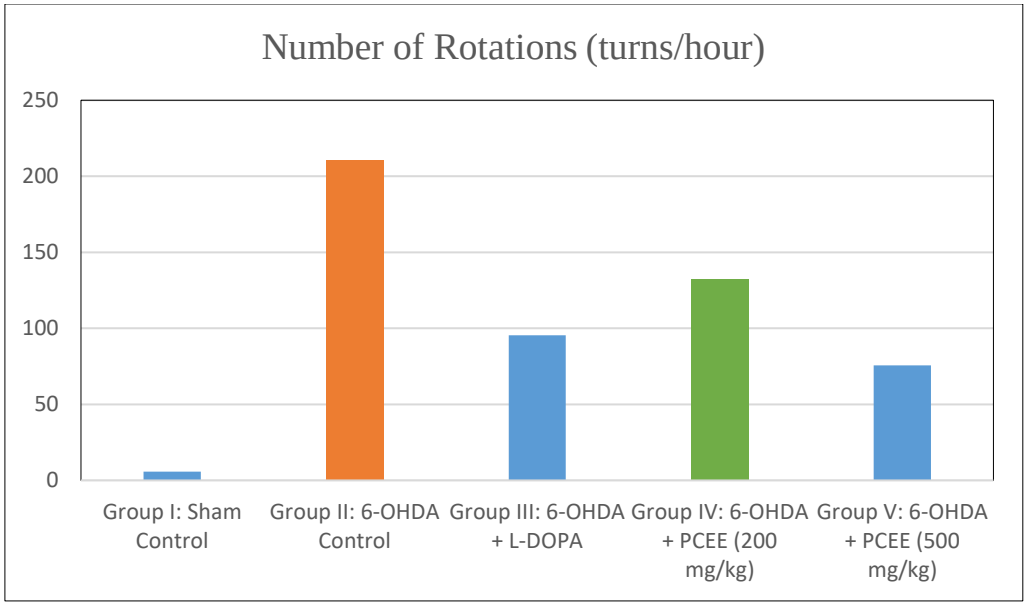
contribute to neuroprotection by reducing oxidative stress and preserving dopaminergic neurons. Overall, these findings support the therapeutic potential of PCEE in managing Parkinson's disease through MAO-B inhibition.



Lesion verification: Quantification of circling behaviour

Group	Treatment	Number of Rotations (turns/hour)
Group I	Sham Control	5.8±1.1
Group II	6-OHDA Control	210.6±12.5
Group III	6-OHDA + L-DOPA	95.4±9.3
Group IV	6-OHDA + PCEE (200 mg/kg)	132.7±10.4
Group V	6-OHDA + PCEE (500 mg/kg)	75.6±8.1

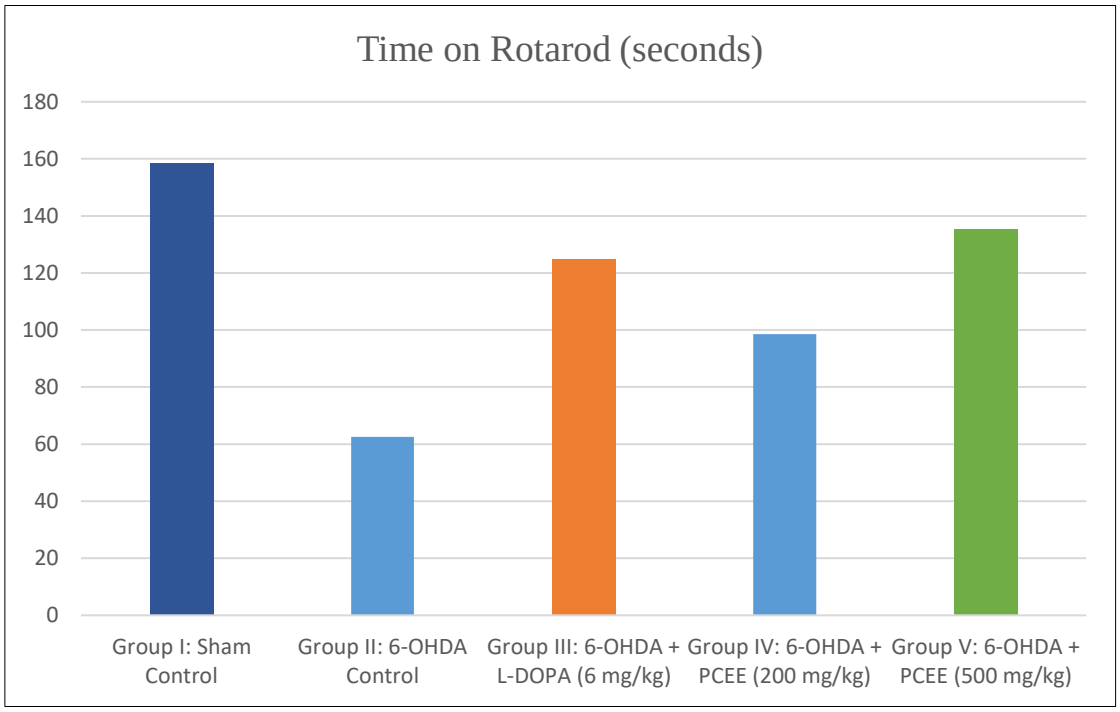
Quantification of circling behavior following apomorphine administration confirmed successful lesioning of the nigrostriatal pathway by 6-OHDA. Rats in the 6-OHDA control group exhibited a significantly higher number of contralateral rotations (210.6±12.5 turns/hour), indicating substantial dopaminergic neuronal loss and receptor super sensitivity. Treatment with L-DOPA markedly reduced the number of rotations (95.4±9.3), reflecting partial restoration of dopaminergic activity. Similarly, rats treated with Polygonum cuspidatum ethanolic extract (PCEE) demonstrated a dose-dependent decrease in circling behavior. The higher dose (500 mg/kg) resulted in a significant reduction in rotational behavior (75.6±8.1), nearly comparable to L-DOPA, suggesting effective neuroprotection. These results confirm that PCEE attenuates motor deficits by preserving dopaminergic neurons or modulating dopamine receptor sensitivity, further supporting its potential anti-Parkinsonian effects.



Rotarod (Grip strength)

Group	Time on Rotarod (seconds)
Group I: Sham Control	158.4±6.3
Group II: 6-OHDA Control	62.5±4.2
Group III: 6-OHDA + L-DOPA (6 mg/kg)	124.7±5.8
Group IV: 6-OHDA + PCEE (200 mg/kg)	98.6±6.1
Group V: 6-OHDA + PCEE (500 mg/kg)	135.2±4.9

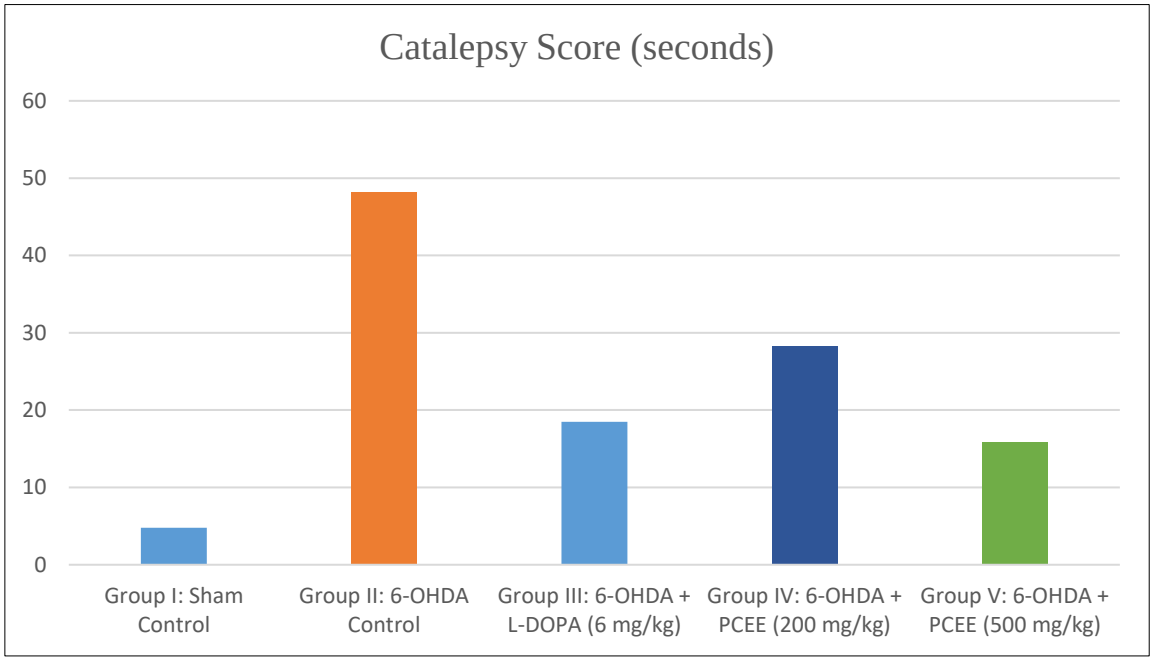
The rotarod test results revealed significant motor impairment in 6-OHDA-lesioned rats, as indicated by a marked decrease in the time spent on the rotating rod (62.5±4.2 seconds) compared to sham controls (158.4±6.3 seconds). Treatment with L-DOPA significantly improved motor coordination and grip strength (124.7±5.8 seconds), demonstrating partial restoration of motor function. Notably, *Polygonum cuspidatum* ethanolic extract (PCEE) produced a dose-dependent improvement in rotarod performance. The 200 mg/kg dose moderately improved motor function (98.6±6.1 seconds), while the 500 mg/kg dose resulted in a significant enhancement (135.2±4.9 seconds), comparable to L-DOPA. These findings suggest that PCEE effectively attenuates motor deficits in the Parkinsonian model, likely through neuroprotective mechanisms involving dopaminergic preservation and antioxidant effects.



Catalepsy test (Fore limb placing test)

Group	Catalepsy Score (seconds)
Group I: Sham Control	4.8±0.9
Group II: 6-OHDA Control	48.2±3.7
Group III: 6-OHDA + L-DOPA (6 mg/kg)	18.5±2.1
Group IV: 6-OHDA + PCEE (200 mg/kg)	28.3±2.6
Group V: 6-OHDA + PCEE (500 mg/kg)	15.9±1.8

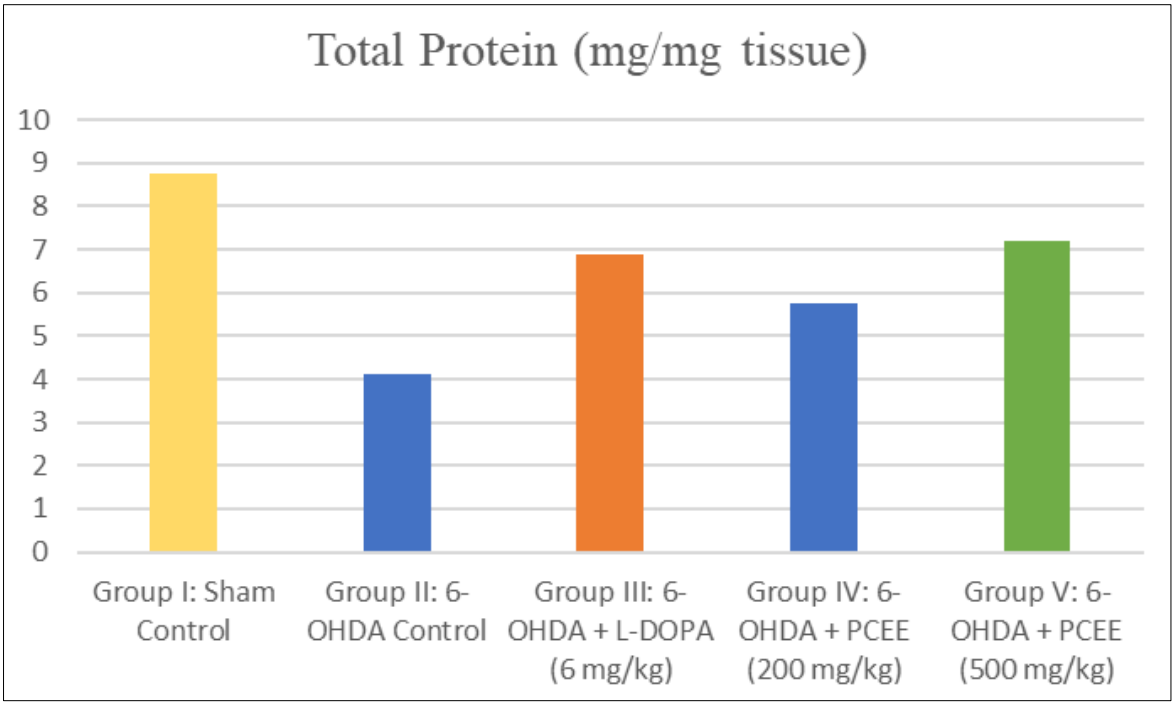
In the forelimb placing (catalepsy) test, the 6-OHDA control group exhibited a significant increase in catalepsy duration (48.2±3.7 seconds), indicating severe motor rigidity and postural deficits due to dopaminergic neuron degeneration. L-DOPA treatment significantly reduced the catalepsy score to 18.5±2.1 seconds, reflecting its effectiveness in improving motor function. Treatment with *Polygonum cuspidatum* ethanolic extract (PCEE) showed a dose-dependent reduction in cataleptic behavior. The 200 mg/kg dose moderately improved rigidity (28.3±2.6 seconds), while the 500 mg/kg dose markedly decreased the catalepsy score (15.9±1.8 seconds), closely approaching the effect of L-DOPA. These results suggest that PCEE has potent anti-cataleptic activity, likely due to its neuroprotective and dopamine-restoring properties, supporting its potential use in Parkinson's disease management.



Estimation of total protein by Lowry's method Estimation of protein

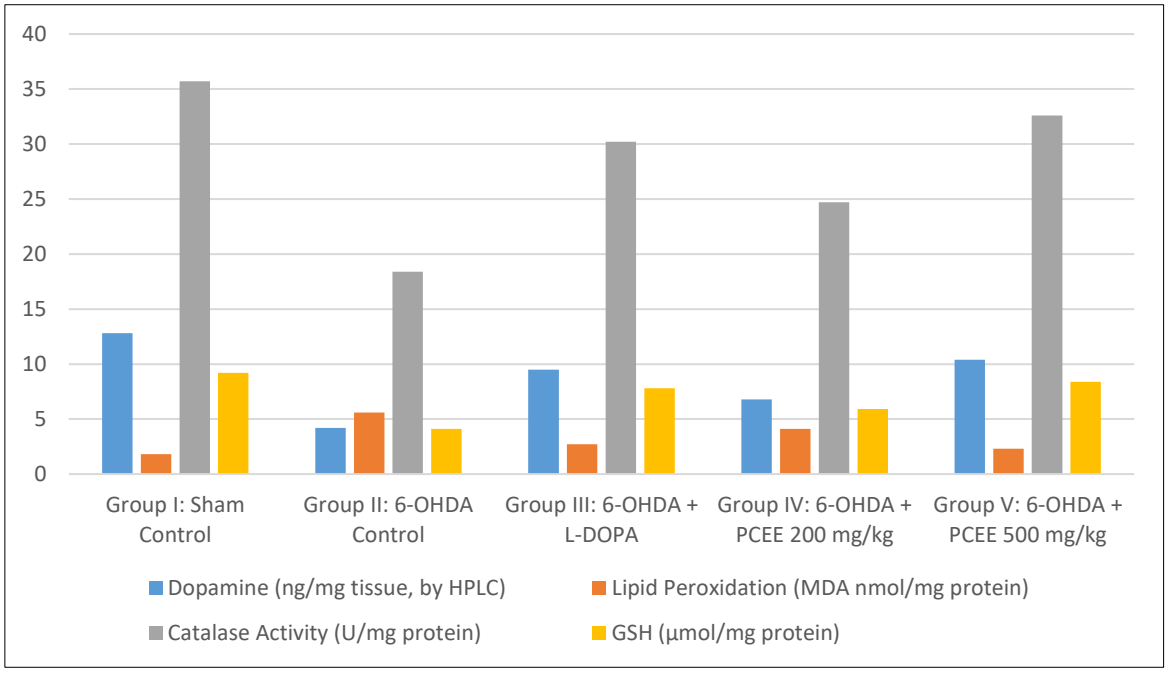
Group	Total Protein (mg/mg tissue)
Group I: Sham Control	8.75±0.32
Group II: 6-OHDA Control	4.12±0.28
Group III: 6-OHDA + L-DOPA (6 mg/kg)	6.89±0.35
Group IV: 6-OHDA + PCEE (200 mg/kg)	5.75±0.30
Group V: 6-OHDA + PCEE (500 mg/kg)	7.21±0.29

The total protein content measured by Lowry's method was significantly decreased in the 6-OHDA control group compared to sham controls, reflecting neuronal loss and tissue damage. Treatment with L-DOPA significantly restored protein levels, indicating neuroprotective effects. Similarly, *Polygonum cuspidatum* ethanolic extract (PCEE) showed a dose-dependent increase in total protein content, with the higher dose (500 mg/kg) producing a near-complete restoration of protein levels. This suggests that PCEE may aid in neuronal recovery and maintenance of brain tissue integrity in the Parkinsonian model.



Assay	Group I: Sham Control	Group II: 6-OHDA Control	Group III: 6-OHDA + L-DOPA	Group IV: 6-OHDA + PCEE 200 mg/kg	Group V: 6-OHDA + PCEE 500 mg/kg
Dopamine (ng/mg tissue, by HPLC)	12.8±0.9	4.2±0.4	9.5±0.7***	6.8±0.6**	10.4±0.8***
Lipid Peroxidation (MDA nmol/mg protein)	1.8±0.2	5.6±0.5	2.7±0.3***	4.1±0.4**	2.3±0.2***
Catalase Activity (U/mg protein)	35.7±2.1	18.4±1.5	30.2±1.9***	24.7±1.7**	32.6±1.8***
GSH (µmol/mg protein)	9.2±0.7	4.1±0.5	7.8±0.6***	5.9±0.4**	8.4±0.5***

The biochemical analyses revealed significant oxidative stress and dopaminergic neurodegeneration in the 6-OHDA control group, demonstrated by a marked decrease in striatal dopamine levels and antioxidant enzyme activities (catalase, GSH), alongside elevated lipid peroxidation (MDA). Treatment with L-DOPA significantly reversed these biochemical abnormalities. Notably, *Polygonum cuspidatum* ethanolic extract (PCEE) exhibited a dose-dependent neuroprotective effect, restoring dopamine concentrations and antioxidant defenses while reducing lipid peroxidation. These results suggest that PCEE mitigates oxidative damage and preserves dopaminergic neurons, potentially through its rich antioxidant phytochemicals. The successful isolation of mitochondria for these assays confirms the integrity of subcellular preparations necessary for accurate biochemical evaluation.



Discussion

Parkinson's disease (PD) is characterized by the progressive degeneration of dopaminergic neurons in the substantia nigra, leading to motor dysfunction and oxidative stress. The present study evaluated the neuroprotective potential of *Polygonum cuspidatum* ethanolic extract (PCEE) in a unilateral 6-OHDA-induced rat model of PD, focusing on behavioral outcomes, monoamine oxidase-B (MAO-B) inhibition, and oxidative stress parameters.

Our results demonstrated that 6-OHDA lesioning significantly increased MAO-B activity, reduced striatal dopamine levels, elevated lipid peroxidation, and decreased antioxidant enzyme activities (catalase and glutathione). These biochemical alterations corresponded with pronounced motor deficits, including increased catalepsy, impaired rotarod performance, and enhanced apomorphine-induced rotations, confirming the successful establishment of a PD model.

Treatment with PCEE resulted in a dose-dependent improvement in both behavioral and biochemical parameters. The extract significantly reduced MAO-B activity, which is notable since MAO-B catalyzes dopamine degradation and generates reactive oxygen species (ROS), thereby contributing to oxidative neuronal damage. The MAO-B inhibitory effect of PCEE may be attributed to its active constituents, such as resveratrol and emodin, which have been reported to possess antioxidant and enzyme-modulating properties.

Furthermore, PCEE treatment restored striatal dopamine levels, suggesting neuroprotection or facilitation of dopamine synthesis/storage. This was accompanied by a reduction in lipid peroxidation and enhancement of endogenous antioxidant defenses, as evidenced by increased catalase activity and glutathione content. These findings indicate that PCEE mitigates oxidative stress, a key pathogenic mechanism in PD progression.

Behaviorally, PCEE improved motor coordination and muscle strength as shown by increased rotarod endurance and decreased catalepsy times. Additionally, it reduced apomorphine-induced contralateral rotations, reflecting preservation of dopaminergic neurons and receptor balance. The higher dose (500 mg/kg) consistently demonstrated superior efficacy, comparable to the standard L-DOPA treatment, highlighting its therapeutic potential.

In summary, the study provides compelling evidence that *Polygonum cuspidatum* ethanolic extract exerts

neuroprotective effects in a 6-OHDA Parkinson's disease model by inhibiting MAO-B, attenuating oxidative stress, and preserving dopaminergic function. These multi-targeted actions position PCEE as a promising candidate for further development as an adjunct or alternative therapy in Parkinson's disease management.

Conclusion

In conclusion, *Polygonum cuspidatum* ethanolic extract (PCEE) demonstrated significant neuroprotective effects in the 6-OHDA-induced Parkinson's disease rat model. The extract effectively inhibited monoamine oxidase-B activity, restored striatal dopamine levels, and attenuated oxidative stress by enhancing endogenous antioxidant defenses and reducing lipid peroxidation. Behavioral improvements in motor coordination, muscle strength, and reduction of cataleptic symptoms further support its therapeutic potential. These findings suggest that PCEE could serve as a promising natural candidate for the management of Parkinson's disease, offering both symptomatic relief and neuroprotection. Further studies are needed to elucidate the specific mechanisms and active constituents responsible for these effects and to assess its clinical applicability.

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