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Soujanya KG
Dept. of Pharmacology, SJMCP, SJM Campus, Chitradurga, Karnataka, India

Nataraj GR
Dept. of Pharmacology, SJMCP, SJM Campus, Chitradurga, Karnataka, India

Abubaker Siddiq
Dept. of Pharmacology, SJM College of pharmacy, SJM Campus, Chitradurga, Karnataka, India

Ananya YA
Dept. of Pharmacology, SJM College of pharmacy, SJM Campus, Chitradurga, Karnataka, India

Corresponding Author:
Nataraj GR
Dept. of Pharmacology, SJMCP, SJM Campus, Chitradurga, Karnataka, India

Assessment of Anti-Parkinsonism Activity of Ethanolic Extract of *Abutilon indicum* L. Leaves in Chlorpromazine-Induced Rats

Soujanya KG, Nataraj GR, Abubaker Siddiq and Ananya YA

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Abstract
Parkinson's disease (PD) is a progressive neurodegenerative condition marked by the loss of dopaminergic neurons in the substantia nigra, resulting in oxidative stress and motor impairments. Although conventional therapies offer symptomatic relief, they are often limited by side effects and reduced effectiveness with long-term use. Consequently, plant-derived compounds with neuroprotective and antioxidant properties are increasingly being explored as potential therapeutic alternatives.
Objective: To evaluate the anti-parkinsonian potential of the ethanolic extract of *Abutilon indicum* L. leaves (EELAI) in a chlorpromazine-induced rat model using behavioral and biochemical analyses.
Methods: Shade-dried powdered leaves of *Abutilon indicum* were extracted with 70% ethanol, rats.
Methods: Shade-dried *A. indicum* leaves were extracted with 70% ethanol, Phytochemical screening was conducted for alkaloids, flavonoids, glycosides, tannins, saponins, triterpenoids, and phenolics. Wistar albino rats (150-200 g) were divided into five groups (n=6): control, chlorpromazine-induced (1 mg/kg, i.p.), standard (levodopa + carbidopa 100+25 mg/kg), and (ethanolic extract of leaves *abutilon indicum* Linn) EELAI-treated (200 mg/kg and 400 mg/kg, p.o.) for 21 days. Behavioral tests (actophotometer, open field, rotarod, bar test) were performed on days 0, 7, 14, and 21, followed by biochemical assays of MDA, GSH, and catalase on day 22.
Results: The extraction of *Abutilon indicum*leaves using 70% ethanol yielded 24.84% w/w. phytochemical constituents confirmed for alkaloids, flavonoids, glycosides, tannins, saponins, triterpenoids, and phenolics. High-dose EELAI (400 mg/kg) produced significant improvement in locomotor activity, exploratory behavior, and motor coordination while reducing catalepsy compared to the chlorpromazine-induced group (P < 0.05-0.0001). Biochemical findings revealed a marked reduction in MDA levels and an increase in GSH and catalase activity (P < 0.05-0.001), indicating strong antioxidant efficacy.
Conclusion: Ethanolic extract of *Abutilon indicum* leaves exhibits notable anti-parkinsonian and antioxidant effects in chlorpromazine-induced rats, suggesting its potential as a complementary therapeutic agent for Parkinson's disease.

Keywords: *Abutilon indicum* L., ethanolic extract, Parkinson's disease, oxidative stress, etc.

Introduction
Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by the loss of dopaminergic neurons in the substantia nigra pars compacta, resulting in reduced dopamine levels in the striatum. First described by Dr. James Parkinson in 1817 as "shaking palsy," PD is clinically characterized by bradykinesia, tremor, rigidity, and postural instability. It occurs in two main forms: familial (genetic) and sporadic (idiopathic). The prevalence of PD increases with age, affecting over 1% of individuals above 60 years and up to 4% of those above 80 years. Globally, an estimated 7-10 million people suffer from PD, with men being more frequently affected than women. Due to the aging global population, the number of cases is expected to rise sharply. In India, although the prevalence rate is comparatively low, the total disease burden remains significant owing to the large population size. Therefore, increasing research and awareness are essential for early detection, prevention, and improved management strategies. Currently, the pharmacological management of PD mainly focuses on restoring dopamine levels through dopaminergic agents such as levodopa (combined with dopa-decarboxylase inhibitors), monoamine oxidase-B (MAO-B)

inhibitors, catechol-O-methyltransferase (COMT) inhibitors, and dopamine agonists. Although these treatments effectively alleviate motor symptoms initially, prolonged use often leads to complications such as dyskinesia, "on-off" phenomena, and wearing-off effects. Furthermore, these therapies do not stop disease progression. Consequently, interest has shifted toward natural antioxidants and neuroprotective agents derived from medicinal plants.

Among various experimental models of Parkinsonism, the chlorpromazine-induced Parkinsonism model is widely utilized to simulate extrapyramidal symptoms similar to those seen in PD. Chlorpromazine, a typical antipsychotic, acts as a dopamine D_2 receptor antagonist in the basal ganglia. Chronic administration of this drug leads to dopamine receptor blockade, producing PD-like symptoms such as rigidity, tremor, and bradykinesia. This model is therefore useful for evaluating agents that can prevent or reverse dopaminergic dysfunction.

Herbal medicines have gained significant attention as potential neuroprotective and antioxidant agents due to their rich phytochemical content, including alkaloids, flavonoids, glycosides, tannins, saponins, and phenolics. These compounds exhibit free radical scavenging and anti-inflammatory activities, which are beneficial in managing neurodegenerative disorders. According to the World Health Organization (WHO), approximately 80% of the global population relies on plant-based traditional medicine for primary healthcare needs.

Abutilon indicum (L.) Sweet, commonly known as "Country Mallow," belongs to the family Malvaceae and is widely distributed in tropical and subtropical regions, including India. Traditionally, various parts of the plant—leaves, roots, bark, seeds, and flowers—are used for their diuretic, anti-inflammatory, analgesic, and sedative properties. Phytochemical investigations have revealed that *A. indicum* contains alkaloids, flavonoids, glycosides, tannins, saponins, triterpenoids, and phenolic compounds, which contribute to its broad range of pharmacological activities, including neuropharmacological effects.

Given the limitations and adverse effects associated with conventional PD therapies, there is an increasing need to explore herbal alternatives with antioxidant and neuroprotective potential. Based on its traditional uses and reported pharmacological profile, *Abutilon indicum* has been selected for evaluation in chlorpromazine-induced Parkinsonism models. The ethanolic extract of *A. indicum* leaves (EELAI) is hypothesized to exert neuroprotective effects by alleviating oxidative stress, restoring dopaminergic function, and improving motor and behavioral impairments associated with Parkinsonism.

Therefore, the present study aims to investigate the anti-Parkinson potential of the ethanolic extract of *Abutilon indicum* leaves (EELAI) against chlorpromazine-induced Parkinsonism in experimental animals, with particular emphasis on its antioxidant and neuroprotective mechanisms.

Materials and Methods

Plant Material

The leaves of *Abutilon indicum* L. were collected from the local garden of Chitradurga, Karnataka and they were washed, weighed and dried in fresh circulating air under shade. The leaves material was identified and authenticated by botanist, Mrs. Niveditha B.T.

Animals

Healthy adult Wister albino rats were used for experimental studies. Animal ethical clearance was obtained from institutional animal ethical committee (IAEC) for experimental purpose (ref.no.3E/ SJMCP/IAEC/06/Aug 2023-24). The animals were obtained from liveon Biolabs pvt. Limited Tumkur-572106. Before the initiation of the experiment, the animals were randomized into various groups and acclimatized for 10 days under standard environmental conditions such as temperature (26±2ºC), relative humidity (50 ±5%), and 12hrs light/dark cycle maintained as per Committee for Control and Supervision of Experiments on Animal (CCSEA) guidelines. All the animals were allowed free access to standard laboratory pellets and drinking water ad libitum under strict hygiene conditions.

Extraction [7]

Abutilon indicum Linn. Leaves were collected from the local garden and shade-dried. The leaves were powered by using a grinder to a coarse powder. The leaf powder of *Abutilon indicum* Linn. were packed well in the Soxhlet apparatus and extracted using 70% ethanol as a solvent in the ratio of 1:10 w/v at 60°C for 48 hrs. The extraction process was repeated according to the need for extract. The extract was filtered while hot. The excess of solvent was removed using water bath. The obtained crude extract was stored in air-tight container in refrigerator, below 10^0C for further studies.

Preliminary Phytochemical Investigation [8]

The ethanolic extract of *Abutilon indicum* Linn. leaves were subjected to standard qualitative tests to identify the presence of various phytochemical constituents like alkaloids, flavonoids, glycosides, tannins, saponins, triterpenoids, and phenolics.

Selection of screening dose [9]

The selection of doses for the ethanolic extract of *Abutilon indicum* Linn. in this study was guided by the findings of an acute oral toxicity study conducted by N.L. Dashputre. The study, following OECD guidelines 425, reported that the median lethal dose (LD50) of the extract was 2000 mg/kg (2 g/kg). Based on these findings, 1/5th & 1/10th of the LD50 is considered as high dose and low dose respectively.

Low dose: 200mg/kg
High dose: 400mg/kg

Experimental design

Chlorpromazine induced Anti-parkinsonism activity [10]

The present study was investigated the potential anti-Parkinson's effects of *Abutilon indicum* Linn. in Albino Wistar rats. The experimental design included five groups:

- Group 1 served as the control.
- Group 2 received the inducing agent chlorpromazine (3mg/kg).

Group 3 served as Inducing agent (CPZ) followed by standard carbidopa and levodopa (100mg/kg +25 mg/kg).

Groups 4 and 5 were administered with inducing agent (CPZ) followed by EELAI at low dose (200 mg/kg) and high dose (400 mg/kg) doses, respectively.
Treatments were given once daily for 3 weeks. Behavioral tests like open field test, rotarod test, bar test were performed on Days 0, 7, 14 and 21, 1 hour after administration of the treatment, distilled water, or the standard drug. Biochemical assays like catalase assay, glutathione estimation, lipid peroxidation were conducted at the 22nd day after treatment period.

Behavioral parameters assessment

1. **Actophotometer:** One of the cardinal features of Parkinson's disease is bradykinesia, which manifests as slowed movement and leads to reduced locomotor activity. To assess locomotor behavior, we used an actophotometer, which comprises infrared sensors and a digital counter. Each animal was placed individually in a 30 cm × 30 cm black metal chamber featuring a wire-mesh floor and a light-tight lid. Six red light beams were projected 2 cm above the floor toward photocells on the opposite wall. Whenever the animal crossed a beam, it interrupted the infrared signal, and these interruptions were recorded and displayed digitally. This setup yields the total locomotor activity of each animal. Prior to performing the cognitive task, each animal was placed in the actophotometer for 5 minutes, and the total activity count over that period was recorded. Locomotor activity is thereby expressed as "total counts per 5 min per animal [11].
2. **Open field test:** The open-field test evaluates the exploratory behavior of model animals and is commonly used to assess a drug's impact on locomotor dysfunction. In our experiment, we used a wooden arena measuring 50 cm × 50 cm with walls 50 cm high. A white sheet divided into 16 equal squares was placed on the floor. Each rat was marked with a small black spot on the neck, then placed in the center of the arena, and its movements were recorded for 5 minutes [12].
3. **Rotarod model:** The effect on motor coordination was evaluated using a Rota-rod test. The rotarod assay is a standard method in rodents to detect even "minimal neurological deficits" affecting motor function and coordination due to muscle rigidity or neural impairment. Each rat underwent a training session before the actual testing. During the test, animals were placed on a rotating rod (7 cm in diameter) set at a fixed speed of 12 rpm. The rat was continuously observed, and the time (in seconds) taken to fall off the rod was recorded as the "latency to fall." The test was repeated over multiple trials for each rat, and the mean latency across trials was used to reduce variability and provide a reliable measure of motor coordination [13, 14].
4. **Bar test:** Catalepsy characterized by a diminished ability to initiate movement and an inability to correct abnormal posture was assessed using the bar test. In this procedure, animals were placed such that their hindquarters remained on the bench while their forelimbs were rested on a horizontal bar (1 cm in diameter) positioned 6-9 cm above the bench. A stopwatch was used to measure how long the animal maintained that imposed posture, up to a maximum of 180 seconds. Each subject underwent three consecutive trials (with 1-minute intervals between trials), and the mean duration was taken. Animals that held the posture for 30 seconds or more were considered to exhibit catalepsy [15].

5. Bio-chemical parameter

Dissection and homogenization [16]

On day 22, after completing the behavioral measurements, the drug-treated animals were euthanized by cervical dislocation. The brains were then rapidly removed, and the forebrain was dissected out (the cerebellum was discarded). The brains were kept on ice while the cortex, striatum, and subcortical regions were separated and weighed. A 10 % (w/v) tissue homogenate was prepared in 0.1 M phosphate buffer (pH 7.0).

Catalase assay: Catalase activity was measured following the method of Aebi (1974). The assay mixture in a 3 mL cuvette comprised 0.05 mL of the supernatant from the 10 % homogenate and 1.95 mL of 50 mM phosphate buffer (pH 7.0). To this, 1.0 mL of 30 mM hydrogen peroxide (H_2O_2) was added, and the decrease in absorbance was monitored at 240 nm over 30 s, taking readings at 15 s intervals. Using the molar extinction coefficient for H_2O_2 (0.071 $mM^{-1} \cdot cm^{-1}$), catalase activity was calculated and expressed as micromoles of H_2O_2 decomposed per minute per milligram of protein [17].

$$\text{CAT activity} = \frac{\delta \text{O.D.}}{\text{E} \times \text{Vol.of Sample (mL)} \times \text{mg of protein}}$$

where δO.D. is change in absorbance/minute
E is extinction coefficient of hydrogen peroxide (0.071 m mol cm−1).

Glutathione estimation (GSH): One milliliter of tissue homogenate was deproteinized by adding 1 mL of 10 % trichloroacetic acid (TCA). The mixture was centrifuged and the precipitate removed. To an aliquot of the supernatant, 4 mL of phosphate buffer and 0.5 mL of DTNB reagent were added. The color that developed was measured spectrophotometrically at 412 nm. The glutathione (GSH) concentration in the supernatant was expressed as nM per mg of tissue (i.e. µM per g, since 1 µM/g = 1 nM/mg).

$$\text{X} = \frac{Y - 0.00314}{0.0314} \times \frac{Dilution\ factor}{Brain\ tissue\ homogenate \times aliquote\ volume}$$

Where,
Y = Abs412 of tissue homogenate.

Estimation of lipid peroxidation level: The supernatant was treated with 10 % TCA and kept in an ice bath for 15 min. The mixture was then centrifuged for 5 min at 2,000 rpm. Two milliliters of the resulting supernatant were transferred to a new tube and mixed with 2 mL of freshly prepared 0.67 % TBA solution. This mixture was heated in a boiling water bath for 10 min. After cooling, the tubes were centrifuged for 10 min, and the supernatant was used for the measurement. The absorbance of the developed color was read at 532 nm (against a reagent blank). The concentration of TBARS in the supernatant was expressed as nM per mg of tissue

$$\text{Conc. Of MDA} = \frac{\text{Abs532} \times 100 \times \text{VT}}{(1.56 \times 105) \times \text{WT} \times \text{VU}}$$

Where,
Abs532 = Absorbance of tissue homogenate.
VT = Total volume of mixture.
WT = Weight of dissected brain.
VU = Aliquote volume.
1.56× 105 = Extinction coefficient.

Statistical analysis
The statistical analysis was performed by using one way ANOVA followed by Dunnett's multiple comparison test and student-t test (unpaired). By using graph pad Prism version 10.5. The values are expressed as mean ± SEM and $P < 0.05$ was taken as a significant.

Results

The leaves of *Abutilon indicum* L. were shade-dried at room temperature for 15 days, pulverized into a fine powder and 250 g of the material was subjected to Soxhlet extraction using 70% ethanol in a 1:10 w/v ratio at 60°C. The extract was evaporated over a water bath. The dried extract was stored in a desiccator to ensure stability, labelled, weighed, and its percentage yield was calculated as 24.84%.

$$\text{Percentage yield} = \frac{\text{Weight of obtained extract in grams (g)}}{\text{weight of crude powder taken (g)}} = \frac{62.1}{250} \times 100 = 24.85\%$$

The Results are shown in Table.1

Table 1: The percentage yield of ethanolic extract of *Abutilon indicum*L. leaves was found to be 24.84%

Solvent	Colour and consistency	Percentage (%) yield
Ethanol	Dark-green and thick semi-solid	24.84%

Phytochemical investigation of leaves of *Abutilon indicum* L
The preliminary phytochemical investigation of EELAI leaves confirms alkaloids, glycosides, flavonoids, tannins, steroids, saponins, triterpenes, resins, amino acid and carbohydrates.

Table 2: Preliminary phytochemical investigation of EELAI leaves.

Sl. No	Phytoconstituents	Ethanolic extract
1.	Tannins	+
2.	Saponins	+
3.	Triterpenoids	+
4.	Flavonoids	+
5.	Resins	-
6.	Glycosides	+
7.	Alkaloids	+
8.	Steroids	-
9.	Carbohydrates	+
10.	Phenols	+

(+): Present, (-): Absent

Selection of Screening Dose
The acute oral toxicity study was carried out using the up-and-down regulation method. The ethanolic extract of *Abutilon indicum* (EELAI) was found to be safe up to a limit dose of 2000 mg/kg, with no mortality observed in the test animals. Based on this result, 1/10th of the maximum safe dose (200 mg/kg) was selected for subsequent pharmacological screening. Therefore, 200 mg/kg and 400 mg/kg were chosen as the low and high doses, respectively, for the experimental study.
High dose: 400mg/kg.
Low dose: 200mg/kg.

1. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Actophotometer:** The ethanolic extract of *Abutilon indicum* leaves (EELAI) was evaluated for its effect on locomotor activity in Wistar albino rats (150-200 g) using the Actophotometer model. Locomotor activity was recorded on days 0, 7, 14, and 21. The control group (normal saline) showed stable activity, while the inducing group (chlorpromazine) exhibited a progressive decline, confirming CNS depression. The standard group (carbidopa + levodopa) showed a highly significant increase in locomotor counts from day 7 onward (****$P < 0.0001$). EELAI at 200 mg/kg showed mild improvement on day 7 (*$P < 0.05$) and significant enhancement by day 14 (**$P < 0.01$) and day 21 (**$P < 0.01$). The 400 mg/kg dose produced a stronger, dose-dependent increase from day 7 (*$P < 0.05$) to day 21 (***$P < 0.001$). Overall, EELAI improved locomotor activity in a progressive and dose-dependent manner, with the high dose showing effects comparable to the standard drug.

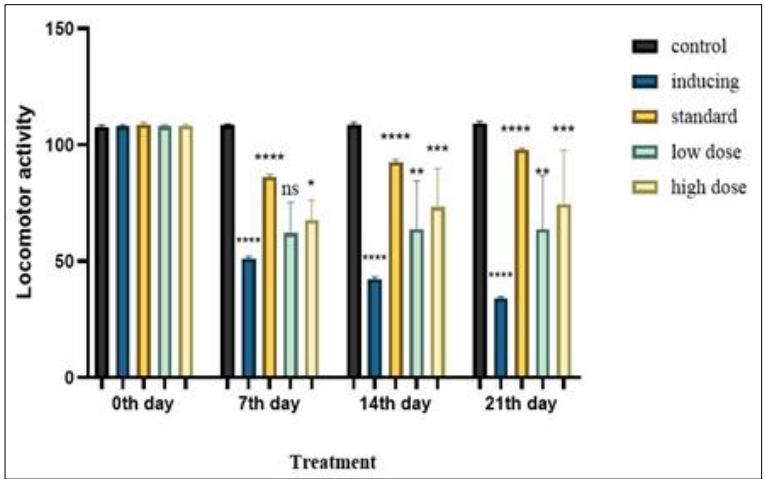

Fig 1: Effect of EELAI in an Actophotometer

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

2. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Open Field Test:** The ethanolic extract of *Abutilon indicum* leaves (EELAI) was evaluated for exploratory behavior using the Open Field Test in Wistar albino rats over 21 days. The number of squares crossed within 5 minutes was recorded on days 0, 7, 14, and 21. The control group showed stable activity with no significant variation. The inducing group (chlorpromazine) exhibited a marked reduction, confirming CNS depression. The standard group (carbidopa + levodopa) showed highly significant restoration (****P < 0.0001). On day 7, the low-dose EELAI (200 mg/kg) showed no significant change, while the high-dose (400 mg/kg) improved moderately (**P < 0.01). By day 14, both doses showed significant improvement, with the high dose showing stronger effects (***P < 0.001). On day 21, the improvement was sustained and dose-dependent. The high-dose EELAI showed results comparable to the standard group. Overall, *Abutilon indicum* enhanced exploratory locomotor activity effectively in PD-induced rats.

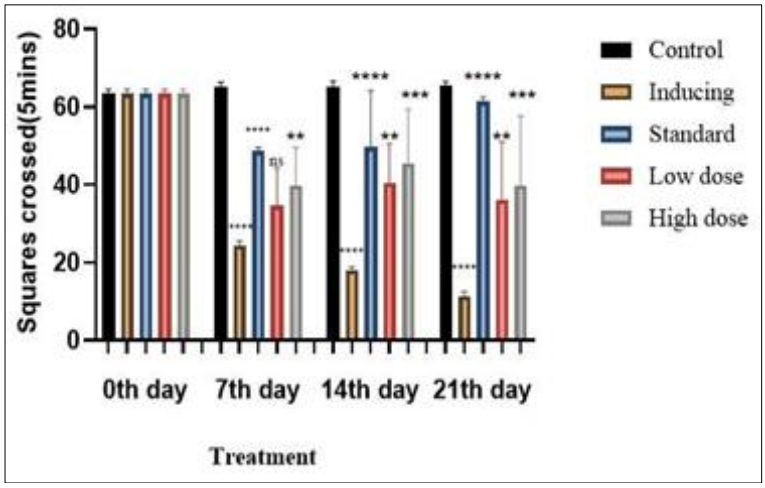

Fig 2: Effect of EELAI in open field model.

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

3. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Rotarod Model:** The ethanolic extract of ***Abutilon indicum*** leaves (EELAI) was evaluated for motor coordination using the Rota-rod test in Wistar albino rats over 21 days. Latency to fall was recorded on days 0, 7, 14, and 21 to assess balance and neuromuscular coordination. The control group showed stable performance throughout the study. The inducing group (chlorpromazine) exhibited a marked decline in latency, confirming CNS depression and motor impairment. The standard group (carbidopa + levodopa) showed significant improvement from day 7 onward (****P < 0.0001).

On day 7, low-dose EELAI (200 mg/kg) showed no major effect, while high-dose (400 mg/kg) produced mild improvement (*P < 0.05). By day 14, both doses showed significant enhancement, with high-dose results

being more pronounced (***$P < 0.001$).
On day 21, improvements were sustained in both groups, especially at the high dose (**$P < 0.01$, ***$P < 0.001$). EELAI improved motor coordination progressively in a dose- and time-dependent manner. High-dose extract showed protective effects comparable to the standard treatment.

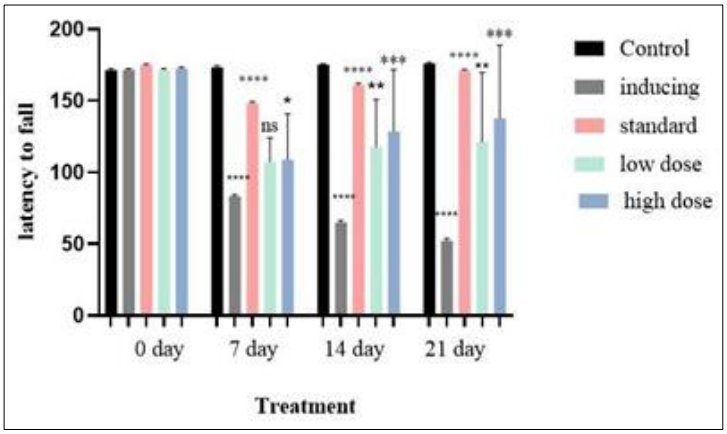

Fig 3: Effect of EELAI in Rotarod Model.

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

4. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Bar Test:** The ethanolic extract of ***Abutilon indicum*** leaves (EELAI) was evaluated for its effect on chlorpromazine-induced catalepsy using the Bar Test in Wistar albino rats over 21 days. Catalepsy scores were recorded on days 0, 7, 14, and 21 to assess muscular rigidity and immobility. The control group showed stable, low scores throughout the study. The inducing group (chlorpromazine) exhibited a marked, progressive increase in catalepsy, confirming CNS depression.
The standard group (carbidopa + levodopa) showed a strong protective effect (***$P < 0.001$, ****$P < 0.0001$). On day 7, low-dose EELAI (200 mg/kg) showed no significant change, while high-dose (400 mg/kg) produced marked improvement (***$P < 0.001$). By day 14, the low dose showed mild reduction (*$P < 0.05$), and the high dose showed stronger protection (***$P < 0.001$). On day 21, both doses-maintained improvement, with high-dose effects comparable to the standard group.
EELAI reduced rigidity and immobility in a progressive, dose-dependent manner.
Overall, *Abutilon indicum* demonstrated strong protective efficacy against chlorpromazine-induced catalepsy.

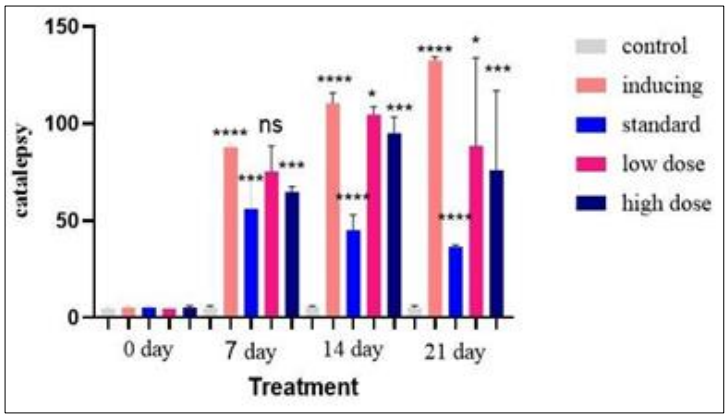

Fig 4: Effect of EELAI in Bar test.

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

5. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Catalase Test:** The ethanolic extract of *Abutilon indicum* leaves (EELAI) was evaluated for its effect on catalase (CAT) activity in Wistar albino rats. Catalase, a key antioxidant enzyme, protects cells by decomposing hydrogen peroxide into water and oxygen. The control group showed normal catalase activity (2.60 ± 0.07), indicating healthy antioxidant defense.
The inducing group (chlorpromazine) exhibited a marked reduction (1.19 ± 0.04****), confirming

oxidative stress and enzyme depletion.

The standard group (carbidopa + levodopa) restored catalase activity close to normal (2.21 ± 0.09****), showing strong antioxidant protection.

Low-dose EELAI (200 mg/kg) caused a mild but significant increase (1.37 ± 0.01*), indicating partial restoration. High-dose EELAI (400 mg/kg) showed a stronger and highly significant rise (1.48 ± 0.01***), reflecting better oxidative stress resistance. Though not fully normalized, catalase levels improved in a dose-dependent manner. The extract enhanced enzymatic antioxidant defense effectively. Overall, *Abutilon indicum* provided significant neuroprotection by restoring catalase activity.

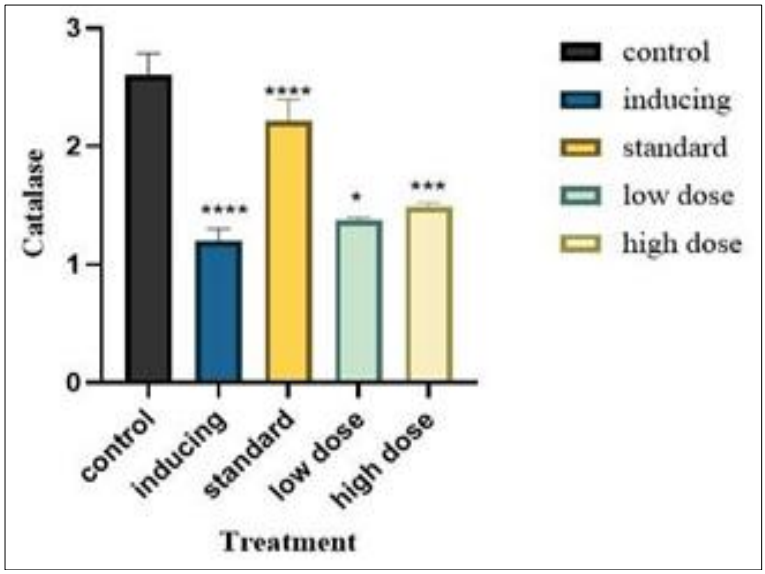

Fig 5: Effect of EELAI in Catalase assay.

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

2. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Glutathione Estimation:** The ethanolic extract of ***Abutilon indicum*** leaves (EELAI) was evaluated for its effect on glutathione (GSH) levels in Wistar albino rats. GSH, a key endogenous antioxidant, protects neuronal tissues from oxidative damage. The control group showed normal GSH levels (10.90 ± 0.09), indicating healthy antioxidant status. Chlorpromazine markedly reduced GSH (4.05 ± 0.04****), confirming severe oxidative stress. The standard group (carbidopa + levodopa) restored GSH to near-normal (9.97 ± 0.10****). EELAI at 200 mg/kg partially increased GSH (4.88 ± 0.03**), while 400 mg/kg produced a stronger effect (5.16 ± 0.02***). The response was dose-dependent, showing enhanced antioxidant defense. Although not matching the standard, EELAI offered significant protection. Overall, ***Abutilon indicum*** mitigates chlorpromazine-induced oxidative stress, supporting its neuroprotective potential.

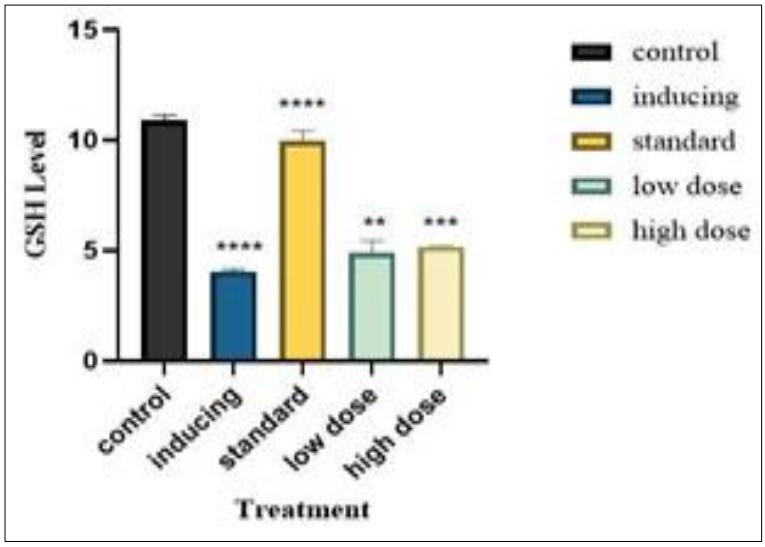

Fig 6: Effect of EELAI in glutathione estimation.

Values are represented as mean ± SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0. 0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

2. **Evaluation of Anti-Parkinsonism Activity of *Abutilon indicum* Linn. Leaves in Lipid Peroxidation Test:** The ethanolic extract of ***Abutilon indicum*** leaves (EELAI) was evaluated for its antioxidant effect by measuring lipid peroxidation (LPO) in Wistar albino rats. LPO is a key indicator of oxidative stress and membrane damage. The control group showed normal LPO levels (3.85 ± 0.007), reflecting balanced redox status. Chlorpromazine markedly elevated LPO (6.51 ± 0.01****), confirming its oxidative toxicity. The standard group (carbidopa + levodopa) significantly reduced LPO (4.31 ± 0.18****), indicating strong antioxidant protection. EELAI at 200 mg/kg slightly lowered LPO (6.40 ± 0.02*), while 400 mg/kg produced a greater reduction (5.87 ± 0.12**). The extract's effect was dose-dependent, with higher doses offering stronger protection. Though not as potent as the standard, EELAI showed notable antioxidant activity. Overall, ***Abutilon indicum*** effectively reduced oxidative damage, supporting its neuroprotective potential.

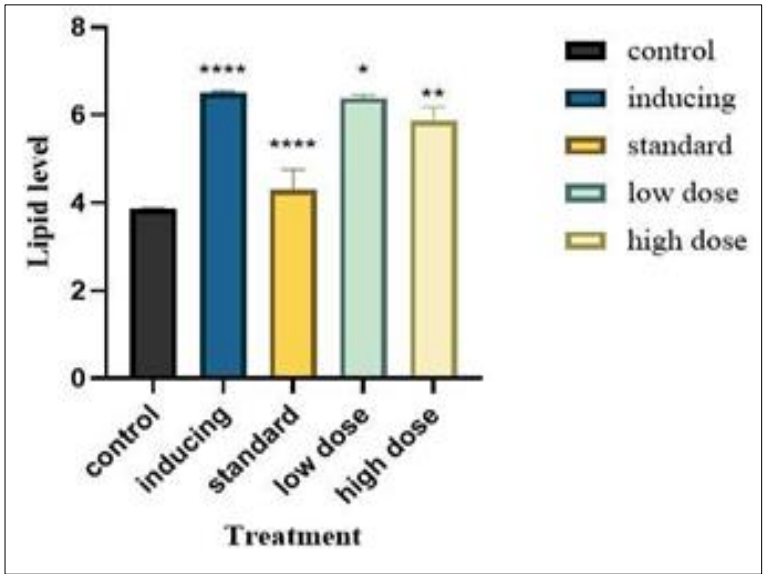

Fig 7: Effect of EELAI in lipid peroxidation test.

Values are represented as mean ±v SEM n=6*p<0.05, **p<0.01, ***p<0.001, ****p<0.0001. Values of normal control and inducing were compared by student t-test and values of treatment groups were compared with inducing group by one-way anova followed by dunnets multiple comparison test.

Discussion

Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily resulting from the degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to decreased dopamine levels in the striatum and subsequent motor dysfunction. Oxidative stress, mitochondrial impairment, and neuroinflammation are key mechanisms contributing to neuronal loss.[18] Chlorpromazine, a typical antipsychotic drug, induces Parkinsonian symptoms in experimental animals by antagonizing dopaminergic D_2 receptors in the nigrostriatal pathway, thereby serving as an established model for evaluating anti-parkinsonian agents. In the present study, the ethanolic extract of *Abutilon indicum* L. leaves (EELAI) was evaluated for its anti-parkinsonian potential using the chlorpromazine-induced model in Wistar rats. The extract yield was 24.84%, and phytochemical screening confirmed the presence of alkaloids, flavonoids, glycosides, tannins, saponins, and phenolic compounds phytoconstituents known for their antioxidant and neuroprotective properties. Chlorpromazine administration resulted in a marked reduction in locomotor activity and motor coordination, reflecting Parkinsonian bradykinesia and rigidity. Treatment with EELAI at doses of 200 and 400 mg/kg significantly improved locomotor performance and rotarod endurance in a dose-dependent manner, with the higher dose showing effects comparable to standard levodopa-carbidopa treatment. The extract also demonstrated significant anti-cataleptic effects in the bar test, indicating improved muscular coordination and reduced extrapyramidal rigidity. In the open-field test, EELAI-treated groups exhibited enhanced exploratory behavior and increased ambulation, suggesting amelioration of motor deficits and anxiety-like behavior induced by chlorpromazine. Biochemical findings supported the behavioral outcomes. Chlorpromazine significantly decreased catalase and glutathione (GSH) levels while increasing malondialdehyde (MDA), indicating oxidative stress. EELAI treatment restored catalase and GSH levels and reduced MDA concentration, suggesting potent antioxidant and neuroprotective actions that may underlie its therapeutic efficacy. Overall, the ethanolic extract of *Abutilon indicum* demonstrated significant neuroprotective and anti-parkinsonian effects in the chlorpromazine-induced model, mediated through enhancement of endogenous antioxidant defense and modulation of dopaminergic function. These findings support the potential of *A. indicum* as a promising natural therapeutic candidate for the management of Parkinson's disease.

Conclusion

The present study evaluated the anti-parkinsonian potential of the ethanolic extract of *Abutilon indicum* Linn. leaves in a chlorpromazine-induced rat model. Phytochemical analysis revealed the presence of alkaloids, flavonoids, glycosides, tannins, saponins, triterpenes, and phenolic compounds known for antioxidant and neuroprotective activity. Chronic administration of chlorpromazine produced marked Parkinson-like symptoms, including reduced locomotor activity, impaired coordination, and increased catalepsy. Treatment with *A. indicum* extract for 21 days produced a dose-dependent improvement in behavioral parameters, with the high dose showing effects comparable to levodopa-carbidopa. Biochemical analysis demonstrated restoration of dopamine levels, normalization of catalase and glutathione activity, and reduction in lipid peroxidation. These findings indicate that *Abutilon indicum* possesses significant neuroprotective and anti-parkinsonian activity in chlorpromazine-induced parkinsonism, likely mediated through antioxidant enhancement and dopaminergic restoration. Further studies are needed to isolate active constituents and establish clinical safety.

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