



Evaluation of the Anti-Parkinson Activity of Curly Kale (*Brassica Oleracea*) In Albino Swiss Mice.

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Parkinson, *Brassica Oleracea*, Extract, Phytochemical Test, Albino Swiss Mice

ABSTRACT:

Plant extract that was submitted to phytochemical analysis revealed the existence of many bioactive compounds including proteins, carbohydrates, saponins, flavonoids, alkaloids, phenols, tannins, and terpenoids, but lacking glycosides. Given its many qualities, the plant could have medicinal and medical applications. The effects of bromocriptine and the plant extract (KEE) on motor coordination, locomotor activity, catalepsy, dopamine concentration, and oxidative stress were evaluated in a Parkinsonian model generated by haloperidol. Using the Rotarod test, KEE (100–500 mg/kg) and bromocriptine (5 mg/kg) were shown to improve motor coordination; the effects of KEE were more noticeable at higher doses. KEE improved in a dose-dependent manner, according to the Actophotometer assessment, and both bromocriptine and KEE reduced the locomotor activity that had been reduced by haloperidol. Larger doses of KEE, particularly the 500 mg/kg dose, also shown favourable benefits. Bromocriptine dramatically reduced catalepsy scores in the catalepsy test. Bromocriptine and KEE both significantly raised dopamine levels; UV spectrophotometry demonstrated a dose-dependent increase in dopamine. Larger doses provided even more significant protection against the oxidative stress generated by haloperidol. Additionally, KEE increased brain catalase activity. Overall, the results show that both bromocriptine and KEE may have neuroprotective benefits, with KEE showing promise as a therapeutic benefit on a number of fronts.

1. Introduction

Students are more likely to ask questions and remember details about anatomical structures (including their names, eponyms, and variants) in a holistic cultural context when they are connected to practical uses. In general, symptoms including psychosis, dementia, orthostasis, and more serious falls tend to manifest later. Up to 82% of people with Parkinson's disease are thought to have oropharyngeal dysphagia. There have been reports of dysphagia, or trouble swallowing, at every stage of the illness. A medical history and neurological examination are usually the foundation of a doctor's first evaluation. They evaluate motor symptoms, such as bradykinesia and rest tremors, using clinical diagnostic criteria [01-04].

The discovery of Lewy bodies during an autopsy in the midbrain is often regarded as conclusive evidence of Parkinson's disease. It is necessary to frequently revisit the presentation in order to verify the correctness of the diagnosis, since the illness's clinical history may deviate from Parkinson's disease over time. Our goal with the therapy is to employ *Brassica oleracea* extract. Plants belong to the following taxonomic families: Family: *Brassicaceae* or *Cruciferae*, Cultivar Group: *Acephala* group, Kingdom: *Plantae*. *Brassica* is the Genus [05-10].

The main phytoconstituents of a plant that have medicinal potential include alkaloids, tannins, flavonoids, saponins, quinone terpenoids, phenylpropanoids, and polyphenol compounds. The ingredients of raw kale include 84% water, 9% carbs, 4% protein, and 1% fat. It's high in manganese, folate, vitamin B6, vitamin C, and vitamin A.



In addition to various nutritional elements including iron, calcium, magnesium, potassium, and phosphorus, kale is

an excellent source of thiamine, riboflavin, pantothenic acid, and vitamin E [11-17].

MATERIAL & METHODS-

Several essential tools and substances will be used in this research to carry out a variety of experimental methods. Precise measurements of substances like haloperidol (5 mg/kg) and bromocriptine (4 mg/kg), which are delivered to investigate their pharmacological effects, are guaranteed by the analytical weighing scale. The UV spectrophotometer is probably used to measure absorbance and investigate enzymatic activity in quantitative analyses of chemical processes, such those involving ferric chloride and potassium ferricyanide. The rotarod device for motor coordination and the actophotometer for locomotor activity may be used in behavioural research, especially to evaluate the effects of medications such as L-DOPA. Using a lyophilizer for freeze-drying and a rotary vacuum evaporator for solvent removal may be necessary during sample preparation. Compounds that are sensitive to temperature must be kept in a deep freezer, and a CO₂ chamber may be used for compassionate animal death. The chemicals are necessary for creating solutions, preserving pH, and carrying out redox reactions that are critical to the project's biochemical tests. These substances include ice-cold saline, different buffers, and H₂O₂.

COLLECTION AND AUTHENTICATION OF PLANT-

In October 2023, the local vegetable market in Mumbai was where the *Brassica oleracea* plants were bought. The botanist at Alrsin Ayurvedic Pharmaceuticals in Andheri, Mumbai, Mr. Mahesh Atale, received the sample specimen voucher. After being cleaned with tap water, the plant stems were shade-dried at room temperature with the use of a fan to help circulate air. After the stem was dried, it was combined with other ingredients to make coarse powder, which was then kept in a jar.

EXTRACTION OF BRASSICA OLERACEA –

A method called soxhlet extraction is used to extract bioactive substances from plant material, including flavonoids, glucosinolates, and other phytochemicals. *Brassica oleracea* is also referred to as cabbage. In this

procedure, a thimble within the Soxhlet device contains dried and powdered *Brassica oleracea* [18-19].

The essential components are dissolved in the plant material when a suitable solvent, such ethanol or methanol, is heated in a flask and its vapours flow through it. The cycle is then constantly repeated as the solvent containing the extracted chemicals condenses in the top chamber and drops back into the flask. This guarantees a concentrated extract by enabling effective component extraction without the need for excessive solvent. Because soxhlet extraction can maximise yield in a controlled and repeatable way, it is the ideal approach for extracting phytochemicals [20-27].

PHYTOCHEMICAL SCREENING [26-35]-

To ascertain if certain phytoconstituents were present in the ethanolic extract of *Brassica oleracea*, preliminary chemical assays were conducted.

ANIMAL STUDY-

Animal Required & Dose Selection [36-40]

A total of 36 Albino Swiss mice are needed for the investigation. These mice are often utilised in research because of their constant genetic make-up and physiological responses. It is recommended that mice between the ages of 8 and 12 weeks be used for research that need fully developed biological systems, since this age range reflects adult mice. It is ensured that the animals are healthy and suitable for the experimental procedures if their weight falls between 20 and 30 grammes. Depending on the experimental design, both male and female mice may be employed, offering flexibility in gender selection and enabling balanced or targeted gender-based experiments. Given that there are 36 animals in all, it is probable that the research uses many experimental groups or duplicates to guarantee statistical validity and repeatability.

Curly kale (*Brassica oleracea*) ethanolic extract was shown to be safe in the literature review, with an LD₅₀ of 5000 mg/kg. Therefore, 100 mg/kg, 250 mg/kg, and 500 mg/kg of Curly Kale (*Brassica oleracea*) extract (KEE) were determined to be the final dosages for a research study.



Evaluation of Antiparkinsons Activity [41-51]

Rotarod Test

Motor coordination test will be conducted using rotarod apparatus. The animals will be placed on the moving rod before the treatment and the mice will stay on the rod without falling for 120 sec. will be chosen for the study. The time animals take to fall from the rotating rod will be noted before and after the treatment with extract.

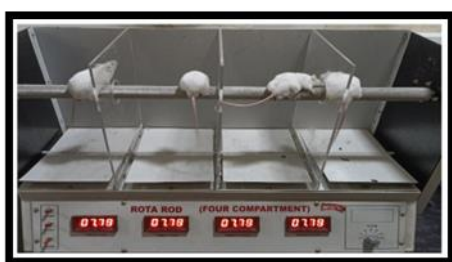


Fig no 01- Determination of motor coordination pattern using rotarod

Actophotometer Test

An actophotometer will be used to measure the locomotor activity. It is made up of a sizable cage of 30 x 30 x 30 cm, six lights, and six photocells positioned along the outside edge of the bottom so that each mouse block has a single beam. When light beams strike photocells, the photocells become active. When an animal passes in front of the light beam, the number of cut interruptions will be recorded for ten minutes.



Fig No 02- Determination of locomotor activity using Actophotometer.

Bar Test-

To gauge the catalepsy, a bar test will be used. The animal will undergo the bar test by placing its front paw on a horizontal bar that is three centimetres above and perpendicular to the base. We will keep the bar. The bar

test will have a predetermined maximum cut off time of 300 seconds.



Fig No 03- Determination of catalepsy using 3cm bar

Grouping of Animals-

Table No 01- Grouping of Animals

Group	Test substances	Albino Swiss Mice required per group	Dose	Total
Group 1	Distilled Water	6	10 ml/kg	6
Group 2	Haloperidol I.P.	6	2mg/kg	6
Group 3	Bromocriptine + Periodontal	6	5 mg/kg +2 mg/kg	6
Group 4	KEE+ Haloperidol	6	100 mg/kg	6
Group 5	KEE+ Haloperidol	6	250 mg/kg	6
Group 6	KEE+ Haloperidol	6	500 mg/kg	6
Total animals required		36		

Biochemical Parameters [46-57]-

Preparation of Brain sample-

The animals were put to sleep in a CO₂ chamber after the aforementioned parameters had been evaluated. The brains were swiftly extracted and placed on a bed of icy salt water. We used a 0.1M Phosphate buffer (pH 8) to weigh and homogenise the tissues. The amount of dopamine and the results of the CAT test were both determined using the collected supernatant.

Determination of dopamine by UV Spectrophotometer-

The amount of dopamine in the brains of mice was determined by mixing 1 millilitre of homogenised supernatant liquid with 1 millilitre of ferric chloride (1.5 X 10⁻² M) and 1 millilitre of potassium ferricyanide (1.5



X 10-2 M) in 25 millilitres of distilled water. We used a UV-visible double beam spectrophotometer set at 735 nm to evaluate the developed colour after 30 minutes of setting it aside.

Determination of Catalase-

Mice in each group will be put to sleep in a carbon dioxide chamber after their motor coordination, locomotor activity, and bar tests have been evaluated in haloperidol-induced Parkinson's disease. Then, their brains will be extracted. Without delay, they will be submerged in icy salt water. We will weigh the tissues and mix them well in a 0.1M phosphate buffer with a pH of 8. For the purpose of analysing Catalase activity, several test tubes will be used to collect samples of rat brain homogenates. The catalase test will be conducted using the supernatant.

Result & Discussion-

Qualitative Phytochemical Screening-

Table No 02- The Phytochemical analysis observation of plant

Phytoconstituents	Test	Observation
Carbohydrates	Molisch test	+ ve
	Benedict's test	+ ve
Proteins	Biuret test	+ ve
	Sulphur powder test	+ ve
Saponins	Froth test	+ ve
Flavonoids	Lead acetate test	+ ve
	Shinoda test	+ ve
Alkaloids	Hager's and Dragondroff's test	+ ve
	Ferric Chloride test	+ ve
Phenols and tannins	Dilute Potassium permanganate test	+ ve
	Legal test	- ve
Terpenoids	Salkowski test	+ ve

Several beneficial substances have been identified in the plant via phytochemical investigation. A good result in both the Molisch and Benedict's tests indicated the presence of carbohydrates. When the Biuret and Sulphur powder tests came back positive, it meant that proteins were present. We used the Froth test to find saponins. Lead acetate and Shinoda tests both came back positive, confirming the presence of flavonoids. Testing by Hager

and Dragondroff verified the presence of alkaloids. The ferric chloride and diluted potassium permanganate assays were used to identify tannins and phenols. The Salkowski test was used to identify terpenoids. The absence of glycosides was confirmed by the negative result of the Legal test. The wide variety of phytoconstituents identified in this exhaustive study point to the plant's possible therapeutic and medical uses.

Rotarod Test –

Table No 03- Effect of Bromocriptine and KEE on motor coordination using Rotarod.

Treatment groups	Fall of time (in seconds) Mean± SEM
Vehicle control	91.90 ± 1.105
Inducing Haloperidol 2 mg/kg	22.81 ± 1.482
Standard Bromocriptine 5 mg/kg	73.72 ± 1.348 ****
KEE 100 mg/kg	26.18 ± 1.257 *
KEE 250 mg/kg	34.54 ± 1.247 *
KEE 500 mg/kg	66.28 ± 6.736 *

Six animals make up each group, and the data is shown as Mean ± SEM. Compared to the Parkinson's control group, there is a significant difference ($p < 0.05$). Every Removable Element $P < 0.05$ Using a Rotarod test, the researchers look at how Bromocriptine and KEE (likely a plant extract) affect motor coordination. With a fall-off time of 91.90 seconds, the vehicle control group demonstrated excellent motor coordination. With haloperidol, motor coordination was severely affected, and the fall-off time was reduced to 22.81 seconds. One conventional medication, bromocriptine, raised the fall-off time by 73.72 seconds, indicating better motor coordination. Similar to the Haloperidol group, KEE enhanced motor coordination at various dosages; the fall-off durations were 26.18 seconds (100 mg/kg), 34.54 seconds (250 mg/kg), and 66.28 seconds (500 mg/kg), suggesting a response that was dose-dependent. Although larger dosages of KEE demonstrated more significant benefits, these findings indicate that both Bromocriptine and KEE may mitigate motor coordination deficiencies caused by Haloperidol.

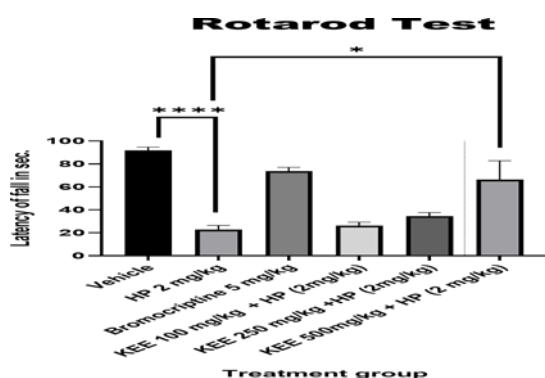


Fig no 04- Effect of bromocriptine and KEE on motor coordination using Rotarod.

In comparison to the vehicle control group, the haloperidol control group had a significantly lower time fall (in seconds) from the rotarod [F (5,30)=26.18, p<0.05 Fig no.4]. The groups given bromocriptine and KEE showed considerable improvements.

Actophotometer Test-

Table No 04- Effect of bromocriptine and KEE on locomotor activity using Actophotometer.

Treatment groups	Ambulation counts/ 10min mean ± SEM
Vehicle control	230.3 ± 8.472
Inducing Haloperidol 2 mg/kg	25.33 ± 6.216
Standard Bromocriptine 5mg/kg	177.8 ± 8.432 ****
KEE 100 mg/kg	45.83 ± 1.922*
KEE 250 mg/kg	63.83 ± 1.797*
KEE 500 mg/kg	148.7 ± 3.879*

The using an actophotometer, the research determines how Bromocriptine and KEE affect locomotor activity. There was a lot of locomotor activity in the vehicle control group, with 230.3 counts every 10 minutes. The inhibitory impact of haloperidol was shown by the dramatically decreased locomotor activity to 25.33 counts. The conventional therapy, bromocriptine, significantly increased locomotor activity to 177.8 counts. In comparison to the Haloperidol group, KEE, when given at several dosages (45.83 (100 mg/kg), 63.83 (250 mg/kg), and 148.7 (500 mg/kg)), increased locomotor activity. These findings point to the fact that KEE, in conjunction with Bromocriptine, may counteract the effects of Haloperidol on locomotor activity, with the latter having a more noticeable impact at larger

dosages. There was a statistically significant difference (p < 0.05) in the benefits shown with KEE.

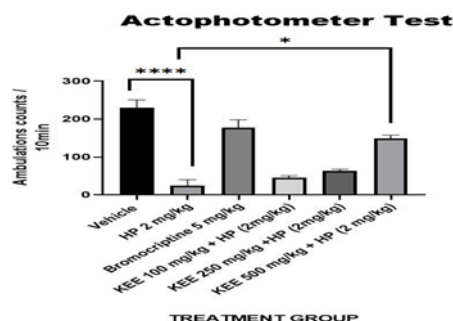


Fig no 05- Effect of bromocriptine and KEE on locomotor activity using actophotometer.

Spontaneous motor activity was significantly decreased in the haloperidol control group as compared to the vehicle control group. [F (5, 30) = 17.45, p < 0.05 Fig.5]. It was improved in bromocriptine and KEE groups.

Bar Test

Table no 05- Effect of bromocriptine and KEE on catalepsy in bar test (Catalepsy score in seconds)

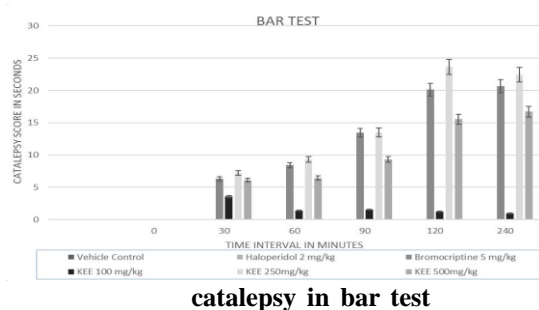
Time in mins.	Vehicle control	Haloperidol control 2 mg/kg	Bromocriptine 5mg/kg	KEE 100mg/kg	KEE 250mg/kg	KEE 500mg/kg
0	0 ± 0	0 ± 0	0 ± 0	0 ± 0	0 ± 0	0 ± 0
30	0 ± 0	6.32 ± 0.27	3.56 ± 0.24	7.24 ± 0.54	6.12 ± 0.32	6.50 ± 0.2
60	0 ± 0	8.42 ± 0.32	1.42 ± 0.45	9.32 ± 0.62	6.45 ± 0.21	4.14 ± 0.1
90	0 ± 0	13.47 ± 0.82	1.53 ± 0.64	13.49 ± 0.21	9.32 ± 0.46	3.33 ± 0.98
120	0 ± 0	20.11 ± 0.63	1.24 ± 0.47	23.63 ± 0.34	15.54 ± 0.24	2.13 ± 0.45
240	0 ± 0	20.66 ± 0.57	0.96 ± 0.23	22.41 ± 0.69	16.70 ± 0.58	2.13 ± 0.89

In this research, the effects of Bromocriptine and KEE on catalepsy, as determined by the bar test at various time intervals, are investigated. During the whole trial, the vehicle control group exhibited no signs of catalepsy. The catalepsy scores of the haloperidol control group increased significantly from 6.32 seconds at 30 minutes to 20.66 seconds after 240 minutes. With a peak of 3.56 seconds at 30 minutes and a tumbling down to 0.96 seconds at 240 minutes, bromocriptine drastically reduced catalepsy scores across the board. With a peak at 23.63 seconds at 120 minutes and a modest drop at 240



minutes, KEE at 100 mg/kg demonstrated higher catalepsy scores compared to the Haloperidol control. With a high of 16.70 seconds at 240 minutes, KEE at 250 mg/kg demonstrated moderate catalepsy scores in comparison to Haloperidol. With a maximum score of 6.50 seconds at 30 minutes, 2.13 seconds at 120 minutes, and stability at 240 minutes, KEE at 500 mg/kg significantly reduced catalepsy scores, comparable to Bromocriptine. This indicates that while bromocriptine is beneficial in reducing catalepsy, greater dosages of KEE also have encouraging results, with a dosage of 500 mg/kg showing the most promise.

Fig no 06- Effect of bromocriptine and KEE on



catalepsy in bar test

The haloperidol group showed an increase in cataleptic time at intervals of 0, 30, 60, 90, 120, and 240 min. Bromocriptine and KEE groups showed a decrease in catalepsy with an increase in the time interval. In comparison, the KEE high dose (500 mg/kg) was found to maximally inhibit catalepsy as compared to low (100 mg/kg) and intermediate (250 mg/kg) groups.

Biochemical Studies-

Determination of Dopamine concentration by UV spectrophotometer-

Table No 06- Effect of bromocriptine and KEE on dopamine concentration using UV

Treatment Groups	Concentration of Dopamine (µg/ml)
Vehicle control	13.27 ± 0.3900
Inducing Haloperidol 2 mg/kg	11.27 ± 0.2800
Standard Bromocriptine 5mg/kg	40.19 ± 0.5300 ****
KEE 100 mg/kg	16.92 ± 0.0810 *
KEE 250 mg/kg	27.18 ± 0.6210 *
KEE 500 mg/kg	33.27 ± 0.4880 *

This research uses ultraviolet spectrophotometry to determine how Bromocriptine and KEE affect dopamine levels. The concentration of dopamine in the vehicle control group was 13.27 µg/ml. The fact that haloperidol administration brought the concentration down to 11.27 µg/ml shows that it inhibits dopamine levels. Dopamine concentration was raised to 40.19 µg/ml by bromocriptine, which was a considerable increase. Concentrations of 16.92 µg/ml (100 mg/kg), 27.18 µg/ml (250 mg/kg), and 33.27 µg/ml (500 mg/kg) of dopamine were likewise increased in a dose-dependent manner by KEE. These findings point to the possibility that Bromocriptine and KEE might mitigate the dopamine depletion caused by Haloperidol. However, it is worth noting that higher dosages of KEE exhibited more significant increases in dopamine concentration.

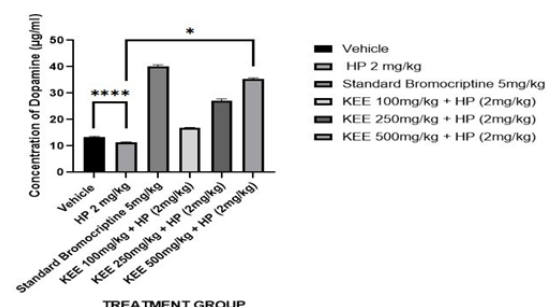


Fig no 07- Effect of bromocriptine and KEE on dopamine concentration using UV

In contrast to the vehicle control group, the haloperidol group demonstrates a reduction in dopamine levels."F" (5,30) =14.64, p <0.05, as seen in the figure. A possible therapeutic approach for PD is shown by the improvement in dopamine levels seen in the bromocriptine and KEE groups. Dopamine concentrations were shown to rise to the greatest extent with the KEE intermediate dosage (250 mg/kg), as compared to the low (100 mg/kg) and high (500 mg/kg) doses.

Determination of Catalase by UV spectrophotometer-

Table No 07 -Effect of KEE on the levels of Catalase (CAT) in the brain of haloperidol-treated mice.

Treatment Groups	CAT (µmoles of H2O2 used/min/mg of protein)
Vehicle	~10
HP 2 mg/kg	~10
Standard Bromocriptine 5mg/kg	~40
KEE 100mg/kg + HP (2mg/kg)	~17
KEE 250mg/kg + HP (2mg/kg)	~27
KEE 500mg/kg + HP (2mg/kg)	~33



Vehicle control	0.1243 ± 0.001284
Inducing Haloperidol 1 mg/kg	0.0378 ± 0.007561
Standard Bromocriptine 5mg/kg	0.1943 ± 0.005948 ****
KEE 100 mg/kg	0.07752 ± 0.001522 *
KEE 250 mg/kg	0.09183 ± 0.003962*
KEE 500 mg/kg	0.1206 ± 0.0120447*

The effect of KEE on catalase (CAT) activity in the brains of mice treated with haloperidol is the focus of this investigation. A CAT activity of 0.1243 μ moles of H₂O₂ utilised per minute per mg of protein was observed in the vehicle control group. Treatment with haloperidol dramatically decreased CAT activity to 0.0378, suggesting an increase in oxidative stress. The significant increase in CAT activity to 0.1943 seen after bromocriptine therapy is indicative of the powerful antioxidant effects of this drug. At 100 mg/kg, 0.09183, and 500 mg/kg, KEE enhanced CAT activity in a dose-dependent fashion, with values of 0.07752, 0.09183, and 0.1206, respectively. This data provides fresh evidence that KEE, at larger dosages, may protect against haloperidol-induced oxidative stress by increasing catalase activity. Haloperidol slowed PD development by lowering catalase levels, which suggests an increase in reactive oxygen species (ROS). While the KEE and bromocriptine groups showed no change in catalase levels, the former did [$F(5,25)=7.892$, $p<0.05$]. Compared to the low (100 mg/kg) and high (500 mg/kg) groups, the KEE intermediate dosage (250 mg/kg) considerably raised the catalase levels.

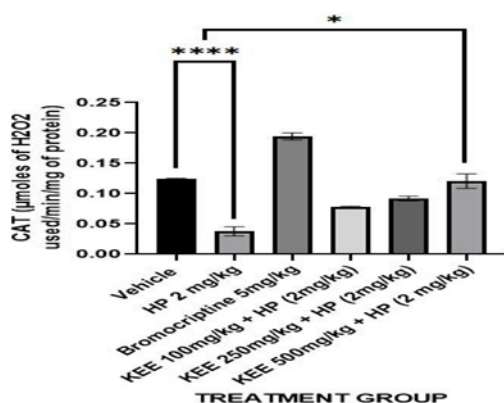


Fig no 08- Effect of KEE on the levels of Catalase (CAT) in the brain of haloperidol-treated mice.

Conclusion –

The plant's phytochemical examination showed that it contained many bioactive chemicals, such as carbohydrates, proteins, saponins, flavonoids, alkaloids, phenols, tannins, and terpenoids; however, glycosides were not found. This suggests that the plant may have medical use. The effects of Bromocriptine and a plant extract (KEE) on muscle tone, gait speed, catalepsy severity, dopamine levels, and catalase (CAT) activity were investigated in animal experiments. Haloperidol reduced motor coordination in the Rotarod test, but KEE and Bromocriptine enhanced it dose-dependently. Similarly, Haloperidol decreased locomotor activity in the Actophotometer test, but KEE and Bromocriptine increased it. The catalepsy bar test showed that KEE, especially at higher dosages (500 mg/kg), decreased the catalepsy caused by Haloperidol. Measuring dopamine concentrations showed that, in contrast to haloperidol, bromocriptine and KEE raised levels, indicating a neuroprotective effect. Finally, CAT activity was shown to be lowered by haloperidol, suggesting oxidative stress. On the other hand, bromocriptine and KEE boosted CAT activity, giving better antioxidant protection at larger dosages. The effects of KEE on Haloperidol-induced motor impairments, dopamine depletion, and oxidative stress were dose-dependent, with higher dosages showing the most promise.

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