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Evaluation of Antiparkinsonian and Neuroprotective Effects of Hydroalcoholic Extract of Capparis Moonii Wight Fruits

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KEYWORDS

Capparis Mooni, Catalepsy, Haloperidol, Bromocriptine, Ascorbic acid , DPPH. PD

ABSTRACT:

The main purpose of present research work is to assess the antiparkinson's and neuroprotective action of hydroalcoholic extract of the Capparis Mooni Wight (HECM) fruits. Parkinson disease comes on second position among neurodegenerative condition and characterised mainly by damage of dopaminergic neuron in the striatum's substantia nigra & dopamine loss (DA). Reported activity of Capparis Mooni plant is Anti-inflammatory, laxative, anti-diabetic, anthelmintic, antibacterial, astringent, digestive activity . Basic purpose of this research is to scientifically verify antiparkinson's and neuroprotective activity of Capparis Mooni using suitable animal models. The antiparkinson's and neuroprotective effects of Capparis Mooni fruits extract was examined using the bar catalepsy, locomotor activity, motor co-ordination, histopathological examination and oxidative parameters. Catalase and other assay were also performed for assessment of biochemical parameters. Using standard as ascorbic acid in UV visible spectrophotometer, evaluation of antioxidant activity by DPPH radical scavenging method were performed. The result of our work indicates that the hydroalcoholic extract significantly reduced haloperidol-induced catalepsy. It can be reported that because of its antioxidant activity and presence of flavonoids, alkaloids and polyphenols that may be essential for antiparkinson's and neuroprotective effect. This finding confirms that Capparis Mooni extract has antiparkinson's and neuroprotective activity.

INTRODUCTION:

Parkinson's disease (PD) is second most common neurodegenerative disorder. Parkinson's disease (PD) caused due to the selective damage of dopaminergic neurons in substantia nigra pars compacta. Oxidative stress plays a major role in the pathophysiology of Parkinson's Disease. Due to oxidative stress Reactive Oxygen Species (ROS) are formed which result in neuronal death of the neurons. This can be detected by decreased levels of endogenous antioxidants. Therefore, the use of antioxidants, along with other protective agents could be a better therapeutic intervention in PD. The current therapeutic agents, because of various side effects, have failed to prove to be a cure-all therapy for the PD patients [1] It is indistinct the cause of that selective cell death. Notably, complexes and aggregation of alpha-synuclein-ubiquitine are known for formation of Lewy bodies.[2-3] Membrane damage occurred by alpha-synuclein may be another Parkinson's disease mechanism.[4] Principal recognized risk factor is age. Gene mutations in α-synuclein (SNCA), tau protein (MAPT), leucine-rich repeat kinase 2 (LRRK2), glucocerebrosidase (GBA) and can also induce inherited PD or increase the risk of developing Parkinson's Disease.[5] The main clinical symptoms of Parkinson's disease are cell death in the basal ganglia of the brain (affecting by the end of life

Journal of Chemical Health Risks

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up to 70 percent of the dopamine secreting neurons in the substantia nigra pars compacta)[6] and the presence of Lewy bodies (protein alpha-synuclein accumulations) in many of the neurons remaining. The death of astrocytes (star-shaped glial cells) and a substantial increase in the number of microglia (another type of glial cell) in the substantia nigra accompany this loss of neurons.[7] Haloperidol induce catalepsy is one of experimental PD model. Main use of haloperidol is in treatment of psychosis as it is neuroleptic drug. It functions by disrupting receptors of dopamine D_2 and D_1 in medium spiny neurons, which contains motor circuit indirect and direct pathways. This causes blockage of striatal dopamine transmission, which leads to abnormal downstream firing in the basal ganglia which produce symptoms of decrease locomotor activity due to muscle stiffness, and catalepsy [8]. Therefore, safer alternative/complementary medicines search for the management of Parkinson's disease is an unfinished task for the researchers in this area of study.

Ayurveda, An Indian traditional medicine method that identified many medicines for use in the diagnosis of PD from indigenous natural sources. *Capparis moonii* W. belonging to the family Capparidaceae/Capparaceae. Widely known as the Large Caper. Waghati in Marathi and Rudanti in Sanskrit. *Capparis moonii* Wight is distributed in Maharashtra, Goa, Karnataka, Tamil Nadu and it is frequently found in Konkan and Sri Lanka which grows normally in the hot climate. Capparis moonii Wight Fruits are sub-globose or ovoid. The fruit contains several large seeds. *Capparis moonii W.* fruits extract contains beta-sitosterol, l-stachyhydrin, rutin, as well as flavonoid, alkaloid, and glycoside.

The herbal medicines are affordable economic, easily available and reflected as time tested and has relatively safer for both human use and environment friendly and hence are used as sources of many lead compounds. One such plant selected for the study was *Capparis Mooni* Wight fruits. It is used for food preparations such as pickle and for treatment of cough, asthma, inflammation and fever. The Capparidaceae family is having antibacterial activity against the staphylococcus aureus, Streptococcus pyogenes, E-coli, Pseudomonas aeruginosa.[9-15] In the present study efforts have been made for evaluation of neuroprotective effect of hydroalcoholic extract of *Capparis Mooni* Wight fruits possesses antiparkinson's and neuroprotective activity due to its well-established antioxidant potential. Hence, in present study, we deliberate the neuroprotective activity of hydroalcoholic extract of *Capparis Mooni* fruits (HECM) (test drug) and bromocriptine (standard drug) in rat model.

MATERIAL AND METHOD:

Capparis Mooni Wight fruits were purchased by local supplier from APMC market, Vashi, Navi Mumbai. The specimen was submitted for authentication and authenticated by Dr. Harshad M Pandit, former Head & Associate Professor of Botany, G. N. Khalsa College, Matunga, Mumbai. After washing the fruits of *Capparis Mooni* were air dried and creased to coarse powder. The powder was passed through sieve No. 40 and used for further studies. The powder was then extracted with ethanol and water under soxhlation 3 cycles of 8 hours to give hydroalcoholic extract of fruits of *Capparis Mooni* Wight. The prepared extract was filtered and evaporated till dryness with a dryer. The percent yield was observed 9.5% w/w. Prepared crude dried extract was put in a suitable container and kept in refrigerator 4°C until use.

A: QUALITATIVE PHYTOCHEMICAL SCREENING:

Phytochemical assessment plays an important part in the standardization of the extracts under investigation and was conducted for detecting the occurrence of various phytoconstituents in the plants under study. Hydroalcoholic fruits extract of *Capparis Mooni* Wight was exposed to preliminary phytochemical evaluation using qualitative chemical tests for detecting the presence of the phytoconstituents like phytosterols, alkaloids, glycosides, tannins, phenolic compounds, carbohydrates, proteins and amino acids etc.

B: Fourier Transform Infrared Spectrophotometer (FTIR) analysis:

Fourier Transform Infrared Spectrophotometer (FTIR) is most powerful tool for identifying the types of chemical bonds (functional groups) present in compounds. FTIR spectrum gives the idea about chemical bond present. By interpretation the infrared

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absorption spectrum, the chemical bonds in a molecule can be determined. FTIR analysis of Hydroalcoholic fruits extract of *Capparis Mooni* was done.

EXPERIMENTAL ANIMALS:

The animals used in present study were healthy, either sex Wistar Albino rats weighing 200-250 gm obtained from Mumbai Veterinary College, Mumbai. Under good hygienic conditions in the registered animal house, the animals were group-housed in standard polypropylene cages (6 rats per cage) and maintained under controlled room temperature (22 +/- 2^0C) and humidity (55 +/- 5%) with 12-hrs light dark cycle with food and water available *ad libitum*. All animal experimental procedures were conducted in accordance with the CPCSEA guidelines. Efforts were made to minimize animal suffering and to use only the number of animals necessary to produce reliable scientific data. The study was performed after approval by the IAEC, Institutional animal ethics committee of Oriental College of Pharmacy (protocol number OCP/ IAEC/2017- 2018/09)

CHEMICALS AND REAGENTS:

1. Hydroalcoholic fruit extract of *Capparis Mooni (HECM)* (100 mg/kg, 200 mg/kg and 300 mg/kg)

2. Standard drug: Bromocriptine (Purchased from Yasfeen Medical stores- Mumbai)

3. Drug for induction of catalepsy: Haloperidol (inj. Serenace, Purchased from Yasfeen Medical stores- Mumbai)

4. Hydrogen Peroxide

5. Distilled water

6. Ascorbic acid

7. DPPH

C: ACUTE ORAL TOXICITY: [16]

The Acute oral toxicity (AOT) test was performed as per guidelines 423 of the Organisation for Economic Co-operation and Development (OECD) by taking nine animals in the weight range of 140 to 200g were first brought and acclimatized for about a week. They were then grouped in three groups as lower concentration (50mg/kg), intermediate (300mg/kg) and higher concentration (2000mg/kg) and fasted 2 hr before the administration of the fruit extract. After this they were observed for any abnormalities at 15, 30, 60, 120 and 180 minutes on 1st day and then once a day till the 14th day.

D: PHARMACOLOGICAL EVALUATION:

After a one-week acclimation period rats were divided randomly into six groups (n = 6), viz., vehicle control (vehicle treated), haloperidol control, bromocriptine, and HECM treated group low dose (100 mg/kg), intermediate dose (200 mg/kg), high dose (300 mg/kg). Bromocriptine and HECM were administered orally for 21 days. Animals from all groups except group I were challenged with haloperidol (1 mg/kg of body weight) by intraperitonial route after 30 m of treatment for 21 days. Catalepsy, locomotor activity, and motor impairment were measured in animals. After study completion, the animals were sacrificed by decapitation, and brain samples were dissected and washed with ice-cold saline and used for histopathology and estimation of oxidative parameters.

ESTIMATION OF BEHAVIOURAL PARAMETERS:

1 – Bar test: [17]

The Bar Test was used for assessment of catalepsy. In the bar test, the animal's front paw was positioned on a horizontal bar situated 3 cm and 5 cm above and alternately parallel to the base. Time was recorded at which the animal removed its paw from the bar. The catalepsy rating was given as follows:

STEP I: The rats were removed from the cage and placed on a floor. A 0.5 score was given if the mice failed to move when touched or pressed gently on the back.

STEP II: Alternately, the front paws of the rat were placed on a 3-cm-high block. If the animal failed to correct the position within 15 sec, a score of 0.5 for each paw was applied to the score of step I.

STEP III: Alternately, the front paws of the animal were placed on a 5-cm-high block, if the animal failed to correct the position within 15 sec, a score of 1 for each paw was applied to the scores of steps I and II.

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

2 – Motor Co-ordination Test (Rotarod Test): [18]

Motor coordination test was performed by using Rotarod apparatus. Prior to treatment the animals were put on the moving rod and the rats which stays on the rod without dropping for 120 seconds were selected for the analysis. Before and after the treatment, the time taken by animals to fall from the rotating rod was noted. With an acceleration rate of 20 rpm, the starting speed of the rota rod was set to 4 rpm. The maximum velocity was 40 rpm.

3 – Test for Locomotor activity (Actophotometer): [19]

Actophotometer was used for measurement of the behavior of the locomotor. Apparatus comprises of a cage with six photocells and six lights that are situated in the outer bottom periphery in such that only one beam is blocked at a time by single animal. When the light rays drop on the photocells, photocells get activated.The light beam is interrupted when the animal crosses the light beam, the number of cut-off has been recorded for 10 minutes.

E: BIOCHEMICAL TEST:

1: Estimation of catalase (CAT) principle:[20]

Brain sample preparation: Once performing the Bar test, Motor Coordination Test and Locomotor Behavior in Haloperidol-induced Parkinson's animals from each group were euthanized by using carbon dioxide chamber; brains were rapidly removed and placed in ice-cold saline. The tissues in the 0.1 M Phosphate Buffer (pH 8) were weighed and homogenized. For examination of oxidative parameters, samples of rat brain homogenates were collected in various test tubes. The supernatant was used for these studies.

Assay:

UV was used for measurement of catalase activity. 0.1 ml of supernatant was added to the cuvette containing 1.9 ml of 50 mM phosphate buffer (pH 7.0). The reaction was initiated by the addition of 1.0 ml of freshly prepared 30 mM H_2O_2. The spectrophotometric determination t of the rate of decomposition of H_2O_2 was based on changes in absorbance at 240 nm. The activity of catalase was expressed as units/mg protein. The reaction occurs instantly after the addition of H_2O_2. The absorbance was measured at a wave length of 240 nm. Solutions were well mixed and after 15 seconds (t1) the first absorbance (A1) was done and after 30 seconds (t2) the second absorbance (A2).

2: DETERMINATION OF MALONYLDIALDEHYDE (MDA): [21]

Supernatant was taken in a tube from the tissue homogenate. 0.5 ml of Trichloroacetic acid (TCA) was added to it, followed by 0.5 ml. of 8% Thiobarbituric acid (TBA) reagent. The tubes were placed in the water bath for 30 min. at 80ºC after covering with aluminum foil. After 30 min. the tubes were taken out and put in the cold water for 30 min. These tubes were centrifuged for 15 min at 3000 rpm. The absorbance was taken at 540 nm, at room temperature against appropriate blank solution. MDA value was expressed as moles MDA/mg of protein.

3: Glutathione Peroxidase Assay (GHS): [22]

3-ml cuvette containing 2.0 ml of phosphate buffer (75 mmol/L, PH 7.0) , 50µl of (60mmol/L) glutathione reductase solution, 50µL of (0.12 mol/L) NaN3, 0.1 ml of (0.15mmol/L) Na2 EDTA ,100µL of (3.0 mmol/L) NADPH, and 100µL of tissue supernatant was added. Water was added to make a total volume of 2.9 ml. The reaction was started by the addition of 100µL of (7.5 mmol/L) H_20_2, and the conversion of NADPH to NADP was monitored by a constant recording of the change of absorbance at 340 nm at 1-min interval for 5 min. Enzyme activity of GSHPx was expressed in terms of mg of proteins.

F: EVALUATION OF ANTIOXIDANT ACTIVITY BY DPPH RADICAL SCAVENGING METHOD: [23]

Free radical scavenging activity of hydroalcoholic fruit extract of *Capparis Mooni* were measured by 1, 1-diphenyl-2-picryl hydrazyl (DPPH). Solution of DPPH in ethanol 0.1mM was prepared. This solution (1 ml) was added to 3 ml of different extracts in ethanol at different concentration (5, 10, 20, 40 µg/ml). Here, only those extracts which are solubilise in ethanol were used and different concentrations were prepared by dilution method. The mixture was then shaken vigorously and allowed to for 30 min at room temp. then, absorbance was measured at 517 nm. by using spectrophotometer

Journal of Chemical Health Risks

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

(UV-VIS). Ascorbic acid was used as Reference standard compound and experiment was done in double.

G: Histopathological examination: -

After treatment and the studies rats were sacrificed and brains were isolated and fixed in 10% phosphate-buffered formalin, dehydrated in graded alcohol, and embedded in paraffin sections. of 3-4 micron thickness was obtained. These sections were placed on glass slides, and counterstained with hematoxylin and eosin. The slides were observed under a light microscope.

H: STATISTICAL ANALYSIS:

All analytical measures like cataleptic behavior, muscle coordination behavior, locomotor activities were characterised in the table, and the graph was denoted as mean ±S.E.M. (n=6). One way ANOVA statistical method followed by Tukey's multiple comparisons was adopted. A significant difference was considered b/w group when $p<0.05$. GraphPad Prism 8.0.2 software was used for all analyses.

RESULT:

A) Phytochemical analysis:

The phytochemical analysis of extract showed that the hydroalcoholic fruit extract of *Capparis Mooni* (HECM) indicates presence of carbohydrates, saponins, flavonoids, alkaloids, phenolic compounds and tannins.

Table 1: Result of qualitative Phytochemical evaluation of powdered fruits of *Capparis Mooni.*

Phytochemical Test/Reagent	*Capparis moonii W.* Extract
Alkaloids	
Mayer's test	+
Dragendroff's test	+
Wagner's test	+
Carbohydrates	
Molisch's test	+
Barford's test	-
Benedicts test	-
Glycosides	
Molisch's test after hydrolysis	+
Phytosterols	
Liebermann's Burchard's test	+
Fixed Oils and Fats - Spot test	+
Saponins - Foam test	+
Phenolic Compounds and Tannins	
Ferric chloride test	+
Lead acetate test	+
Proteins and Amino Acids	
Biuret test	-
Ninhydrin test	-
Flavonoids - Shinoda test	+
Yield (%W/W)	**9.5 %**

Present (+)/Absent (-)

B) Fourier Transform Infrared Spectrophotometer (FTIR) analysis:

The functional groups in the hydroalcoholic fruit extract of *Capparis mooni* W. found to contain Carboxylic acids, Aromatic compound, Alkyl aryl ether, Alkene exhibiting 13 bands (Figure 1) in the frequency of 655 cm-1 to 3369 cm-1

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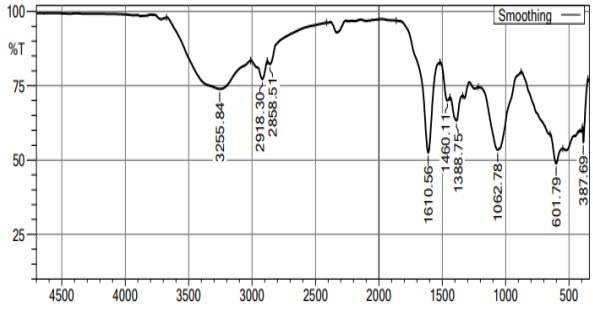

Fig 1: FTIR- analysis of Hydroalcoholic fruit extract of *Capparis Mooni*.

C) Acute oral toxicity studies:

Acute oral toxicity (AOT) analysis was performed as recommended in OECD Guideline 423 and the findings showed that no lethal signs were found in clinical parameters during an acute toxicity test of up to 2000mg/kg. Therefore, it shows that the median lethal dose (LD50) of Capparis moonii W. dried fruits alcoholic extract equals 2000mg/kg. Following detailed survey of different research papers and directed by the guide, doses were selected as 100 mg/kg and 200 mg/kg and 300 mg/kg for the test groups.

D) Haloperidol induced catalepsy in animals:

1: Bar test:

In bar test **Table 02**, haloperidol control group significantly increases cataleptic score as compared to the vehicle control group. Bromocriptine and HECM 300 mg/kg showed significant inhibition against catalepsy by decreasing cataleptic score.

Table 02: Effect of bromocriptine and HECM on catalepsy in bar test

Time interval in mins	**Mean ± SEM (Cataleptic score)**					
	Vehicle control	**Haloperidol control**	**Bromocriptine**	**HECM 100 mg/kg**	**HECM 200 mg/kg**	**HECM 300 mg/kg**
0	0.00 ± 0.00	1.8 ± 0.01#	0.85 ± 0.01***	1.25 ± 0.01***	1.2 ± 0.01***	0.91 ± 0.02***
30	0.00 ± 0.00	1.95 ± 0.01#	1.8 ± 0.01***	4.18 ± 0.04***	2.54 ± 0.01***	1.25 ± 0.01***
60	0.00 ± 0.00	85.87 ± 0.78#	2.76 ± 0.01***	4.95 ± 0.01***	4.76 ± 0.01***	3.78 ± 0.01***
120	0.00 ± 0.00	135.8 ± 0.20#	4.23 ± 0.03***	7.3 ± 0.03***	5.26 ± 0.02***	5.12 ± 0.02***
180	0.00 ± 0.00	185.4 ± 0.11#	4.85 ± 0.02***	13.43 ± 0.02***	5.67 ± 0.04***	6.75 ± 0.02***
240	0.00 ± 0.00	198.86 ± 0.03#	7.26 ± 0.04***	18.3 ± 0.01***	10.84 ± 0.02***	8.89 ± 0.01***

All values are expressed in Mean ± SEM (n = 6).Significance:****indicated P≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

Journal of Chemical Health Risks

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

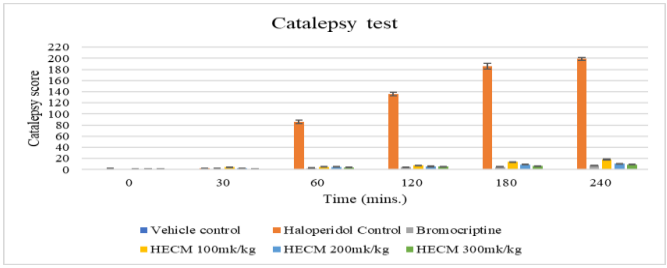

Fig. 02: Effect of bromocriptine and HECM on catalepsy in bar test

2: Motor co-ordination test:

Fall of time from rotarod was significantly decreased in haloperidol treated group as compared to the vehicle control group and it was significantly improved with Bromocriptine, HECM 200 and 300 mg/kg. [Table 03]

Table 03: Effect of bromocriptine and HECM on motor co-ordination test using rotarod

TREATMENT GROUPS	FALL OF TIME(SECS) MEAN±SEM
Vehicle Control	105.66 ± 1.40
Haloperidol Control	15.85 ± 0.04#
Bromocriptine	95.45 ± 0.05**
HECM 100 mg/kg	56.80 ± 0.04**
HECM 200 mg/kg	65.10 ± 0.03**
HECM 300 mg/kg	71.40 ± 0.04**

All values are expressed in Mean ± SEM (n = 6). Significance: ****indicated P≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

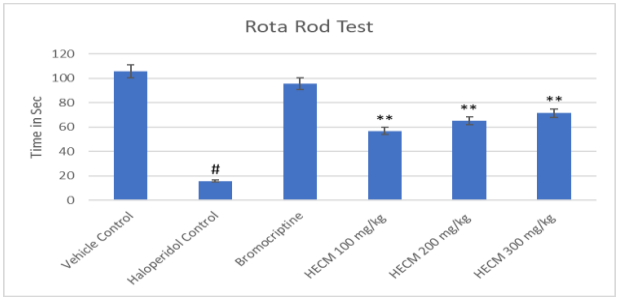

Fig. 03: Effect of bromocriptine and HECM on motor co-ordination test using rotarod

3: Test for locomotor activity:

Spontaneous motor activity was significantly decreased in haloperidol treated group as compared to the vehicle control group. Bromocriptine and HECM 300 mg/kg, significantly increased the locomotor activity as compared to haloperidol treated animals.

Table 04: Effect of bromocriptine and HECM on locomotor activity using actophotometer

TREATMENT GROUPS	AMBULATIONS COUNTS/10 MIN MEAN±SEM
Vehicle Control	210.45 ± 0.10
Haloperidol Control	40.20 ± 0.91#
Bromocriptine	156.35 ± 0.06***
HECM 100 mg/kg	74.16 ± 0.90***
HECM 200 mg/kg	93.06 ± 0.05***
HECM 300 mg/kg	105.40 ± 0.04***

All values are expressed in Mean ± SEM (n = 6). Significance:****indicated P≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

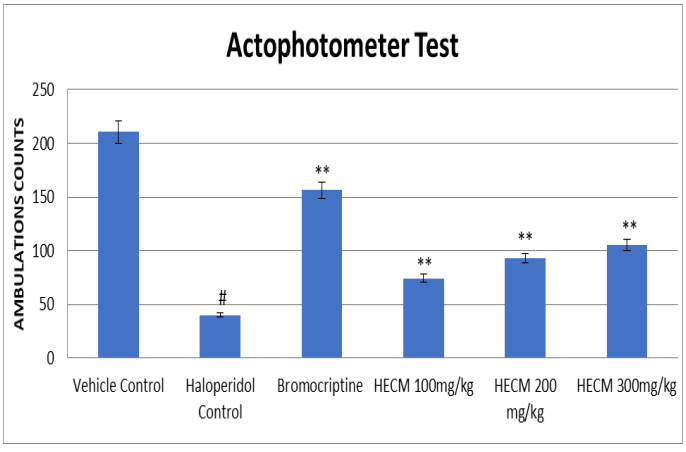

Fig. 04: Effect of bromocriptine and HECM on locomotor activity using actophotometer

Journal of Chemical Health Risks

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

E) BIOCHEMICAL PARAMETERS:

1: Estimation of CATALASE by UV:

In this test **Table 05**, haloperidol control group significantly decrease in catalase level as compared to the vehicle control group. Bromocriptine and HECM 300 mg/kg showed significant increase in catalase level.

Table 05: Effect of bromocriptine and HECM on Catalase level using UV.

TREATMENT GROUPS	UNIT/mg MEAN±SEM
Vehicle Control	35.72±0.23**
Haloperidol Control	23.35 ±1.83
Bromocriptine	18.38±0.43**
HECM 100 mg/kg	22.35±0.45**
HECM 200 mg/kg	16.23±0.33**
HECM 300 mg/kg	14.45±0.25**

All values are expressed in Mean ± SEM (n = 6). Significance:****indicated p≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

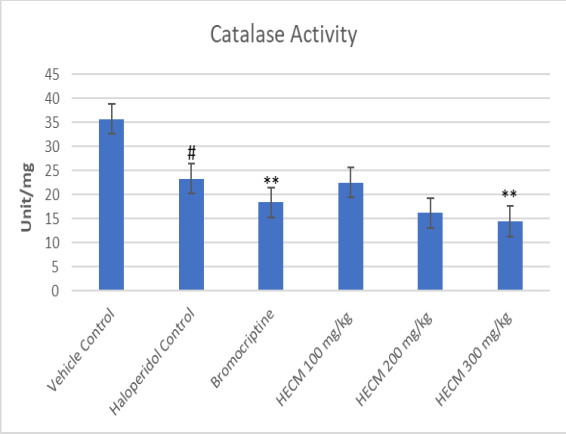

Fig. 05: Effect of bromocriptine and HECM on CATALASE level using UV.

2: Determination of on MDA level:

In this test **Table 06**, haloperidol control group significantly decrease MDA level as compared to the vehicle control group. Bromocriptine and HECM 300 mg/kg showed significant decrease MDA level.

Table 06: Effect of bromocriptine and HECM on MDA level

TREATMENT GROUPS	UNIT/mg MEAN±SEM
Vehicle Control	12 ± 0.439
Haloperidol Control	29.75 ± 1.79#
Bromocriptine	11.1 ± 0.631
HECM 100 mg/kg	23.55 ± 1.271
HECM 200 mg/kg	15.5 ± 2.145**
HECM 300 mg/kg	13.4 ± 2.025 **

All values are expressed in Mean ± SEM (n = 6).

Significance:****indicated p≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

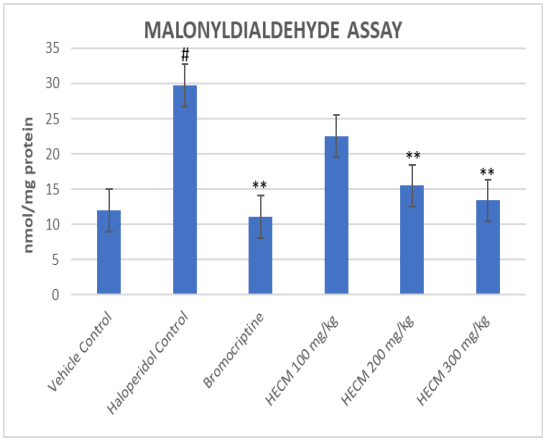

Fig. 06: Effect of bromocriptine and HECM on MDA level

3: Estimation of GHS level:

In this test **Table 07**, haloperidol control group significantly decrease in GHS level as compared to the vehicle control group. Bromocriptine and HECM 300 mg/kg showed significant increase in GHS level.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

Table 07: Effect of bromocriptine and HECM on GHS level.

TREATMENT GROUPS	UNIT/mg MEAN±SEM
Vehicle Control	3.6 ± 0.19
Haloperidol Control	2.04 ± 0.12
Bromocriptine	3.4 ± 0.13
HECM 100 mg/kg	2.9 ± 0.05
HECM 200 mg/kg	3.1 ± 0.07
HECM 300 mg/kg	3.35 ± 0.07

All values are expressed in Mean ± SEM (n = 6).

Significance:****indicated p≤0.0001,***indicated P ≤0.001,** indicated P≤0.01,*indicates P≤0.05 when compared with negative control.

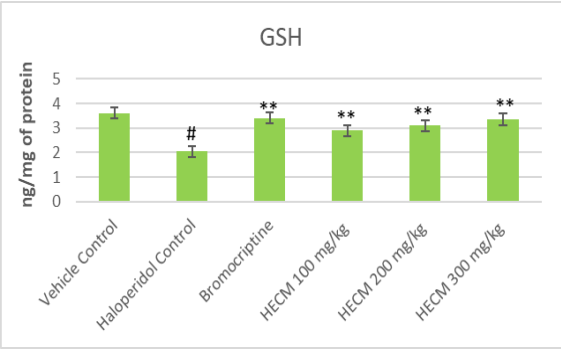

Fig. 07: Effect of bromocriptine and HECM on GHS level

F) EVALUATION OF ANTIOXIDANT ACTIVITY BY DPPH RADICAL SCAVENGING METHOD:

Absorbance of fruit extract *Capparis Mooni* W. fruit with standard ascorbic acid at 517 nm by
UV visible spectrophotometer (DPPH scavenging assay method)

Table 08. Absorbance of *Capparis Mooni* W. fruit with standard ascorbic acid

CONCENTRATION [µg/ml]	ASCORBIC ACID (abs)	EXTRACT (abs)
5	0.038	0.252
10	0.017	0.232
20	0.015	0.248
40	0.01	0.229

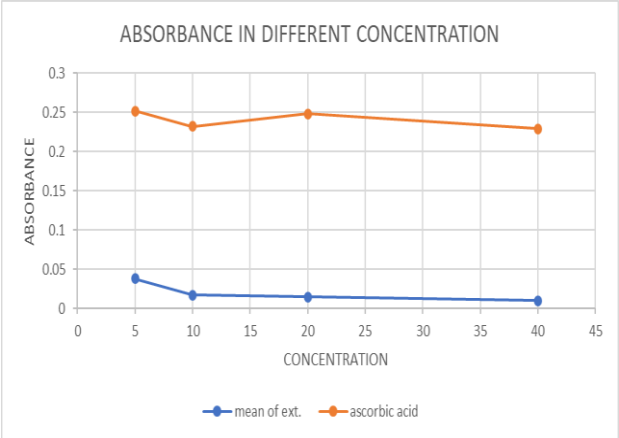

Fig 08: Absorbance at different concentration of extract and ascorbic acid.

In **Table 09**, DPPH scavenging effect (%) or Percent inhibition = A0 - A 1 / A0 × 100.

Where A0 was the Absorbance of control reaction and A1 was the Absorbance in presence of test or standard sample.

CONTROL Reading: 2.102

Table 09. % Inhibition of *Capparis Mooni* Fruits with ascorbic acid

CONCENTRATION [µg/ml]	ASCORBIC ACID (% Inhibition)	EXTRACT (% Inhibition)
5	93.33%	88.01%

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

10	93.62%	88.20%
20	93.91%	88.96%
40	95.62%	89.10%

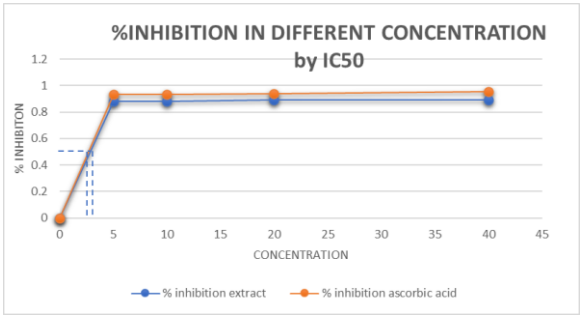

Fig 09: %Inhibition of Extract and Ascorbic Acid at Different Concentration.

G) Effect of bromocriptine and HECM on Histopathology of brain

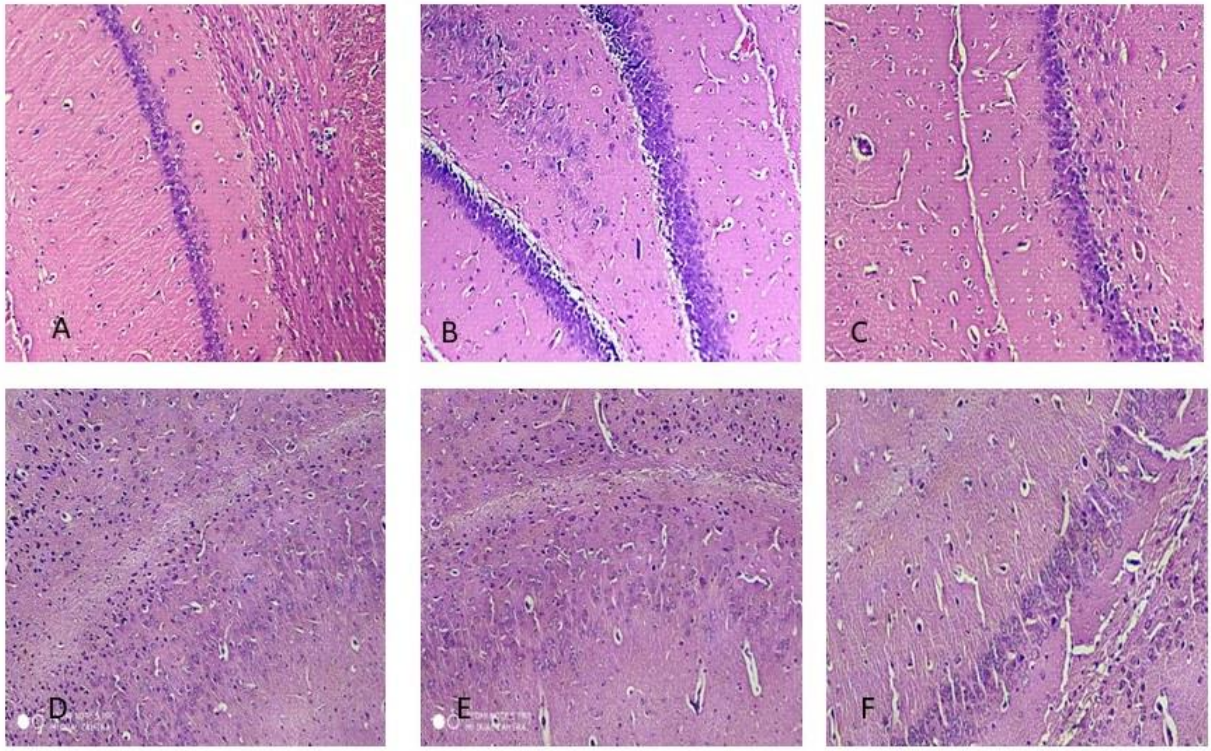

Fig 10: Effect of bromocriptine and HECD on Histopathology of brain A= Normal control, B= Haloperidol control , C= std (bromocriptine), D= HECD 100 mg/kg, E= HECD 200 mg/kg, F= HECD 300 mg/kg. (H&E sectioning, 200x Magnification).

DISCUSSION:

Major symptoms of Parkinson's Disease include catalepsy (rigidity in movements), akinesia (slowing of movement), tremors and memory loss. Amongst above all catalepsy is one of the major symptoms which make the life of Parkinson's Disease patient uneasy. Well-known dopamine (D_2) receptor agonist Bromocriptine is commonly used to improve the symptoms related to rigidity. Hence, Bromocriptine was used as standard in the present study to compare the efficiency of the models. Haloperidol-induced catalepsy in rat bear a resemblance to the deficiency of dopamine in nigrostriatal pathway that give rise to catalepsy. Catalepsy was induced in rat by intraperitoneal (i.p.)

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JCHR (2026) 16(2), 4028-4040 | ISSN:2251-6727

administration of haloperidol. This cataleptic behavior induced by haloperidol and the protective effect of standard (bromocriptine) and HECM used was evaluated by using bar test, rotarod apparatus and actophotometer. The vehicle group values are depicting the basal values of muscle tone and locomotor activity in rats. The disease group i.e. haloperidol challenged animals showed significant ($p<0.05$) reduction in rotarod and locomotor activity when compared to control group animals and depicting the successful induction of catalepsy in rats after haloperidol challenge. The rotarod activity and locomotor activity of animals receiving only haloperidol challenge showed significant reduction ($p<0.05$) in movement when compared with the animals in vehicle group. The rotarod activity was performed to assess the muscle coordination and balancing ability in rats while on rotating rod and was evaluated in terms of latency of fall[24],. Dose dependent increase in locomotor activity was observed among the animals treated with hydroalcoholic extract of fruits of *Capparis Mooni* W. when compared to control group which provided more evidence of the ameliorative effect of *Capparis Mooni* on PD.

Dysfunction of Mitochondrial complex-1 causes generation of oxidative stress and plays an imperative role in the pathogenesis of Parkinson's Disease. The endogenous antioxidants like GSH and CAT are valuable components that combat with free radicals and neutralize them before they can damage the cells and hence prevent damage to cell proteins, lipids and carbohydrates. One of the important neuroprotective enzymes in the brain is Glutathione peroxidase. It behaves as a scavenger of H_2O_2 produced by cellular metabolism besides balancing the composition and disintegration of H_2O_2 in normal conditions. The decreased level of glutathione is the limiting factor in the elimination of H_2O_2. However, in Parkinson's disease, glutathione is reduced extensively in the substantia nigra because of neuronal loss. By considering the antioxidant property of Capparis Mooni, it is assumed that the beneficial effect of Capparis Mooni fruits in aforesaid models of PD could be due to its lessening effect on the oxidative stress in the brain. To confirm the assumption, the levels of catalase, reduced glutathione, and MDA were estimated in the animal brain. Lipid peroxidation is known to take place in a variety of pathological conditions including neurodegenerative disease. The MDA levels are revealing of lipid peroxidation, which was found to be increased in brain homogenate of disease group animal in haloperidol induced catalepsy models. The GSH and CAT levels were found to be decreased in brain homogenate of disease group animal in catalepsy model. present study show that oxidative stress is ameliorate by *Capparis Mooni* Wight fruits extract in dose dependant manner through its antioxidant effect, continuous treatment with *Capparis Mooni* Wight fruits extract significantly decrease oxidative stress, as assessed by increased GSH, and catalase activity in the brain and decreased malondialdehyde levels in the brain, which may be responsible for antiparkinsonian and neuroprotective effect.

CONCLUSION:

Capparis Mooni Wight fruits exhibited significant antiparkinsonian and neuroprotective activity in haloperidol rat model. It appears to be the most promising plant due to its potential antioxidant activity. The predictable mode of action of this plant may be due to antioxidant activity as well as due to its presence of flavonoids and polyphenols. These findings provide evidence for its use as antiparkinsonian medication, including prevention of PD, improvement of PD symptoms. The enormous scope lies in exploring the bioactive present in HECM which can be responsible for the neuroprotective activity and to establish the exact mode of action.

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AUTHORS' CONTRIBUTIONS

Dr. Ashish Kumar Sharma and Dr. Sayyed Mateen directed and guided with designing the study, making of protocol, and obsedrved the work done. Literature search extraction, phytochemical screening, performed the animal models, biochemical test and DPPH, and completed the manuscript writing was done by Mr.

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CONFLICTS OF INTEREST

We announce we do not have conflicting interests.

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