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Original Research Article

Exploring the Neuroprotective Efficacy of Hydroalcoholic Extract of Capparis decidua (Forssk.) Edgew. Fruits

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Abstract: The aim of present study is to evaluate the neuroprotective activity of hydroalcoholic extract of the Capparis Decidua (HECD) fruits. Parkinson disease is second most common neurodegenerative condition characterised by dopaminergic neuron loss in the striatum's substantia nigra & dopamine loss (DA). Anti-inflammatory, laxative, anti-diabetic, anthelmintic, antibacterial, astringent, digestive are the traditional use of Capparis Decidua plant. Basic purpose of this research is to scientifically verify neuroprotective activity of Capparis Decidua using suitable animal models. The neuroprotective effects of Capparis Decidua fruits extract was examined using the bar catalepsy, locomotor activity, motor co-ordination and also oxidative parameters. Catalase and other assay were also carried out to assess biochemical parameters. Using standard as ascorbic acid in UV visible spectrophotometer, evaluation of antioxidant activity by DPPH radical scavenging method were performed. The outcome 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 neuroprotective effect. This finding confirms that Capparis Decidua extract has neuroprotective activity.

Keywords: Capparis Decidua, Catalepsy, Haloperidol, Bromocriptine, DPPH, Ascorbic acid.

INTRODUCTION

The second most common neurodegenerative condition is Parkinson's disease (PD). 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 unclear the cause of that selective cell death. Notably, complexes and aggregates of alpha-synuclein-ubiquitine are known for aggregation within affected neurons in Lewy bodies.[2] Impaired axonal transport of alpha-synuclein may also induce its build-up in Lewy bodies.[3] Membrane damage caused by alpha-synuclein may be another Parkinson's disease mechanism.[4] Age is the principal recognized risk factor. Mutations in genes such as α-synuclein (SNCA), leucine-rich repeat kinase 2 (LRRK2),

glucocerebrosidase (GBA), and tau protein (MAPT) can also induce inherited PD or increase the risk of developing PD.[5] The key clinical symptoms of Parkinson's disease are cell death in the basal ganglia of the brain (affecting by the end of life 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 is experimental PD model. Haloperidol is used for the treatment of psychosis as it is neuroleptic drug. It functions by disrupting receptors of dopamine D2 and D1 in medium spiny neurons, which include motor circuit indirect and direct pathways. This leads to blockage of striatal dopamine transmission, which causes abnormal downstream firing in the basal ganglia as symptoms of muscle stiffness, locomotive activity and catalepsy [8].Hence, the search for safer alternative/complementary medicines for the management of PD is an unfinished task for the researchers in this area of study.

The herbal medicines are considered as time tested and has relatively safer for both human use and environment friendly and hence are used as sources of many lead compounds. They are also economic, easily available and affordable. One such plant selected for the study was *Capparis Decidua* fruits is used for food preparations such as pickle and also treatment for cough, asthma, inflammation and fever. The Capparidaceae family shows the antibacterial activity against the staphylococcus aureus, Streptococcus pyogenes, E-coli, Pseudomonas aeruginosa.[9] It is a small, succulent annual, mat-forming species. The authors have reported that various parts of *Capparis Decidua* is having alkaloids, glycosides, terpenoids, sterols, flavanoids, phenols and fatty acids. Compounds like N-triacontane, n-triacontanol, n-pentacosane, 6-(1-hydroxy-non-3-enyl)tetrahydropyran-2-one,2-carboxy-1-dimethylpyrrolidine,ß-sitosterol, ß-carotene, Glucosinolates, Ascorbic acid, proteins, carbohydrates, Calcium, Potassium, Phosphorous, Zinc, Iron and Manganeseare located in aerial components of flowers, fruits, stems and seeds.[10]Anti-inflammatory, laxative, anti-diabetic, anthelmintic, antibacterial, astringent, digestive, diaphoretic and anodyne are found to be shown in Capparis plant. Numerous diseases, such as rheumatism, asthma, diabetes, liver disorders, hypercholesterolemia, hypertension and microbial infections are stated to have beneficial effects.[11] This study was designed to investigate if hydroalcoholic extract of *Capparis Decidua* fruits possesses neuroprotective activity due to its well-established antioxidant potential. Hence, in this research, we studied the neuroprotective activity of hydroalcoholic extract of *Capparis Decidua* fruits (HECD) (test drug) and bromocriptine (standard drug) in rat model.

MATERIAL AND METHOD:

Capparis Decidua plant was collected from Jalgaon and specimen was submitted to the Department of Botany, Blatter Herbarium, St. Xavier's College, Mumbai 400001. The plant material was authenticated by Dr. Rajendra Shinde. The fruits of *Capparis Decidua* were washed, air dried for 2 days and crushed to coarse powder. The powder obtained was passed through sieve no. 40 and used for further studies. The fruits were cut into small pieces and dried at controlled temperature 450C and powdered. The powder was then extracted with ethanol and water under soxhlation 3 cycles of 8 hours to give hydroalcoholic extract of fruits of *Capparis Decidua*. The extract was filtered and evaporated to dryness with a dryer. The percent yield was observed 7.5% w/w. These crude dried extracts were put in a suitable container and kept in refrigerator 4°C until use.

A: QUALITATIVE PHYTOCHEMICAL SCREENING:

Phytochemical evaluation plays an important role in the standardization of the crude extracts and was carried out for detecting the presence of various phytoconstituents in the plants under investigation. Hydroalcoholic fruits extract of *Capparis Decidua* was subjected to preliminary phytochemical evaluation using qualitative chemical tests for detecting the presence of the phytoconstituents like alkaloids, glycosides, tannins, phenolic compounds, phytosterols, carbohydrates, proteins and amino acids etc.

B: Fourier Transform Infrared Spectrophotometer (FTIR) analysis:

Most powerful tool for identifying the types of chemical bonds (functional groups) present in compounds is Fourier Transform Infrared Spectrophotometer (FTIR). FTIR spectrum gives the idea about chemical bond present. By interpreting the infrared absorption spectrum, the chemical bonds in a molecule can be determined. FTIR analysis of Hydroalcoholic fruits extract of *Capparis Decidua* was done.

EXPERIMENTAL ANIMALS:

The animals used for the experiments were healthy, either sex Wistar Albino rats weighing 200-250 gm procured from Mumbai Veterinary College, Mumbai. The animals were group-housed in standard polypropylene cages (6 rats per cage) under good hygienic conditions in the registered animal house 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 experiments 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 approved by the Institutional animal ethics committee (protocol number OCP/ IAEC/2017-2018/09)

CHEMICALS AND REAGENTS:

1. Hydroalcoholic fruit extract of *Capparis Decidua (HECD)* (100 mg/kg, 200 mg/kg, 300 mg/kg)
2. Standard drug: Bromocriptine (Purchased from Yasfeen Medical stores- Mumbai)
3. Drug to induce catalepsy: Haloperidol (inj. Serenace, Purchased from Yasfeen Medical stores-Mumbai)
4. Hydrogen Peroxide
5. Distilled water
6. Ascorbic acid
7. DPPH

C: PHARMACOLOGICAL EVALUATION:

After a one-week acclimation period rats were randomly divided into six groups (n = 6), viz., vehicle control (vehicle treated), haloperidol control, bromocriptine, and HECD treated group low dose (100 mg/kg), intermediate dose (200 mg/kg), high dose (300 mg/kg). Bromocriptine and HECD 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 completion of study, the animals were sacrificed by decapitation, and brain samples were dissected and rinsed in ice-cold saline and used for estimation of oxidative parameters.

ESTIMATION OF BEHAVIOURAL PARAMETERS:

1 – Bar test: [12]

To assess catalepsy, the Bar Test was used. In the bar test, the front paw of the animals 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 rating for catalepsy was given as follows:

STEP I: The rats were removed from the home cage and placed on a floor. A score of 0.5 was assigned 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.

2 – Motor Co-ordination Test (Rotarod Test): [13]

Motor coordination test was performed by using Rotarod apparatus. Prior to treatment The animals were put on the moving rod and the rats which remains 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): [14]

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

D: BIOCHEMICAL TEST:

1: Determination of catalase (CAT) principle:[15]

Preparation of brain sample: After 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 put 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 triggered by the addition of 1.0 ml of freshly prepared 30 mM H_2O_2. The spectrophotometric measurement 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 immediately 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 read and after 30

seconds (t2) the second absorbance (A2).

2: DETERMINATION OF MALONYLDIALDEHYDE (MDA): [16]

From the tissue homogenate supernatant was taken in a tube. 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 covered with aluminum foil and kept in the water bath for 30 min. at 80ºC. After 30 min. the tubes were taken out and placed 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): [17]

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 were 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) H202, and the conversion of NADPH to NADP was monitored by a continuous 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.

E: EVALUATION OF ANTIOXIDANT ACTIVITY BY DPPH RADICAL SCAVENGING METHOD: [18]

Free radical scavenging activity of hydroalcoholic fruit extract of *Capparis Decidua* 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 (UV-VIS). Reference standard compound being used was ascorbic acid and experiment was done in double.

G: STATISTICAL ANALYSIS:

All analytical measures like cataleptic behavior, muscle coordination behavior, locomotor activity were represented 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 $p<0.05$. All analyses were done with GraphPad Prism 8.0.2 software

RESULTS

A) Phytochemical analysis:

The phytochemical analysis of extract revealed that the hydroalcoholic fruit extract of *Capparis Decidua* (HECD) shows presence of carbohydrates, saponins, flavonoids, alkaloids, phenolic compounds and tannins.

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

Phytochemicals	Observations
Carbohydrates	+
Proteins	-
Steroids	+
Saponins	+
Flavonoids	+
Alkaloids	+
Phenolic compounds	+
Tannins	+

Present (+)/Absent (-)

B) Fourier Transform Infrared Spectrophotometer (FTIR) analysis:

The functional groups in the ethanol bark extract of Capparis sepiaria found to contain Carboxylic acids, Aromatic compound, Alkyl aryl ether, Alkene exhibiting 10 bands (Figure 1) in the frequency of 655 cm-1 to 3369 cm-1

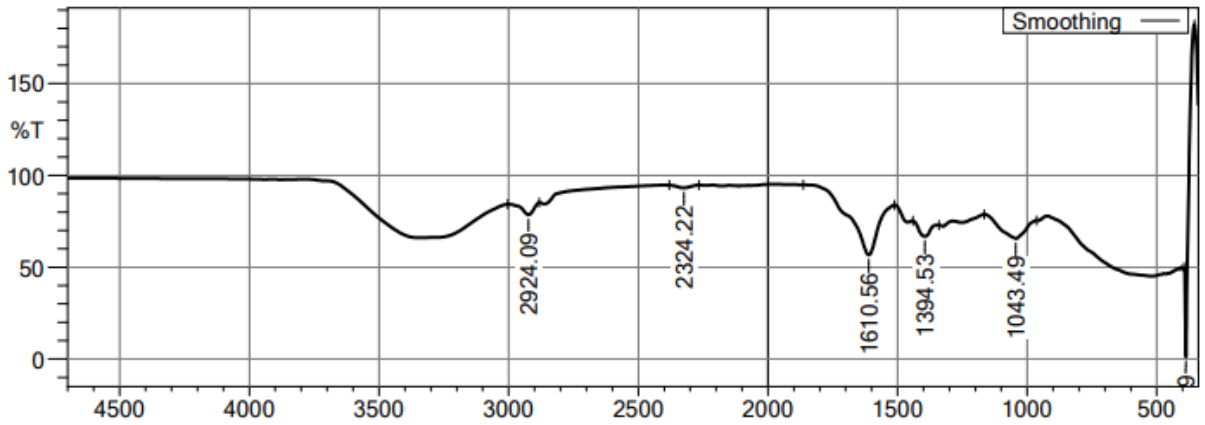

Fig 1: FTIR- analysis of Hydroalcolic fruit extract of *Capparis Decidua*.

C) 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 2.5 mg/kg and HECD 300 mg/kg showed significant inhibition against catalepsy by decreasing cataleptic score.

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

Time interval in mins	Mean ± SEM (Cataleptic score)					
	Vehicle control	Haloperidol control	Bromocriptine 2.5 mg/kg	HECD 100 mg/kg	HECD 200 mg/kg	HECD 300 mg/kg
0	0.00	0.00 ±0.00	o.92**	1.28	1.21	0.92
30	0.00	19.91	1.95***	4.07***	3.07***	2.44***
60	0.00	82.75	2.61**	4.95**	4.53***	3.36***
120	0.00	132.14	4.23***	6.38***	5.46***	4.8***
180	0.00	177.67	4.84***	12***	8.97***	6.57***
240	0.00	195.57	7.18***	17.94***	10.55***	8.25***

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.

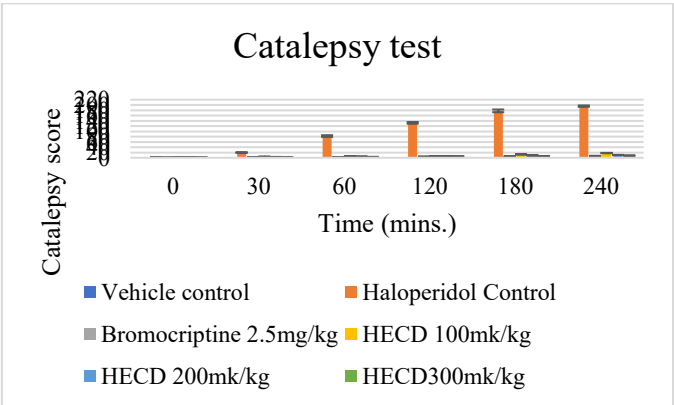

Fig. 02: Effect of bromocriptine and HECD on catalepsy in bar test

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

2.5mg/kg, HECD 200 and 300 mg/kg. [Table 03]

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

TREATMENT GROUPS	FALL OF TIME(SECS) MEAN±SEM
VEHICLE CONTROL	108.66 ± 1.40
HALOPERIDOL CONTROL	15.85 ± 0.04#
BROMOCRIPTINE 2.5mg/kg	105.09 ± 0.05***
HECD 100 mg/kg	76.65 ± 0.04***
HECD 200 mg/kg	90.10 ± 0.03***
HECD 300 mg/kg	103.09 ± 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.

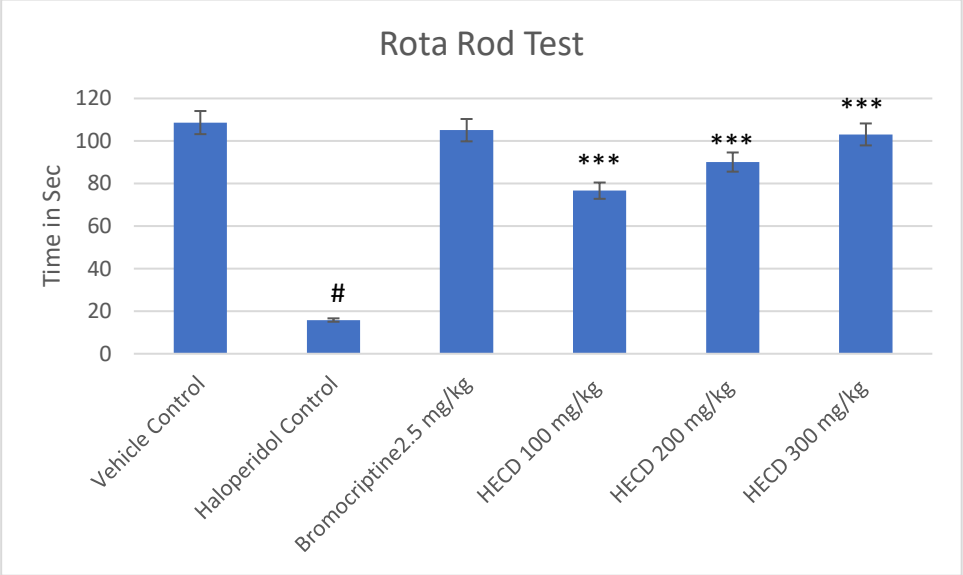

Fig. 03: Effect of bromocriptine and HECD on motor co-ordination test using rotarod

3: Test for locomotor activity:

Spontaneous motor activity was significantly decreased in haloperidol treated group as compared to the vehicle control group. Bromocriptine 2.5 mg/kg and HECD 300 mg/kg, significantly increased the locomotor activity as compared to haloperidol treated animals.

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

TREATMENT GROUPS	AMBULATIONS COUNTS/10 MIN MEAN±SEM
VEHICLE CONTROL	410.27 ± 0.10
HALOPERIDOL CONTROL	76.33 ± 0.91#
BROMOCRIPTINE 2.5 mg/kg	300.27 ± 0.06***
HECD 100 mg/kg	190.16 ± 0.90***
HECD 200 mg/kg	200.06 ± 0.05***
HECD 300 mg/kg	22531 ± 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.

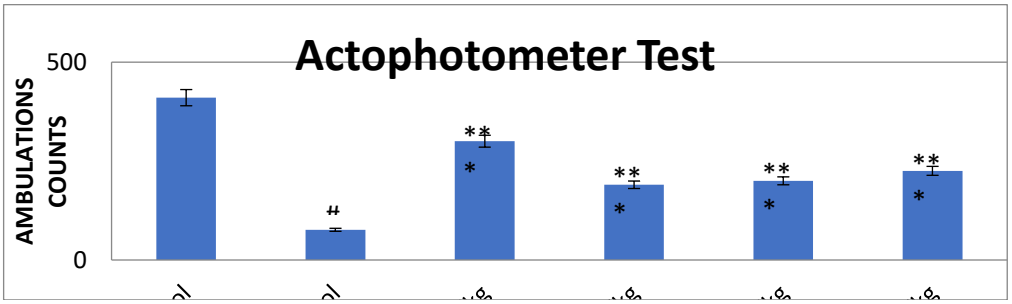

Fig. 04: Effect of bromocriptine and HECD on locomotor activity using actophotometer

D) BIOCHEMICAL PARAMETERS:

1: Determination 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 2.5 mg/kg and HECD 300 mg/kg showed significant increase in catalase level.

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

TREATMENT GROUPS	UNIT/mg MEAN±SEM
VEHICLE CONTROL	30.71±0.23**
HALOPERIDOL CONTROL	22.29±1.93
BROMOCRIPTINE 2.5 mg/kg	43.21±0.23**
HECD 100 mg/kg	30.89±0.55**
HECD 200 mg/kg	35.36±0.47**
HECD 300 mg/kg	41.43±0.19**

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.

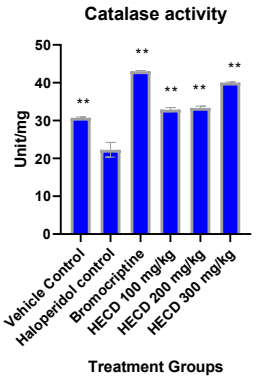

Fig. 05: Effect of bromocriptine and HECD on CATALASE level using UV.

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 2.5 mg/kg and HECD 300 mg/kg showed significant decrease MDA level.

Table 06: Effect of bromocriptine and HECD on MDA level

TREATMENT GROUPS	UNIT/mg MEAN±SEM

VEHICLE CONTROL	12.03 ± 0.4390
HALOPERIDOL CONTROL	28.55 ± 1.790#
BROMOCRIPTINE 2.5 mg/kg	11.13 ± 0.6315
HECD 100 mg/kg	23.55 ± 1.271
HECD 200 mg/kg	16.53 ± 2.143**
HECD 300 mg/kg	13.06 ± 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.

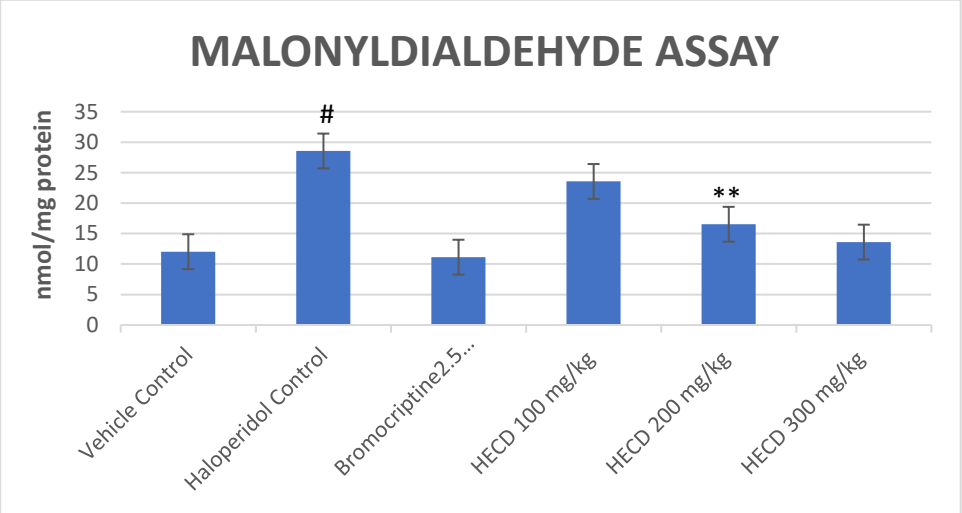

Fig. 06: Effect of bromocriptine and HECD on MDA level

3: Determination of GHS level:

In this test **Table 07**, haloperidol control group significantly decrease in GHS level as compared to the vehicle control group. Bromocriptine 2.5 mg/kg and HECD 300 mg/kg showed significant increase in GHS level.
Table 07: Effect of bromocriptine and HECD on GHS level.

TREATMENT GROUPS	**UNIT/mg MEAN±SEM**
VEHICLE CONTROL	3.1 ± 0.17
HALOPERIDOL CONTROL	2.02 ± 0.1
BROMOCRIPTINE 2.5 mg/kg	3.68 ± 0.13
HECD 100 mg/kg	2.97 ± 0.05
HECD 200 mg/kg	3.1 ± 0.09
HECD 300 mg/kg	3.13 ± 0.09

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.

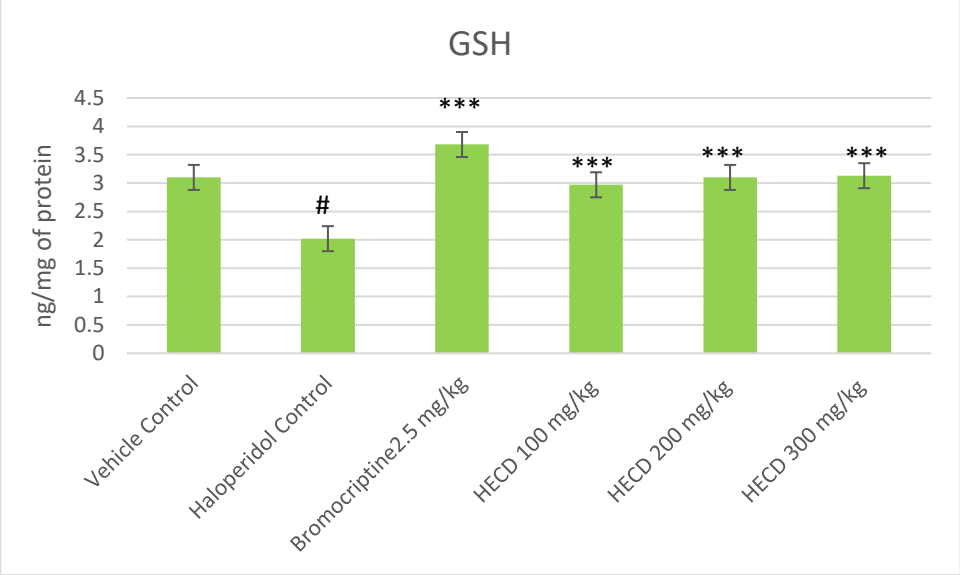

Fig. 07: Effect of bromocriptine and HECD on GHS level

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

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

Table 08. Absorbance of *Capparis Decidua (stem)* with standard ascorbic acid

CONCENTRATION [µg/ml]	ASCORBIC ACID (abs)	EXTRACT (abs)
5	0.038	0.134
10	0.017	0.14
20	0.015	0.128
40	0.01	0.092

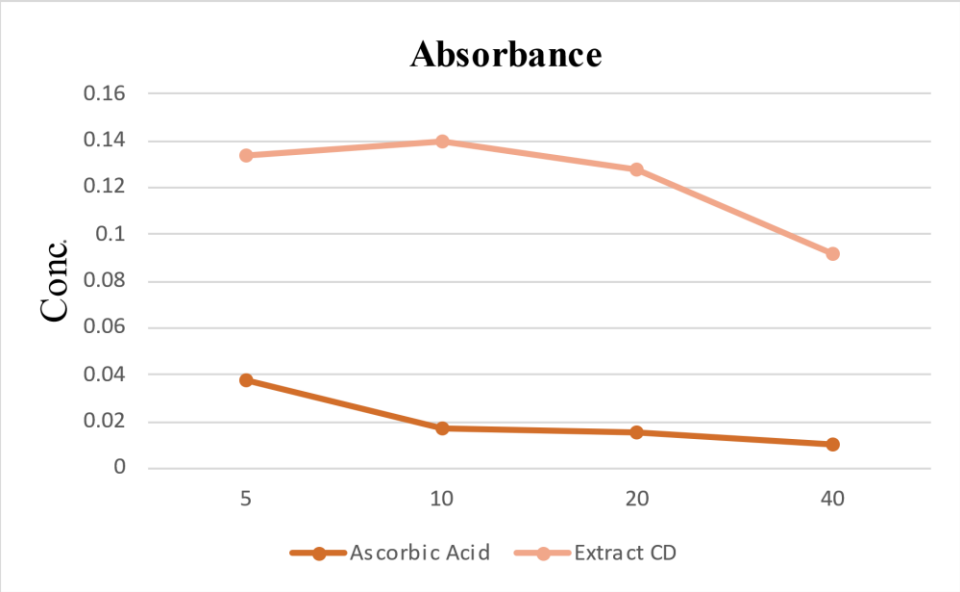

Fig 08: Absorbance at different concentration of extract and ascorbic acid.

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 Decidua (stem)* with ascorbic acid

CONCENTRATION [µg/ml]	EXTRACT (% Inhibition)	ASCORBIC ACID (% Inhibition)
5	93.33%	98.19%
10	93.62%	99.19%
20	93.91%	99.28%
40	95.62%	99.52%

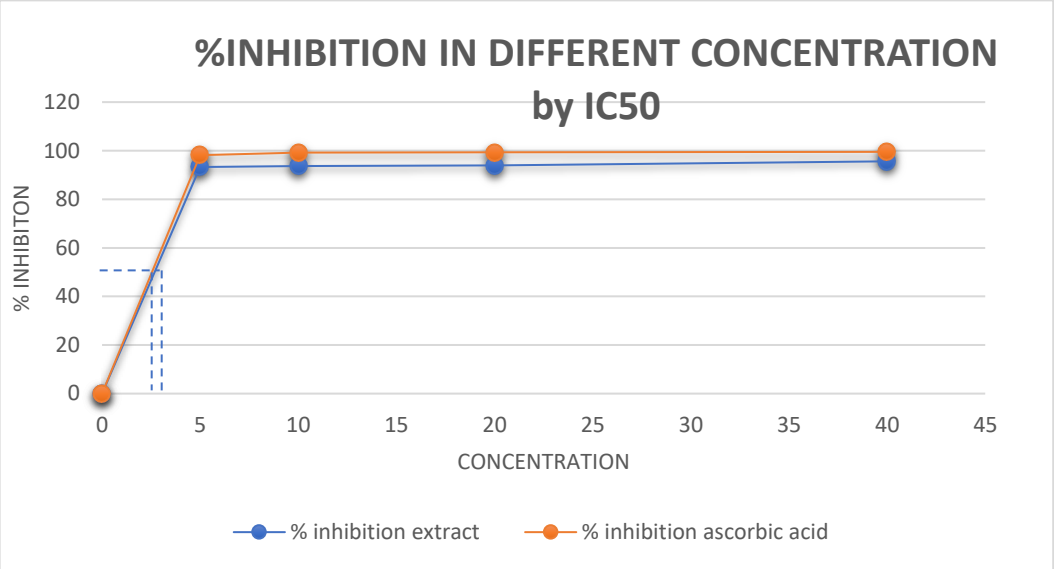

Fig 09: %Inhibition of Extract And Ascorbic Acid At Different Concentration.

DISCUSSION

Major symptoms of Parkinson's Disease includes Catalepsy (rigidity in movements), akinesia (slowing of movement), tremors and memory loss. Amongst this catalepsy is one of the major symptoms which make the life of PD patient uneasy. Bromocriptine is a well-known dopamine (D2) receptor agonist and is commonly used to improve the symptoms related to rigidity. Hence, this drug was used as standard in the present study to compare the efficiency of the models. haloperidol-induced catalepsy in rat resembles the deficiency of dopamine in nigrostriatal pathway that give rise to catalepsy. Catalepsy was induced in rat by intraperitoneal (i.p.) administration of haloperidol (1 mg/kg). This cataleptic behavior induced by haloperidol and the protective effect of standard (bromocriptine) and HECD 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 ($p<0.05$) 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 activity 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[19]. Dose dependent increase in locomotor activity was observed among the animals treated with hydroalcoholic extract of fruits of Capparis Decidua when compared to control group which provided more evidence of the ameliorative effect of Capparis Decidua on PD.

Mitochondrial complex-1 Dysfunction cause generation of oxidative stress and plays an imperative role in the pathogenesis of Parkinson's Disease. The endogenous antioxidants like GSH and CAT are beneficial components that fight with free radicals and neutralize them before they can attack 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 acts as a scavenger of H2O2 produced by cellular metabolism besides balancing the composition and disintegration of H2O2 in normal conditions. The decreased level of glutathione is the limiting factor in the elimination of H2O2. However,

in Parkinson's disease, glutathione is reduced extensively in the substantia nigra because of neuronal loss. Considering the antioxidant profile of Capparis Decidua fruits, it is assumed that the beneficial effect of Capparis Decidua 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 occur in a variety of pathological conditions including neurodegenerative disease. The MDA levels are indicative 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, which may be responsible for neuroprotection.

CONCLUSION:

Capparis Decidua fruits exhibited significant 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 huge scope lies in exploring the bioactive present in HECD 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 guided with designing the study, making of protocol, and managed the work done. Mr. Imtiyaz Ansari performed the literature searches, performed the biochemical test and DPPH, models, phytochemical screening, and completed the manuscript writing.

CONFLICTS OF INTEREST

We announce we do not have conflicting interests.

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