**Bauhinia purpurea**: A Seed with potential Anti-Parkinson’s Activity

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**ABSTRACT**

**Aim:** Parkinson’s disease (PD) is a neurodegenerative disorder characterized by progressive loss of the dopaminergic neurons in the substantia nigra pars compacta which innervates the dorsal striatum. The present study was carried out to evaluate Anti Parkinson’s Activity of ethanolic seed extract of *Bauhinia purpurea* [ESEBP] in Haloperidol induced experimental animal model.

**Method:** The effects of ethanolic seed extract of *Bauhinia purpurea* [200 mg.kg⁻¹ & 400mg.kg⁻¹] was studied using in-vivo parameter like Catalepsy and behavioural parameters by using Actophotometer, Hole board, elevated plus maze and Block methods.

**Results:** Rats treated with haloperidol (Control Group) showed a significant increase in immobility and muscular rigidity at 60 min, 90 min, 120 min, 150 min and 180 min, when compared to the normal group. Loco motor activity was significantly (P< 0.0001) and dose dependently increased at 30 mins, 60 mins, 90mins, 120mins, 150mins and 180mins of treatment with Ethanolic seed extract of *B. Purpurea* at given doses as compared to Haloperidol treated group.

**Conclusion:** In haloperidol induced parkinson’s the ESEBP had shown a significant protection against the parkinson’s condition in the early phase of the disease that was confirmed by observing the various parameters like catalepsy, muscle rigidity and locomotion.

**Keywords:** Parkinson’s disease, ethanolic seed extract, *Bauhinia purpurea*, Haloperidol, Catalepsy.

**INTRODUCTION**

Parkinson’s disease (PD) is a neurodegenerative disease that occurs due to progressive damage to the dopaminergic neurons in the nigrostriatal tract of the brain. Oxidative stress, low glutathione levels, DNA damage and iron deposition are the main causes for degradation of dopaminergic neurons in PD. Oxidative stress not only responsible for degradation of the dopaminergic neurons, but it also compromises mitochondrial oxidative phosphorylation, leading to decreased energy output and eventually to secondary cell death. Loss of dopaminergic neurons in nigrostriatetm tract results in the depletion of dopamine which is involved in coordinating movement [1].

The neuroleptic drug like haloperidol is one of the major causes for drug induced Parkinson’s worldwide. The incidence of drug induced Parkinson’s progresses with age [2]. It blocks dopamine D₂ receptors and produces a state of catalepsy in human or animals by reducing dopaminergic transmission in basal ganglion [3].The usages of synthetic drugs to treat Parkinson’s disease are like L-Dopa with Carbidopa, Entacapone, Amantidine, Selegiline, Bromocriptine, Benzotropine etc.
Which treat PD with different mechanism of actions either increase the dopamine levels in brain or prevent dopamine degradation or by stimulate the dopamine receptors.

The greatest disadvantage in presently available potent synthetic drugs lies in their adverse effects like constipation, ulcer, respiratory depression and hypertension, toxicity and reappearance of symptoms after discontinuation. Hence search for new pharmacological agents that retain therapeutic efficacy and yet devoid of adverse effects are justifiable.

Herbs have been important contributors to the quality of human life for thousands of years. Herbal medicine is the oldest and most widely used form of the medicine in the world today. Throughout history of plants have served mankind as valuable components of seasonings, teas, cosmetics, dyes and medicines. The term herb is used to refer not only to herbaceous plants but also to barks, leaves, roots, seeds, and flowers fruit of trees, shrubs and woody vines. Herbal medicines are an essential part of traditional medicine in almost any culture.

According to estimates, there are around 15,000 to 50,000 species of higher plants in the world and out of these only about 6% have been screened biologically while 15% have been evaluated chemically, thus giving an idea as to how much more work can still be done in herbal medicine research [4].

Some of the plants which have been reported to possess antiparkinson’s activity are Bacopa monnieri, Withania somnifera, Nardostachys Jatamansi, Ocimum sanctum. These herbs have been in use to treat Parkinson’s disease, due to its safe and free of side effects.

WHO currently encourages, recommends and promotes traditional as well as natural remedies in national health care programmes, as they are easily available at low cost, comparatively safe, and are culturally acceptable. The usage of herbs in Parkinson’s disease as they are safe and alternative medicine.

Bauhinia purpurea Linn. (caesalpiniaceae/fabaceae) is a medicinal plant traditionally used to treat various ailments, in order to establish pharmacological properties of seed of Bauhinia purpurea; studies were performed on antiparkinsonism activity. Traditionally this plant is used in the treatment of dropsy, pain, rheumatism, convulsions, delirium, Septicemia, etc. the bark of the plant is used as an astringent in the treatment of diarrhea. The extensive literature survey indicated the presence of phytoconstituents like triterpenoids, steroids, and saponins from the whole plant. The aerial plant is reported to contain flavones, glycosides, foliar flavonoids, 6- butyl-3-hydroxy flavanone, phenyl fatty ester, butane and β seto sterols. They are reported to exhibit various pharmacological activity such as CNS activity, cardiotonic activity, lipid-lowering activity, anti-oxidant activity hepatoprotective activity, hypoglycemic activity, etc. eleven new secondary metabolites together with two known flavanones and five benzyols, were isolated from the root extract of Bauhinia purpurea. The preliminary phytochemical studies have relieved that genus Bauhinia is mainly constituted of steroidal glycosides, terpenoids lactones and flavonoids [5].

More over Bauhinia purpurea Linn. Seeds contain about 2.2% of levodopa content [5] and hence may be useful in treating Parkinson’s disease. As no scientific evidences were reported in the literatures, the present study was undertaken to evaluate antiparkinson’s potency of ethanolic seed extract of Bauhinia purpurea.

MATERIALS AND METHODS

Collection of seed:
The seeds of Bauhinia purpurea Linn. was been collected during the month of March 2014 and dried under the shade.

Preparation of extract:
The seeds of Bauhinia purpurea Linn. was shade dried and the dried seeds were powderered to get coarse granules. The coarse powder was subjected to continuous hot extraction in Soxhlet apparatus using ethyl alcohol. The solvent was removed by distillation under reduced pressure, which produces a dark brown sticky residue.

Experimental Animals:
Albino Wistar rats (females), Weighing 220-250g were used. The selected animals were housed in acrylic cages in standard environmental conditions (20-25°C), fed with standard rodent diet and water. The experiments on animals were conducted in accordance with the internationally accepted principles for laboratory animal used and the experimental protocols duly approved by the institutional Ethical committee.

Acute Toxicity Studies:
Acute toxicity study of ESEBP was carried out in rats according to OECD-423 guidelines. Different doses of ESEBP were administered up to 2000 mg/kg b.w. (p.o.) and the rats were observed for a period of 72hr for behavioral changes, toxic symptoms and mortality.

Experimental setup:
Assessment of antiparkinson’s activity
Catalepsy induced by chronic haloperidol administration in experimental rats

Haloperidol (1.0mg.kg\(^{-1}\).p.o) was administered daily to the rats for a period of 20 days to induce catalepsy. Plant extracts and standard drug were administered orally 30 min before to the haloperidol treatment. The animals were divided into five groups each containing 6 animals.

GROUP 1: The animals received distilled water (5 ml/kg\(^{-1}\).p.o) and served as normal.

GROUP 2: The animal received haloperidol (1mg.kg\(^{-1}\).p.o) and served as control.

GROUP 3: The animal received haloperidol (1mg.kg\(^{-1}\).p.o) and treated with levodopa + carbidopa (100+25mg.kg\(^{-1}\).p.o) suspended in 1% CMC.

GROUP 4: The animal received haloperidol (1mg.kg\(^{-1}\).p.o) and treated with alcoholic extract of Bauhinia purpurea (200 mg.kg\(^{-1}\).p.o) which was suspended in 1% CMC.

GROUP 5: The animal received haloperidol (1mg.kg\(^{-1}\).p.o) and treated with alcoholic extract of Bauhinia purpurea (400mg.kg\(^{-1}\).p.o) which was suspended in 1% CMC.

BEHAVIOUR PARAMETERS:

Plus maze

- Weigh and number the animals. Divide them into two groups each consisting of 4-5 min. one group is used as control and other for (diazepam) treatment.
- Place the animals individually in the centre of maze, head facing towards open arm and start the stop watch and note following parameters for five minutes.
- 1st preference of mouse to open or enclosed arm.
- Number of entries in open and closed arm (an arm entry defined as the entry of four paws into the arm).
- Average time each animal spends in each arm (average time = total duration in the arm /no. of entries).
- Inject diazepam to the test group. After 30 min place the animals individually in the centre of the maze and note all parameters as described under step 2.
- Compare the preference of the animal to open or enclosed arm, average time spent in open arm and no. of entries in open arm in each group.

HOLE BOARD

This test was done using hole board. The hole board consisted of a 0.5m\(^{3}\) wooden board with 16 holes (3cm in diameter). The mice was placed at the corner of the board and allowed to move freely. First two minutes were allowed for adaptation and the number of head dripping in next four minutes was counted.

ACTOPHOTOMETER

- Weigh the animals and number them.
- Turn on the equipment (check and make sure that all the photo cells are working for accurate recording) and individually each mouse in the activity cage for 10 min. note the basal activity score of all the animals.
- Inject chlorpromazine (1 ml/100mg) and after 30 min re-test each mouse for activity scores for 10 min. note the difference in the activity before and after chlorpromazine.
- Calculate percent decreases in motor activity.

BLOCK METHOD:

The effect of test drug and standard drug on haloperidol induced catalepsy was studied by the following method.

STAGE 1: The rats were taken out of the home cage and placed on a table. Rats moves freely no score was given.

STAGE 2: If the rat failed to move when touched gently on the back or pushed, score of 0.5 assigned.

STAGE 3: The front paws of the rat were placed alternately on a 3cm high block. If the rat failed to correct the posture within 15 sec, a score of 0.5 sec for each paw was added to the score of step 1.

STAGE 4: The front paw of the rat was placed alternately on a 3cm high block. If the rat failed to correct the posture within 15 sec, a score of 1 for each paw was added to the score of step 1, step 2. Thus for an animal, the highest score was 35 (cut off score) and that reflects in total catalepsy.

STATISTICAL ANALYSIS

All the data was expressed as mean ± S.E.M. Statistical significance between more than two groups was tested using one way ANOVA followed by the Tukey test using computer based fitting program (Prism graph pad). Statistical significance was taken as P< 0.0001.

RESULTS

Acute toxicity study

The acute toxicity of Ethanolic seed extract of Bauhinia purpurea Linn. Was determined as per the OECD guideline no. 423 (acute toxic class method). It was observed that test extract was not Lethal to the rats even at the 2000mg.kg\(^{-1}\) doses. Hence, 1/10\(^{th}\) (200mg.kg\(^{-1}\)) and 1/5\(^{th}\) (400mg.kg\(^{-1}\)) of this dose were selected for further study.

Anti-parkinson’s activity
On catalepsy

Effect of ESEBP on catalepsy by block method

Catalepsy was characterized by immobility. Rats treated with haloperidol (Control Group) showed a significant increase in immobility and muscular rigidity at 60 min, 90 min, 120 min, 150 min and 180 min, when compared to the normal group. The group rats treated with standard drug L-Dopa & carbidopa (100mg+ 25 mg/kg) showed a significant decrease in immobility and muscular rigidity on 60 min, 90 min, 120 min, 150 min and 180 min, when compared to the control.

The group-IV (Test-1) and group-V (Test-2) were receiving ESEBP at two different doses (200 mg/kg & 400mg/kg, p.o.) showed a significant decrease in immobility and muscular rigidity when compared to control group. But interestingly the group-V (Test-2) shows good significant decrease in immobility and muscular rigidity at 120 min, 150 min and 180 min, than group-IV (Test-1). The results were shown in Table. 1 and Graph. 1

ON BEHAVIORAL PARAMETERS

The effect of the 1, 5-benzothiazepine derivatives on anti-anxiety activity was given in Table. 2. The treatment with Diazepam (4 mg/kg) and Test drug (5mg/kg) showed significant increase in the number of open arm entries 3.35, (P<0.01) and 3.0 (P<0.01) respectively and also spent more time in open arm, when compared with the control group (DMSO treated). Animal received only DMSO (control) had shown much decrease in the number of open arm entries 2.355(P<0.01) and also spend less time in open arm.

Animals treated with Haloperidol showed significant reduce in alertness as compared to control group. Alertness was significantly increased with Ethanolic seed extract of B. Purpurea at doses of 200 & 400 mg.kg⁻¹ and it was significantly dose dependent increase with given doses at 30 mins, 60 mins, 90mins, 120mins, 150mins and 180mins. The results were shown in Table. 3.

### Table. 1: Effect of ESEBP on catalepsy by block method

<table>
<thead>
<tr>
<th>S.N</th>
<th>Groups</th>
<th>Treatment</th>
<th>Immobility (Score) (Mean ± S.E.M) on 21st day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>1% CMC (p.o.)</td>
<td>0.0 ± 0 0.0 ± 0 0.0 ± 0 0.0 ± 0 0.0 ± 0 0.0 ± 0</td>
</tr>
<tr>
<td>2</td>
<td>Control</td>
<td>Haloperidol (1mg/kg, p.o.)</td>
<td>0.0 ± 0 3.33±0.16 3.41±0.08 3.33±0.08 3.5±0 3.41±0.08 3.41±0.08</td>
</tr>
<tr>
<td>3</td>
<td>Standard</td>
<td>L-Dopa &amp; carbidopa (100+25 mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>0.0 ± 0 1.33±0.10 1.33±0.10 1.33±0.10 1.25±0.1/7 1.25±0.11 1.25±0.11</td>
</tr>
<tr>
<td>4</td>
<td>Test 1</td>
<td>ESEBP (200mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>0.0 ± 0 2.08±0.15 2.08±0.15 2.08±0.15 2.25±0.1/1 2.16±0.10 2.16±0.10</td>
</tr>
<tr>
<td>5</td>
<td>Test 2</td>
<td>ESEBP (400mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>0.0 ± 0 0.83±0.10 0.83±0.10 0.83±0.10 0.83±0.1/0 0.83±0.10 0.83±0.10</td>
</tr>
</tbody>
</table>

ESEBP: Ethanolic seed extract of B. Purpurea.

n=6 animals in each group; All values were expressed as Mean±S.E.M
Neurodegenerative diseases are progressive neurological disorders highly linked to brain injuries from which there is no recovery. Selective neuronal loss in particular regions of our brain causes different types of neurodegenerative diseases such as Alzheimer’s disease (AD), Parkinson’s disease (PD), stroke, Amyotrophic lateral sclerosis (ALS), and many others [6].

Parkinson’s disease is a chronic progressive neurodegenerative disorder of the substantia nigra, a nucleus of basal ganglia. Parkinson’s disease occurs when nerve cells or neuron in an area of the brain known as the substantia nigra degenerate [7]. Dopamine is a chemical messenger responsible for transmitting signals between the substantia nigra and the next "relay station" of the brain, the corpus striatum to produce smooth, purposeful movement. The loss of dopaminergic neurons in the pars compacta region of the substantia nigra is the cause for the progressive movement disorder [8].

A number of the drugs in current clinical use for PD have shown efficacy in the haloperidol model, including L-DOPA, Bromocriptine, Pramipexole, Trihexyphenidyl and Amantadine [9-12]. Other drugs including Benztropine, Tolcapone, Selegiline and Rasagiline have also been shown to enhance the effects

**DISCUSSION**

Spontaneous motor activity was significantly ($P<0.0001$) decreased in Haloperidol treated group as compared to control group. Loco motor activity was significantly ($P<0.0001$) and dose dependently increased at 30 mins, 60 mins, 90mins, 120mins, 150mins and 180mins of treatment with Ethanol seed extract of *B. Purpurea* at given doses as compared to Haloperidol treated group. The results were shown in Table. 4.

### Table 2: Evaluation of Anti-Anxiety effect of ESEBP by elevated Plus maze method

<table>
<thead>
<tr>
<th>Sl.no</th>
<th>Group</th>
<th>Treatment</th>
<th>Number of entries</th>
<th>Time spent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Open arm</td>
<td>Closed arm</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>Normal</td>
<td>1% CMC (p.o.)</td>
<td>0.66±0.33</td>
<td>2.33±0.333</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>42.1±19.31</td>
<td>514.5±30.06</td>
</tr>
<tr>
<td>2.</td>
<td>Control</td>
<td>Haloperidol (1mg/kg, p.o.)</td>
<td>1.5±0.22</td>
<td>1.83±0.30</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>93.33±4.40</td>
<td>470.8±20.34</td>
</tr>
<tr>
<td>3.</td>
<td>Standard</td>
<td>L-Dopa &amp; carbidopa (100+25 mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>2.83±0.307</td>
<td>1.66±0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>287.5±27.1</td>
<td>300±28.86</td>
</tr>
<tr>
<td>4.</td>
<td>Test1</td>
<td>ESEBP (200mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>1.33±0.21</td>
<td>1.66±0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>262.5±15.47</td>
<td>350±20.41</td>
</tr>
<tr>
<td>5.</td>
<td>Test2</td>
<td>ESEBP (400mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td>2.83±0.30</td>
<td>1.33±0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>171.6±9.71</td>
<td>291.6±15.59</td>
</tr>
</tbody>
</table>

ESEBP: Ethanol seed extract of *B. Purpurea*.

n=6 animals in each group; All values were expressed as Mean±S.E.M
Table 3: Effect of ESEBP on Alertness (Hole board test) in Haloperidol induced Parkinson’s in rats.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Alertness (No. of Head dipping)</th>
<th>30min</th>
<th>60min</th>
<th>90min</th>
<th>120min</th>
<th>150min</th>
<th>180min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1% CMC (p.o.)</td>
<td></td>
<td>12.02±2.09</td>
<td>12.02±2.09</td>
<td>11.9±2.05</td>
<td>11.9±2.05</td>
<td>12.01±2.09</td>
<td>12.02±2.09</td>
</tr>
<tr>
<td>Control</td>
<td>Haloperidol (1mg/kg, p.o.)</td>
<td></td>
<td>2.83±0.166</td>
<td>2.83±0.16</td>
<td>2.75±0.11</td>
<td>2.88±0.15</td>
<td>2.84±0.12</td>
<td>2.88±0.15</td>
</tr>
<tr>
<td>Standard</td>
<td>L-Dopa &amp; carbidopa (100+25 mg/kg, p.o.)+ Haloperidol (1mg/kg, p.o.)</td>
<td></td>
<td>9±0.36</td>
<td>9±0.36</td>
<td>9±0.40</td>
<td>9±0.40</td>
<td>9±0.36</td>
<td>9±0.32</td>
</tr>
<tr>
<td>Test1</td>
<td>ESEBP (200mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td></td>
<td>7.16±0.30</td>
<td>7.16±0.32</td>
<td>7.16±0.32</td>
<td>7.14±0.33</td>
<td>7.13±0.33</td>
<td>7.16±0.32</td>
</tr>
<tr>
<td>Test2</td>
<td>ESEBP (400mg/kg, p.o.) + Haloperidol (1mg/kg, p.o.)</td>
<td></td>
<td>11.6±0.49</td>
<td>11.5±0.49</td>
<td>11.6±0.45</td>
<td>11.08±0.42</td>
<td>11.6±0.35</td>
<td>11.6±0.45</td>
</tr>
</tbody>
</table>

Table 4: Effect of ESEBP on Spontaneous motor activity by using Actophotometer in Haloperidol induced Parkinson’s in rats.

<table>
<thead>
<tr>
<th>Group</th>
<th>Spontaneous motor activity</th>
<th>30min</th>
<th>60min</th>
<th>90min</th>
<th>120min</th>
<th>150min</th>
<th>180min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>236.5±5.41</td>
<td>235.6±4.99</td>
<td>236±4.95</td>
<td>236±4.56</td>
<td>237.1±4.54</td>
<td>237.8±4.43</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>96.5±1.38###</td>
<td>96.3±1.42###</td>
<td>96.6±1.11###</td>
<td>96.8±1.35###</td>
<td>96.8±1.22###</td>
<td>97±1.26###</td>
</tr>
<tr>
<td>Standard</td>
<td></td>
<td>186±2.95***</td>
<td>184.8±2.94***</td>
<td>185.3±3.19***</td>
<td>185±2.87***</td>
<td>185±3.36***</td>
<td>184.8±3.10**</td>
</tr>
<tr>
<td>Test-1</td>
<td></td>
<td>183.3±2.15***</td>
<td>184.5±2.04***</td>
<td>184.8±2.2***</td>
<td>184.8±2.12***</td>
<td>185±2.23***</td>
<td>185±2.25***</td>
</tr>
<tr>
<td>Test-2</td>
<td></td>
<td>214.6±3.82***</td>
<td>215.5±3.64***</td>
<td>214.5±3.26***</td>
<td>214.5±3.54***</td>
<td>214.3±3.27***</td>
<td>214.1±3.37**</td>
</tr>
</tbody>
</table>

ESEBP: Ethanolic seed extract of B. Purpurea.  
n=6 animals in each group; All values were expressed as Mean±S.E.M
of L-DOPA [13-16]. But associated with serious side effects including extra pyramidal, ulceration and habit formation. In spite of tremendous strides in the modern medicines there is no satisfaction remedy for these disorders without any side-effects; hence there is a great need to explore the herbal drugs or their formulations in this context [17].

Furthermore there is a need to search for an ideal agent which treat the ailment with reduce undesirable side effects. Many natural products like Ocimum sanctum, Moringa oleifera [3], Nardostachys jatamansi [18] having neurological effect with more efficacies and less toxic have been discovered and used by man since prehistoric times for instance.

Like above one of the most commonly available plant Bauhinia purpurea Linn. belonging to family fabaceae, extensively used in traditional medicine throughout the world for the treatment of Various disease. The literature survey reveals that flavonoids, L-Dopa are responsible for anti-parkinson’s effects, which are main constituents present in the Bauhinia purpurea Linn. Seed.

In the folk medicine practice also the Bauhinia purpurea Linn. used for various neurological disorders and different parts of Bauhinia purpurea Linn. can be used in the treatment of dropsy, pain, rheumatism, convulsions, delirium, septicemia etc [19].

Hence, considering the above mentioned therapeutic values and constituents of Bauhinia purpurea Linn. and also the literature review reveals that anti-parkinson’s activity of Bauhinia purpurea Linn. seed has not been reported. In view of this, the present study is aimed to investigate the anti-parkinson’s activity of Ethanolic seed extract of Bauhinia purpurea Linn. against haloperidol induced Parkinson’s in Wistar rats.

Haloperidol works by antagonizing dopamine D2 and, to a lesser extent, D1 receptors in medium spiny neurons that comprise the indirect and direct pathways of the motor circuit respectively. The resultant block of striatal dopamine transmission results in abnormal downstream firing within the basal ganglia circuits that is manifest as symptoms of muscle rigidity and catalepsy within 60 min of haloperidol (0.5–5 mg/kg, i.p.) injection [20]. Although rigidity is a feature of PD, providing this model with some face validity, catalepsy, which is expressed as the inability of an animal to correct itself from an abnormally imposed posture, is not directly associated with PD [21].

Anti-parkinson’s activity of Bauhinia purpurea Linn. was evaluated by measuring following parameters like catalepsy (block method), akinesia, loco motion (by Actophotometer), Head drippings (by Hole board) and . Every result was compared with the standard drugs i.e. L-Dopa & carbidopa (100 + 25 mg/kg, p.o.) to demonstrate anti-parkinson’s activity.

*Bauhinia purpurea* Linn. at doses of 200 and 400 mg/kg significantly and dose dependently decreased in muscle rigidity and motor impairment. L- Dopa & carbidopa (100 + 25 mg/kg, p.o) significantly decrease muscle rigidity and motor impairment rats.

The catalepsy was measured by block method and metal bar test. In both methods EEBP at the dose of 200 mg/kg and 400 mg/kg b.w along with haloperidol at a dose 1 mg/kg b.w showed a significant and dose dependent decrease in immobility and muscle impairment when compared control group. Amazingly the *Bauhinia purpurea* Linn. at 400 mg/kg b.w was showed comparable results with standard L-Dopa& carbidopa. Similar kind of results was reported with *Nardostachys jatamansi* reported by Rasheed ahmed *et al.*, 2012 [18]. Dopamine depletion is considered as a cardinal feature in causing a parkinson’s disease in humans (or) in animal models. The enhancement of dopamine content by *Bauhinia purpurea* Linn. treatment may be one of the reasons to restores the alteration in immobility and muscle impairment.

The locomotion and exploration was measured by Actophotometer. In Parkinson’s condition the locomotion movements are decreased because the serotonin levels are decreased in hypothalamus due to decrease of dopamine levels in substantia nigra in brain [22]. The EEBP (200 mg/kg & 400 mg/kg) showed significant and dose dependent increase in locomotion as compare to control group. This may be due to increased levels of serotonin in hypothalamus in brain.

In nutshell in the present study, following a single injection of haloperidol (1 mg/kg, p.o.) induces catalepsy and rigidity in rats at 22nd day. *Bauhinia purpurea* Linn. of both doses (200 mg/kg, 400 mg/kg) showed remarkable improvement in muscle impairment, loco motor action in various experimental models. This may be due to increasing the dopamine and serotonin levels by *Bauhinia purpurea* Linn. At higher dose, the results were comparable with standard treatment.

Phytochemical investigations on *Bauhinia purpurea* Linn. have shown the presence of large concentration of flavonoids, phenols, saponins, glycosides, and tannins. More over literature survey reveals the presence of L-DOPA content about 2.2%. Thus, the anti-parkinson’s activity of *Bauhinia purpurea* Linn. could be due to its flavonoidal components and L-DOPA content. These components may exert its anti-parkinson’s by increasing the dopamine and serotonin levels in brain. The presence of these compounds in *Bauhinia purpurea* Linn. may involved in the
increasing the dopamine levels in brain thus it showed anti-parkinson’s properties of this fruit. Taken together, our results strongly support the anti-parkinson’s potential of the Bauhinia purpurea Linn. and its use in traditional medicine.

The present study indicates that the Ethanolic seed extract of Bauhinia purpurea Linn. may be used as an effective anti-parkinson’s agent. Further studies on isolation and structural determination of active principles might be worthy.

CONCLUSION

In haloperidol induced parkinson’s the ESEBP had shown a significant protection against the parkinson’s condition in the early phase of the disease that was confirmed by observing the various parameters like catalepsy, muscle rigidity and locomotion.

As per earlier reports reveal that, the phytoconstituents such as flavonoids, L-DOPA are responsible moieties in most of the plants for their anti-parkinson’s activities. Indeed, the Ethanolic seed extract of B. Purpurea also having these chemical constituents like flavonoids and L-DOPA. Hence these moieties in the B. Purpurea may be responsible for several effects like increasing dopamine, serotonin levels and antioxidant property. All this above effects may be responsible for the exhibition of anti-parkinson’s activity B. Purpurea. However, there is need to isolate and structural determination of active principles might be worthy.

Conflict of Interests

The authors declare that they have no conflict of interests regarding the publication of this paper.

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