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Neuroprotective activity of aerial parts of *Aeschynomene indica* L. against scopolamine-induced memory impairment in rats

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Alzheimer's disease is the most common cause of dementia and death. The initial symptom is a gradually falling ability to remember new information. *Aeschynomene indica* L. belongs to the family Leguminoseae and there is a lack of scientific reports regarding its aerial parts on memory improvement. Hence our study was to investigate the ameliorative role of the extract of aerial parts of *A. indica* against scopolamine-induced Alzheimer type disease in rats. The rats were divided into five groups, with six animals each. Elevated plus maze and light and dark apparatus were used to assess learning memory and acquisition memory, and an actophotometer was used to assess locomotor activity. In addition, biochemical parameters were also estimated. The hippocampal lesions were studied utilizing an electronic microscope at 40X magnification. In the scopolamine-treated group, escape latency time and locomotor activity were significantly elevated (P <0.001 and P <0.001), and there was a reduction in the time spent in the target quadrant, which was overturned in the case of the *A. indica* extract treatment (P <0.05 and P <0.05). Treatment with *A. indica* extract improved the changes in malondialdehyde (P <0.05), acetylcholinesterase (P <0.05), glutathione peroxidase (P <0.05), catalase (P <0.05) and superoxide dismutase (P <0.05) as compared to scopolamine treated group. Histopathological studies showed the pretreatment with *A. indica* L. extract groups minimized cerebral congestion, cerebral oedema, congestion of meninges and neuronal eosinophilia compared to the scopolamine-treated group. We conclude that the biochemical and histopathological results data support the Neuroprotective activity of aerial parts of *A. indica* against scopolamine-induced memory impairment in rats, which might be credited to its flavonoids and phenols content.

Keywords: *Aeschynomene indica* L., Memory, Neuroprotective, Rats

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Introduction

Neurodegenerative disorders promote nerve cell degradation, impair nervous system function, and can affect locomotion, language, perception, cognition, and memory depending on the regions affected and dementia is a major health issue worldwide and its prevalence is rising among the elderly[1,2]. Alzheimer's disease (AD) is an example of dementia, which is marked by an increase in amyloid beta (A) and phosphorylated Tau protein, as well as forebrain cholinergic neurons fall off, and reduced Acetylcholine levels, and permanent loss of cognitive function, culminating in memory degradation[3,4]. There is still no cure for the sickness, nor is it even curable. However, current therapy options are primarily symptomatic, halting disease progression. Around the world, individuals with dementia are roughly 47 million, among these 6.7 million are in the US and is the sixth driving reason for death in US. However, in India, it is more than 2 million[5,6]. The reported mechanisms are amyloid plaque deposition, expression of inflammatory mediators, oxidative stress, steroid hormone reduction, etc. Though many experimental studies have been carried out for its treatment, a hopeful intervention for curing the disease remains a task. Although the US FDA has approved many drugs for the treatment. The main treatments for the disease are acetylcholinesterase inhibitors and N-methyl-D-aspartate receptor antagonists[4]. Unfortunately, these drugs have been connected to several adverse effects like nausea, vomiting, anorexia, and insomnia, due to nonselective action on a variety of organ tissues both centrally and peripherally[7]. Prevention of AD with natural products has gained interest recently and Medicinal plants are a promising source of future drugs for AD. Considering

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the above statements and review of literature about the plant, the present study was focuses on the potential of *Aeschynomene indica* L. extract as a potent neuroprotective agent against memory impairment.

A. indica belongs to the family Leguminoseae/ Fabaceae. The main phytoconstituents are alkaloids, flavonoids, phenols, saponins, tannins, terpenoids, lignins, steroids, glycosides, indoles and anthocynadins[8]. The plant has been reported for various activities such as antimicrobial property[9], wound healing activity[10], anthelmintic activity[11], antibacterial activity[12], antifungal activity[13], anti-inflammatory[14] and antioxidant[15] activities. In earlier studies, it has been reported that antioxidants, flavonoids and phenols are considered as major neuroprotective agents[16,17].

Scopolamine is a muscarinic receptor antagonist, that inhibits cholinergic neurotransmission in rats, causing memory impairment. According to recent research, scopolamine increases the accumulation of reactive oxygen species, which causes oxidative stress and memory impairment[18]. The cholinergic hypothesis can be tested with an injection of scopolamine, which causes cognitive abnormalities similar to those seen in Alzheimer's disease, and treatment will seek to restore cholinergic system activity by blocking the acetylcholinesterase enzyme.

Materials and Methods

Plant material

The *A. indica* was collected in September 2020 from Anchatgeri village of Dharwad district in Karnataka, India. It was identified and authenticated by Dr. S. N. Emmi, H.O.D of Botany Dept. of H. S. Kotambari Science Institute Vidyanagara, Hubballi. (Ref no: KLECOPH/Plant/1920-21/184). The plant was thoroughly washed and rinsed with distilled water to remove soil and external material. The plant was shade-dried and exposed to size reduction to obtain a uniform powder of forty mesh size. The obtained powder has been subjected to organoleptic evaluation like odour, colour and taste.

Ethanolic extract preparation and phytochemical investigation

Extraction was carried out using ethanol by standard maceration process. Accurately weighed, 20 g of powder was macerated with 200 mL of ethanol for 3 days, followed by filtering through Whatmann No.1 filter paper. Transferred 25 mL of filtrate to the beaker and evaporated on the water bath, dried for 6 hours and cooled in a desiccator for 30 min. Contents of extractable matter were calculated in percentage[19].

Chemicals and drugs

Scopolamine and Donepezil were brought from Vivekanand Pharmacy, Hubballi. Other chemicals of analytical grade and enzyme assay kits were procured from Sigma Aldrich and ERBA Mannheim India, respectively.

Animals

Male Wistar rats (150-200 g, n=30) and female mice (18-25 g, n=6) were used after getting the Institutional Animal Ethical Committee (Ref. No. IAEC/01 /KLECOPH/2021) approval. They were housed in a group of six rats in an environmentally controlled room with a 12 hours light/dark cycle in polypropylene cages at 22±2°C and 40 to 60% relative humidity. They were fed with standard laboratory chow (Gold Mohur Lipton India Ltd.), and water *ad libitum* was provided.

Acute oral toxicity

Studies were carried out as per the revised draft OECD 423 guidelines. The albino mice were used to perform acute toxicity studies using the up-and-down method. Ethanolic extract of *A. indica* was administered at a dose of 2 g/kg body weight by oral route, and food was withheld for up to four hours after administration of the extract. The animals were observed for changes in general behaviour, weight, tremor, convulsion, salivation, sleep, skin, eye and death at 30 min, 1, 2, 3, 4, 24 h and once daily for the remaining 14 days[20].

Experimental design[21]

After the acclimatization period of one week, the rats were divided into five groups of six animals each, Group I received Normal saline 1 mL/kg *p.o*. Group II were treated with scopolamine (1 mg/kg) *i.p*, Group III (Std) received Scopolamine (1 mg/kg) *i.p* followed by Donepezil (5 mg/kg) *p.o.*, Group IV (LEAI) received scopolamine (1 mg/kg) *i.p.* followed by Low dose of ethanolic extract of *A. indica* (200 mg/kg/day) *p.o.* and Group V (HEAI) received scopolamine (1 mg/kg) *i.p.* followed by High dose of ethanolic extract of *A. indica* (400 mg/kg/day) *p.o*.

Procedure

Group I received normal saline for 17 days. Group II was received only scopolamine (1 mg/kg, *i.p.*). Group III received Donepezil (5 mg/kg, *p.o*.) prior 8 and 9 days

with scopolamine (1 mg/kg, *i.p.*) Group IV received a low dose of *A. indica* extract (200 mg/kg/day *p.o.*) prior 8 and 9 days with scopolamine (1 mg/kg, *i.p.*). Group V received a high dose of *A. indica* extract (400 mg/kg/day *p.o.*) prior to 8 days and 9 days with scopolamine (1 mg/kg, *i.p.*). All the animals were subjected to exteroceptive behavioral models of memory using Elevated plus maze (EPM), Locomotor (LMA), Light – Dark (LD) test before (6^{th}, $7^{th}$and 8^{th} Day), and after (15^{th}, 16^{th} and 17^{th}) induction of scopolamine. Rats were sacrificed by euthanasia after completing *in-vivo* studies, and brain tissue was isolated to measure the acetylcholinesterase, lipid Peroxidation, superoxide dismutase, catalase and glutathione levels.

In-vivo screening models for memory (Exteroceptive behavioural models)

Elevated Plus Maze (EPM)[22]

Elevated Plus Maze consists of 2 open arms crossed over 2 closed arms with the same dimensions of 50 × 10 cm. These four arms are associated with utilizing a central square of 10 × 10 cm. During the assessment of memory, rats were independently put toward one side of the open arm confronting far from the central stage and the transfer latency (TL) was recorded utilizing a stopwatch. The animals were permitted to explore the EPM for 5 min. If the rat did not enter the closed arms within 5 min it was directed on the back into one of them, and TL was given as 5 min. Later, the rat was permitted to explore the EPM for 30 sec to become familiar with the maze and then returned to its cage. A drop in TL time during treatment sessions was considered an index of memory improvement.

Light and dark apparatus (LD)[23]

The apparatus consists of two compartments (45×21×21cm) with $1/3^{rd}$ painted white and $2/3^{rd}$ painted black and are separated by a wall with a 3.5×3.5cm opening at floor level. The white compartment was lighted. Each rat was placed in the corner area of the white, away from the dark area and observed for over 5 min. The measured parameters were the number of transfers and time spent on the light side. After testing the rat, the apparatus was systematically washed with wet and dry cloths. An increase in the number of transitions time during treatment sessions was considered an index of memory improvement.

Actophotometer (LMA-Locomotor activity)[24]

Weighed the animals and placed each rat individually in the activity cage for 10 min. Noted the basal activity score of all the animals. The drug was injected, and after 30 min, each rat was retested for activity scores for 10 min. Noted the difference in the activity before and after drug administration. Decreased activity score was taken as an index of decrease in motor activity.

Biochemical estimation

Acetylcholinesterase (AChE) enzyme estimation in Cerebral cortex and Hippocampus[25]

Isolated the cerebral cortex and hippocampus from rat brain, centrifuged and homogenate. 0.4 mL of homogenate was added to 2.6 mL Phosphate buffer [0.1 M (pH 8)] and 100 µL of DTNB (10 mM) reagent and mixed well. To this, added 20 µL of AChI (0.075M) solution. The Absorbance was noted at 412 nm for 5 minutes.

The rate of moles of substrate hydrolyzed/min/g of tissue was calculated by

$R=5.74\times10\text{-}\ 4\ (\Delta A/Co)$

where R= Rate, in moles of substrate, hydrolyzed per min per gram of tissue, ΔA= change in Abs per min, and Co= Conc. of tissue (mg/mL).

Estimation of LPO in rat brain[26]

The 0.2 mL of homogenate + 0.2 mL of SLS (8.1% w/v) + 1.5 mL of 20% CH3COOH + 1.5 mL TBA (0.8%) were mixed. The mixed mixture was diluted to 4 mL using DM water and boiled (90°C) for 1 hour using a water bath. The mixture was cooled in running water, 1 mL of DM water + 5 mL mixture of pyridine:n-butanol (1:15 v/v) were added, centrifuged at 4000 rpm for 10 min and Absorbance of the organic layer was noted at 532 nm. The malondialdehyde/mg of protein was calculated and expressed in nanomoles.

Malondialdehyde = nmoles of MDA/mg of protein in the Concentration = $A \times (V/E) \times P$

where A= Abs at 535 nm, V= Volume of mixture, E= Extinction coefficient (1.56 × 105 m/cm), P= mg of protein per g of tissue. All values were indicated in nM of MDA/mg of protein

Estimation of reduced GSH[27]

To 0.25 mL of brain homogenate, added 2.5 mL of sodium phosphate buffer and 50 µL DTNB [pH 7.0]. The solution was mixed well and incubated at 25-27°C. Absorbance was read at 412 nm within 15 min. GSH level was expressed in µmoles/mg of tissue.

$C0 = A/€ \times D$

where, C0 - original concentration, A - Absorbance at 412 nm, € - molar extinction coefficient i.e. 13,600/M/cm, D - dilution factor

Estimation of Superoxide dismutase (SOD)[28]

The process of estimation was determined by Kakkar *et al.* method. 0.1 mL of Sample was mixed with 1.2 mL sodium pyrophosphate, 0.1 mL phenazine methosulphate and 0.3 mL nitro blue tetrazolium. The reaction was started by adding NADH (0.2 mL) and was incubated for 90 secs at 30°C. The reaction was stopped by adding 0.1 mL glacial acetic acid and stirred briskly with 4 mL n-butanol. It was permitted to stand for ten minutes, centrifuged and separated the layer of butanol. The intensity of chromogen colour was measured (against butanol) at 560 nm by spectrophotometer. A system devoid of enzyme activity was defined as enzyme concentration required to decrease the rate of reaction by 50% in 1 minute under the assay conditions.

Histopathological study[29]

After sacrificing, the rat brain was isolated and washed with saline. Brain tissue was cut into pieces and preserved in a ten per cent neutral formalin solution for 2 days. After two days, the pieces were washed using water for 12 hours and dehydrated with alcohol. The tissue pieces were cleaned using xylene for 15-20 minutes, then subjected to paraffin infiltration in an automatic tissue processing unit.

Melted hard paraffin was poured into a square-shaped block, and the brain pieces were dropped quickly and waited to cool. Blocks were cut to make 5 µ thickness sections using a microtome, followed by applying a sticky substance to these sections placed over the microscopic slide and dried completely before staining. We used eosin and hematoxylin to stain these sections and finally observed them in the microscope.

Statistical Analysis

The experimental data were statistically analysed using a one-way analysis of variance (ANOVA) followed by Tukey's Multiple Comparison Test using Graph Pad Prism 5.0 software. Data were expressed as Mean±S.E.M. Differences were considered significant at $P < 0.05$.

Results

Percentage yield of *A. indica* L.

The percentage yield (w/w) of *A. indica* L. Ethanolic extract by maceration method was 12%.

Phytochemical constituents in ethanolic extract of *A. indica* L.

The phytochemical investigation showed the presence of carbohydrates, glycosides, flavonoids, tannins and alkaloids.

Acute oral toxicity

By acute oral toxicity studies, we found that the lethal dose of ethanolic extract of *A. indica* L. was more than 2000 mg/kg body weight. So, $1/5^{th}$ and $1/10^{th}$ of the 2000 mg/kg body weight was chosen for further studies.

Behavioural tests

Elevated plus maze (EPM)

All groups were exposed to EPM for Transfer Latency (TL) to assess the retrieval of memory in the behavioural model. The TL was significantly decreased on the 8^{th} day compared to the 7^{th} for all groups (before scopolamine administration). In contrast, the TL was significantly increased on the 17^{th} day compared to day 16^{th} for the Scopolamine-treated group ($P < 0.001$), but TL was significantly reduced on 17^{th} day compared to day 16^{th} in the pretreated with ethanolic extract of *A. indica* groups ($P < 0.05$ and $P < 0.001$) respectively (Fig. 1a-b).

Light and dark test (LD)

Time spent in dark partition: The time spent in the dark partition was significantly increased in the Scopolamine group compared to the control group ($P < 0.001$). The Donepezil 5 mg/kg, *A. indica* L. 200 and 400 mg/kg showed significant decreases in the time spent in dark partition compared to the Scopolamine group ($P < 0.001$, $P < 0.05$, and $P < 0.001$), respectively (Fig. 2).

Time spent in Light partition: The time spent in light partition was significantly decreased in the Scopolamine group compared to the control group. The Donepezil 5 mg/kg, *A. indica* L. 200 and 400 mg/kg showed a significant increase in the time spent in light partition compared to the Scopolamine group.

Number of Transitions: The number of transitions was significantly decreased in the Scopolamine-treated group compared to the control group. The Donepezil 5 mg/kg, *A. indica* L. 200 and 400 mg/kg showed a significant increase in the number of transitions compared to the Scopolamine group.

Locomotor activity (LMA)

The rats were exposed to photocells in an Actophotometer to assess the motor activity in the

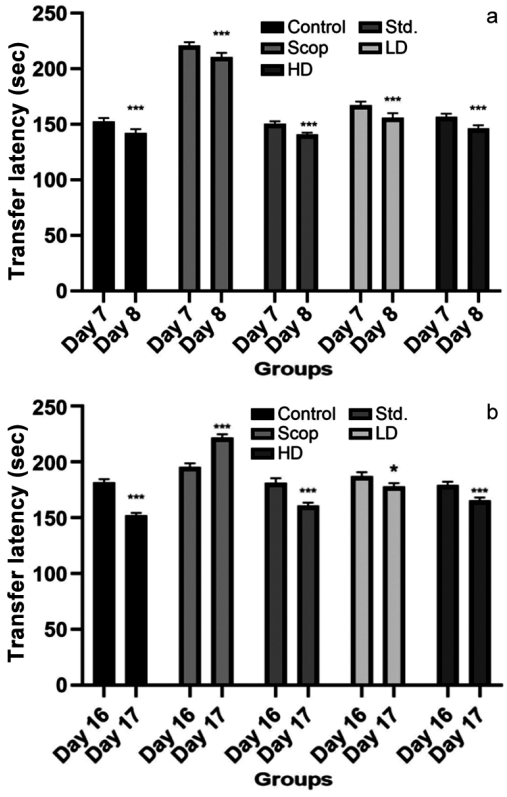

Fig. 1 — Effect of ethanolic extract of *A. indica* L. on behavioural model EPM. Mean±SEM; (N=6): analysed by one-way ANOVA "Tukey's Multiple Comparison Test", whereas $^{***}P$ <0.001 compared to control, $^{**}P$ <0.01, $^{*}P$ <0.05 compared to day a) 7; and b) 16.

behavioural model. The motor activity was significantly decreased on the 16th day, i.e., the Scopolamine group, compared to the control group. The Donepezil 5 mg/kg *A. indica* L. 200 and 400 mg/kg showed a significant increase in motor activity on day 16th compared to the Scopolamine group.

Biochemical parameters

Scopolamine treated rats showed decrease in SOD (P <0.001), CAT (P <0.001) and GSH (P <0.001), and increase in AChE (P <0.01) and LPO levels (P <0.001) in brain tissue as compared with control group, but pretreatment groups i.e. high dose of extract of *A. indica* has increased the levels of SOD (P <0.01) and CAT (P <0.05) and GSH (P <0.001) and decreased levels of AChE (P <0.001) and LPO (P <0.001) as compared to scopolamine group (Table 1).

Histopathological investigation

The histopathology of the control group (Group I) brain section indicated no cerebral oedema, congestion,

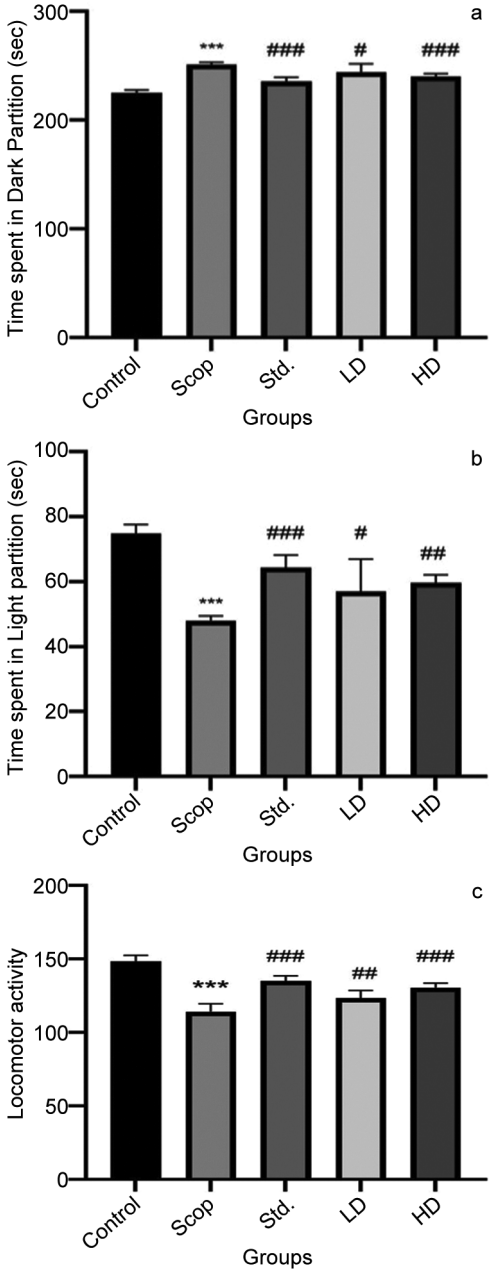

Fig. 2 — Effect of *A. indica* L. extract on the behavioural model. a) Light and dark test (Time spent in Dark partition); b) Light and dark test (Time spent in Light partition); and c) Locomotor activity. Mean±SEM; (N=6): analysed by one-way ANOVA "Tukey's Multiple Comparison Test", whereas $^{***}P$ <0.001 compared to control, $^{###}P$ <0.001, $^{##}P$ <0.01, $^{#}P$ <0.05 compared Scopolamine.

neuronal eosinophilia, and there was no change in the anatomy of visualized in neuronal micro vacuolization, neuronal nuclear pyknosis, neuronal karyorrhexis, neutrophilic infiltration, RBC extravasation, macrophage influx, vascular proliferation and reactive gliosis. However, the scopolamine-treated group (Group II)

Table 1 — Effect of ethanolic extract of *A. indica* L. on biochemical parameters

Treatment Groups	AChE (in moles x 10^{-6}/min/g of tissue)	LPO (in nmoles of MDA/mg protein)	GSH (in µmoles/mg of protein)	SOD (Units/mg protein)	Catalase (mmole/mg protein)
Control	5.35±0.20	11.51±0.19	2.46±0.13	10.12±0.13	3.20±0.25
Scopolamine	14.05±0.05***	39.28±0.23***	0.93±0.01***	8.18±0.16***	1.49±0.17***
Std (Donepezil+Scop)	6.42±0.17###	14.27±0.24###	2.35±0.05###	9.22±0.15###	2.42±0.15##
LD (Low dose Extract+Scop)	9.51±0.19###	25.29±0.31###	1.24±0.02#	8.33±0.12	1.80±0.12
HD (High dose extract+Scop)	7.52±0.23###	17.125±0.23###	1.44±0.02###	8.96±0.02##	2.24±0.13#

Values are Mean±SEM whereas ***P <0.001 compared to control, ### P <0.001, ##P <0.01, #P <0.05 compared to scopolamine group.

Control group - received Normal saline 1 mL/kg *p.o*., Scopolamine group - treated with scopolamine (1 mg/kg) *i.p*., Std group - received scopolamine (1 mg/kg) *i.p*. + Donepezil (5 mg/kg) *p.o*., LD group - received scopolamine (1 mg/kg) *i.p*. + Low dose of ethanolic extract of *A. indica* (200 mg/kg/day) *p.o.,* and HD group - received scopolamine (1 mg/kg) *i.p*. + High dose of ethanolic extract of *A. indica* (400 mg/kg/day) *p.o*.

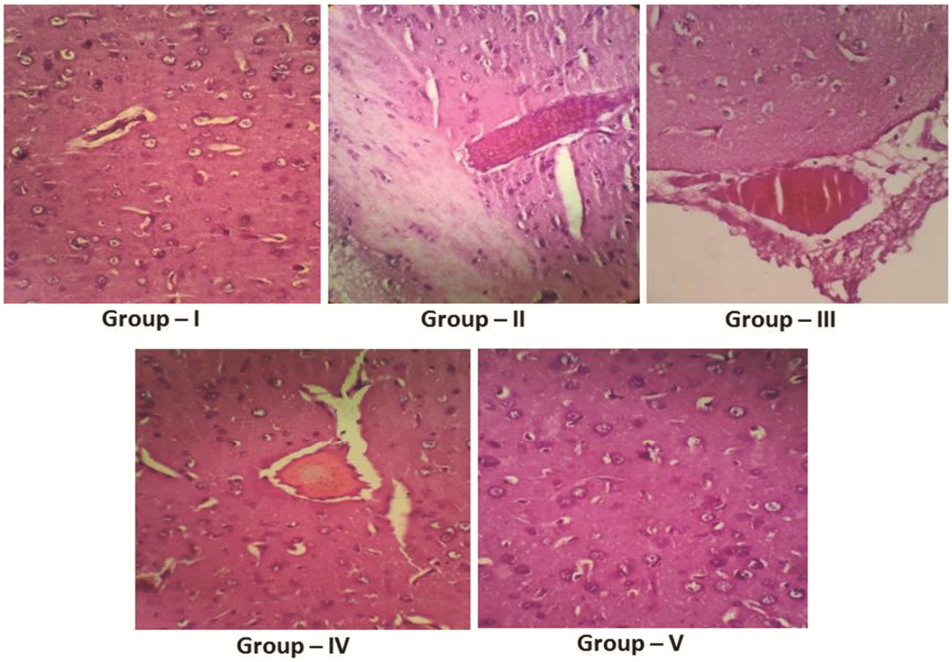

Fig. 3 — Effect of *A. indica* L. extract on histopathology of rat brain. Group I is control rats not having any congestion, oedema and infiltration, whereas Group II is Scopolamine treated showing congestion, oedema and infiltration, but in Groups III, IV and V, these changes were minimised.

indicated sensible changes in cerebral congestion, cerebral oedema, moderate meningeal congestion, neuronal eosinophilia and mild neuronal micro vacuolization, neuronal nuclear pyknosis, neuronal karyorrhexis, neutrophilic infiltration, RBC extravasation, macrophage influx, vascular proliferation, and reactive gliosis. The standard and treatment groups (Group III, IV, and V) demonstrated moderate to no cerebral congestion, cerebral oedema, meningeal congestion and neuronal eosinophilia, and there was no change observed in neuronal micro vacuolization, neuronal nuclear pyknosis, neuronal karyorrhexis, neutrophilic infiltration, RBC extravasation, macrophage influx, vascular proliferation, and reactive gliosis (Fig. 3).

Discussion

It is an advanced neurodegenerative disorder of old persons associated with loss of cognitive dysfunctions and memory[30]. Many proofs have proposed that the

cognitive symptoms of Alzheimer's disease are a result of the impairment in the brain hippocampus neurogenesis[31]. It has become a problem due to the increase of the ageing population and increases in the lifetime expectancy[32].

In the present study, we estimated the effect of the extract of *A. indica* against the scopolamine model. Scopolamine is an alkaloid that impairs memory (short-term and long-term) in animals as well as humans. It can cause oxidative stress by interfering with the neurotransmitter acetylcholine in the brain, resulting in cognitive impairment[33]. Therefore, scopolamine-induced memory impairment is an effective screening model for the valuation of anti-amnesic effects.

In this study, prior treatment with an extract of *A. indica* significantly reduced the percentage of spontaneous alternation induced by scopolamine, indicating a significant improvement in memory[34].

The EPM test was commonly used to evaluate learning and memory. It depends on the apparent natural aversion of rats to open and high spaces, and earlier, it was used for anxiety studies[22]. The time taken to move an animal from the open arm to the closed arm (Retention transfer latency) is commonly noted for assessing memory in the EPM test. If the animal has an acquisition trial, there is shortened transfer latency in the retention trial. In this test, an extract of *A. indica* significantly reduced the transfer latency, indicating that it has a nootropic effect because it improves the retention of information in the absence of any memory impairment inducer. Additionally, *A. indica* reduced the retention transfer latency significantly after scopolamine injection, indicating that *A. indica* improves learning and retention of information and plays a role in memory formation.

The Light and Dark test relies on both the innate aversion of animals to intensely illuminated areas and their tendency to exhibit spontaneous movement behaviour in response to new environments[35].

The locomotor activity was reduced in patients suffering from Alzheimer's disease because of dementia, and Alzheimer's disease is associated with circadian activity rhythm disturbances[36]. Continuous monitoring of locomotor activity over several days exposes abnormalities of the amplitude. The actophotometer is the equipment generally used to observe the locomotor behaviour of animals. In the present study, scopolamine-induced animals reduced locomotion activity because of dementia and abnormalities of the amplitude.

The biochemical assays showed that administration of scopolamine increased the activity of acetylcholinesterase and the MDA level (a measure of brain lipid peroxidation) and reduced the GSH level of (the main antioxidant enzyme of the body) as compared to the control group. Our results agree with literature that showed that administration of scopolamine in animals can lead to increased acetylcholinesterase activity and oxidative status in the brain[37]. Prior treatments with extract of *A. indica* overturned the increase of the activity of acetylcholinesterase and oxidative stress induced by scopolamine, thus guarding animals against learning and memory loss.

Pretreatment with an extract of *A. indica* significantly prevented the cell decrease in the hippocampus dentate gyrus. The dentate gyrus is part of the brain where adult neurogenesis takes place, and it is also implicated in hippocampal neurogenesis and plasticity[38]. In scopolamine-treated animals, memory impairment occurs as a result of an increase in acetylcholinesterase activity and brain oxidative status and impairs neurogenesis in the brain, which in turn leads to cognitive deficits as in Alzheimer's Disease[39]. By preventing the cell decease in the dentate gyrus induced by scopolamine, an extract of *A. indica* could be a good treatment for cognitive deficits and Alzheimer's disease. There is growing evidence in the literature that scopolamine impacts the acquisition, consolidation and recall of information[40].

Conclusion

The present study was conducted to evaluate the protective role of ethanolic extract of *A. indica* in scopolamine induced memory loss in rats, which is anacetylcholine (muscarinic) receptor antagonist. The behavioural parameters and memory and learning insufficiencies were evaluated by using elevated plus maze, light and dark test and actophotometer apparatus. The biochemical parameters were estimated by reagents and also histopathological studies of rat brain was done using microscope. The high dose of extract of *A. indica* improved the learning of information and spatial short-term as well as long-term memory and recognition memory by improving the transfer latency (P <0.05 and P <0.001) in EPM as well as increased time spent in dark partition (P <0.001) in LD. The biochemical and histopathological data from the present study clearly support the neuroprotective property of the *A. indica* and this might be related to the decrease in the level

of acetylcholinesterase associated with antioxidants as well as neurogenesis improvement. Our future study will be fractionation and isolation of the plant phytoconstituents responsible for its activity.

Conflict of interest

The authors declare that there is no conflict of interest.

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