RESEARCH ARTICLE

Evaluation of Anti-amnesic Activity of Ethanolic Extract of *Averrhoa carambola* Leaves

Dinesh Kumar[1*], Asheesh K Gupta[1], Navneet Verma[2], Sushil Kumar[1]

[1]*School of Pharmaceutical Sciences, IFTM University, Moradabad, Uttar Pradesh, India.*
[2]*Pharmacy Academy, IFTM University, Moradabad, Uttar Pradesh, India.*

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ABSTRACT

The current research aims to screen the anti-amnesic and neuroprotective efficacy of ethanolic extract of *Averrhoa carambola* (EEAC) leaves. To fulfill the above purpose, phytochemical screening, quantity of total phenolic, flavonoids, behavioral study, biochemical study and histopathological study were performed. The results of phytochemical screening, quantities of total phenolic flavonoids, behavioral study, biochemical study and histopathological studies revealed the anti-amnesic and neuroprotective activity of the EEAC. EEAC shows anti-amnesic and neuroprotective activity due to numerous phytoconstituents and high amounts of phenolic and flavonoids. The results of this study show that the EEAC may be useful for managing amnesia.

Keywords: Anti-amnesic, Neuroprotective, *Averrhoa carambola*, Alzheimer's disease, Donepezil, Scopolamine.

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INTRODUCTION

Amnesia is a condition expressed as an impairment of learning and memory.[1] Memory problems have been related to a variety of oxidative stress, emotions, dementia, anxiety, stress, amnesia, Alzheimer's disease (AD) etc.[2] The World Health Organization (WHO) estimates that around 55 million individuals are affected by a neurological condition.[3]

AD is a complex illness in which various components, such as Aβ protein accumulation, cholinergic neurotransmission change, the imbalance between oxidative stress and antioxidants, and neuro-inflammation, are to be blamed.[4-6]

Scopolamine is an acetylcholine inhibitor that impairs learning and memory. Additionally, it induces oxidation in the brain. This drug is used to induce memory impairment in animal models. Tacrine, rivastigmine, aniracetam, and piracetam are synthetic medications used to treat AD. Allopathic medications are costly as well as produce more adverse effects. Scientists are looking for ayurvedic or homeopathic therapies because ayurvedic and homeopathic therapies are cheaper and produce fewer side effects.[7]

As per the literature survey, plants rich in phenolic and flavonoid phytoconstituents are responsible for increasing learning, memory and cognition.[8]

The plant chosen for this study is *Averrhoa carambola*, which belongs to the family oxalidaceae and can be found in practically every environment on the planet. Its antibacterial, hepatoprotective, antidiarrheal, antioxidant and other properties make it useful in various illnesses. As per the literature survey *A. carambola* leaves rich in phenolic and flavonoids. However, no scientific evidence exists to support its neuroprotective and anti-amnesic efficacy against scopolamine induced memory impairment in rats.

In this research, to confirm its neuroprotective activity we performed phytochemical screening, measured total phenolic and flavonoid content, as well as performed behavioral study, biochemical study and histopathological study.

MATERIALS AND METHODS

Material Purchased

Donepezil and scopolamine were procured from Signa-Aldrich USA. Gallic acid and quercetin were acquired from Mumbai's yucca firm. Reagents and chemicals used in this study were obtained from the Central Store at IFTM University Moradabad, UP, India.

Collection and Authentication

Plant specimen was obtained from the garden of Amroha District, Uttar Pradesh, India. Authentication was done from the NIScPR, Delhi. The scientist confirmed the specimen as leaves of *A. carambola* and provided authentication number 3914-15-1. Specimen was deposited in NIScPR.

**Author for Correspondence: dineshpharma181@gmail.com*

Leaves Extract Preparation

Leaves were gathered, cleaned with water and shade dried. Completely dried leaves were converted into coarse powder with the help of grinder. About 100 gm coarse powder was filled in 1000 mL soxhlet and extracted with petroleum ether to defatt the sample. After this same sample was again extracted with absolute alcohol. After extraction, excess solvent was removed from the extract by steam distillation method, concentrated extract was further dried on a water bath at a temp 40°C and the extractive value was determined.[9,10]

Phytochemical Evaluation

Analysis for alkaloids, flavonoids, saponins, carbohydrates, and tannins were performed on the EEAC.[11]

Phenolic and flavonoid content measurement

Previously reported techniques were employed to estimate the phenolic and flavonoid content of EEAC leaves. To determine the total phenolic quantity, one mL of one mg/mL extract was combined with one ml of FC reagent, and subsequently, 10 mL of 7% Na_2CO_3 liquid was added. About 13 mL of condensed water was thoroughly mixed after a few seconds. After storing the aforesaid solution in the dark for 90 minutes at 23 to 25°C, the absorbance at 760 nm was measured. The total phenolic amount of EEAC was calculated using a calibration curve produced with gallic acid. The whole phenolic amount in EEAC is expressed as gallic acid mg/g of dry extract. To determine total flavonoid quantity, the aluminum chloride technique was used. Quercetin solution was also evaluated in this manner. The whole flavonoid content in EEAC is expressed as quercetin mg/g of dry extract.[12-14]

Acute oral toxicity

Acute oral toxicity (AOT) study of EEAC at a dose of 2000 mg/kg was completed in accordance with OECD 423 guidelines. This research included Swiss albino female rats weighing around 180 and 200 g. Rats were starved for three to four hours before receiving EEAC, but water was continuously provided, and food was withheld for 1 to 2 hours following treatment. EEAC-treated rats was examined first day at 30, 60, 120, 240 minutes and then after were examined once a day for 14 days regarding behavioral, morbidity and mortility.[15]

Experimental Animal

The current study was employed Swiss albino rats of both sexes weighing around 150 to 200 g. The research was conducted using the accepted methodology specified by CPCSEA. CPCSEA and IAEC of IFTM University, Moradabad Approval No. 2021/837ac/Ph.D./02 duly approved the research project.

Grouping of animal and schedule of treatment

Group 1: Normal control group 2: Scopolamine (Scop) 1-mg/kg/day; Group 3: Scop + EEAC 100 mg/kg/day; Group 4: Scop (1-mg/kg/day) + EEAC 200 mg/kg/day; Group 5: Scop (1-mg/kg/day) + EEAC 400 mg/kg/day; Group 6: Scop (1-mg/kg/day) + Donepezil (5 mg/kg/day). Treatment was given to rats for 21 days. Each group was consisting minimum 6 rats.

Behavioral research

Before starting the behavioral research, animals were trained and only fully trained animals were selected for investigation. The research was performed in a soundproof room.

Nonel Object Recognition Test

Apparatus was a white-colored open box of plywood. The box was 170 cm long, 50 cm wide and 40 cm high. Experiments were carried out in three phases: habituation, familiarization, and testing. To habituate and diminish the rats' dread of a new environment, they were exposed to the device without items for 5 minutes, with the freedom to explore the equipment. The rats were placed in the same setting the next day, but with two identical items A1 and A2 (plastic balls). The familiarization exploring time was 5 minutes. After 24 hours the rats were introduced for testing phase. During this phase, rats were returned to the apparatus containing a novel item (Plastic Square) utilized to modify A2. When the rat smelled or made contact with the things with their nose, this was termed exploration. The contact time plastic square and the plastic ball were noted. The device was washed with disinfectant at the end of each session to hide any smell residues. The discrimination index/memory index (DI) was calculated by using below formula 1.[16]

$$Memory\ Index\ (\%) = \left[\frac{T(novel)}{T(novel) - T(familiar)}\right] \times 100$$

Formula 1: Percentage memory index

Y Maze

This apparatus was employed to estimate the experimental rats' percentage alteration (Memory Index). Y maze used in this experiment contains three similar arms, P, Q and R. The arm was 40 cm in length, 35 cm in height and 12 cm in width, positioned at 120° angles. For the test, every animal was sited on maze triangle center and permitted to freely explore it for 8 minutes. The arm entry was pronounced complete if all body parts were completely in the arm. A series of arm entries was visually recorded. The maze floor was washed with 70% ethanol between trials to avoid odors. The overall number of visits was utilized to calculate the degree of percentage alternation (memory index) using the following formula 2.[17]

$$Percentage\ alteration = \left[\frac{(Number\ of\ alteration)}{(Total\ Number\ of\ Arms\ Visited - 2)}\right] \times 100$$

Formula 2: Percentage alteration

Morris Water Maze

As previously reported, the rat cognitive function was tested using the Morris water maze (MWM) test. Round shaped water reservoir 50 cm in height and 150 cm in diameter constructed of black plastic and filled with water up to 30 cm served as the testing equipment. Full cream milk (liquid) was used to made water opaque. The water reservoir was split into four equal

quadrants labeled as W, X, Y, and Z. During the acquisition phase, a stand made by iron was put one cm above the water surface in the target quadrant. The same stand was put 1 cm under the water level for retention phase. During the assessment of both stages, the stand location was not modified. Each trial required placing the rat in one of the four pool quadrants. During the acquisition phase, rats were free to swim until they discovered the escape platform. The rat's time to search the visual stand was used to calculate the initial acquisition latency (IAL). During the first acquisition phase, rats were tested sequentially four times each day for four days. time it took to reach the concealed platform on day 5 was recorded and is known as escape latency (EL). Table 1 shows the order of the trials.[18]

Biochemical Parameters Measurement in Brain

Rats were euthanized by cervical dislocation method on 23rd day of the study, brain was removed, kept on a watch glass containing ice and the brain distributed into two equal parts called hemispheres. One hemisphere was stored in cold, normal saline. A 10% sample was produced using 30 millimole of Na_3PO_4 in a homogenizer. The above sample was rotated at 20,000 rpm for 2 hours at 4°C, and the supernatant was collected in a tube and used to quantify AchE and antioxidant parameters. Other hemisphere was stored in 4% formalin for histopathological analysis. Bovine serum albumin method was used for the quantification of conc of protein.[19]

AchE level

The level of AchE enzyme was assessed as per the Ellman technique. Ellman reagents are abbreviated as DTNB. In a test tube 0.5 mL brain supernatant, 2.7 mL phosphate buffer and 0.1 mL DTNB were also added. The above solution was stored for five minutes. After that 0.1 mL acetylthiocholine iodide was mixed and at 412 nm, absorbance was noted.[20]

Malondialdehyde level

The level of malondialdehyde (MDA) was assessed as per Ohkawa procedure. In 1-mL of 10% trichloroacetic acid (TCA), 0.5 mL supernatant was added. The above mixture rotated for 10 minutes. The supernatant was collected. From the 8% TBA 0.1 mL was taken in a tube and 0.2 mL supernatant was also added. The solution was kept on water bath at 80°C for 40 minutes before being cool at room temperature. At 532 nm, absorbance was noted. MDA level was measured as nM/mg protein.[21]

Nitric oxide level

Nitric oxide (No) level was measured according to the method of Griess. Griess chemical was made by combining 10 mL of 1% solution of sulfanilamide with 10 mL of 0.1% naphthyl ethylenediamine and kept at 4°C. 0.1 mL of Griess chemical was mixed in a test tube already contains 0.3 mL of brain supernatant and 2.9 mL of sterile water. At 548 nm, the absorbance was measured. No level was measured as µM/mg protein.[22]

Table 1: WMM test conducted according to the following sequence of trials

Day one	*Q W*	*Q X*	*Q Y*	*Q Z*
Day two	Q X	Q Y	Q Z	Q W
Day three	Q Y	Q Z	Q W	Q X
Day four	Q Z	Q W	Q X	Q Y

Superoxide dismutase level

Superoxide dismutase (SOD) conc was assessed according to the Kagiyama. 2.9 mL tris HCl buffer was added in a tube containing 0.1 mL of supernatant. After this 25 µL pyrogallol solution was mixed. Absorbance was noted at 0, 30, 60, 90 and 120 seconds for 2 minutes at 420 nm. SOD level was measured as U/min/mg protein.[23]

Catalase level

The catalase amount was quantified by Claiborne procedure. In a test tube 1-mL of 30 mM hydrogen peroxide and 0.1 mL of sample was taken and well mixed. The breakdown rate of hydrogen peroxide was noted to be 240 nm. Catalase level was measured as U/min/mg protein.[24]

Glutathione level

Glutathione (GSH) conc. was assessed using Jollow technique. One ml brain supernatant was added with one mL of four percent sulfosalicylic acid in a test tube. The tube was stored at 4°C for one hour and centrifuged for ten minutes at 2000 rpm. The sample was filtered. After this 0.1 mL of filtered solution was filled in the test tube, 0.2 mL of 100 mM DTNB, and 2.7 mL of 0.1M phosphate buffer was added and shaken properly. After the development of a yellow-colored, the absorbance of the mixture was taken at 420 nm. GSH level was measured as µg/mg protein.[25]

Histopathological Study

The hippocampal lesions of different group were assessed microscopically at 100 X magnifications.[26,27]

Statistical Analysis

All the dada included in this research article employed as mean ± SEM Tukey test was employed after One-way ANOVA to evaluate the data obtained from the NORT, Y maze and biochemical tests. Bonferroni test was employed after Two-way ANOVA to evaluate the data obtained from MWM. Level of significance was measured as $p < 0.5$ in the Y Maze, NORT, MWM and biochemical test. Graph Pad Prism was utilized for statistical analysis (version 8.2.4.).

RESULTS

Extractive Value

Table 2 displays the result of extractive value.

Phytochemical Evaluation

Table 3 display the results of the phytochemical evaluation.

Table 2: Extractive value of EEAC leaves

S. No	*Extractive value of EEAC (% w/w)*
1	7.308 ± 0.46

Phenolic and flavonoid contents measurements

The whole phenolic and flavonoid amount in EEAC leaves was calculated as 194.48 ± 0.723 and 54.83 ± 0.108 mg/g, equivalent to gallic acid and quercetin (mg/g of dry extract), respectively.

Acute oral toxicity study

Female rats were used for acute oral toxicity investigation because they are more susceptible to medicines than males as per the OECD 423 guideline. EEAC has no harmful effects in the above mention dose. No harmful effect was observed for central nervous system (CNS) stimulation measures such as hyperactivity, irritability, tremors, and convulsions. As well as no harmful effects were observed for CNS depressant characteristics such as hypoactivity, narcosis, and ataxia.

Novel Object Recognition test

The result of novel object recognition test (NORT) is represented in Figures 1, 2 and 3. Figure 1 tells the different treatment groups of rats spending time between two same objects A1 and A2. the mean normalized time spent during trial phase exploring two identical objects A1 and A2 by different treatment groups. As per the statistical analysis results, no significant time spend was found between similar objects A1 and A2 of different groups rats ($p < 0.05$). These results show that scop and EEAC do not affect rat learning power during trail phase.

Figure 2 exhibits the mean normalized time spent during the test period by various treatment groups learning two different items, one familiar and one novel. The standardized time to find a novel object in the control group was substantially longer as compared the familiar object ($p < 0.05$). In the scop group significant difference was not found between novel and familiar objects ($p < 0.05$). Treatment with EEAC at a dose of 100 mg/kg does not produce a significant difference between novel and familiar objects. but at a dose of 200 and 400 mg/kg rat expend high time to novel objects as to familiar objects ($p < 0.05$).

Figure 3 reveals that the treatment group's %memory index was considerably more as compared to the control group ($p < 0.05$). Percentage memory index considerably enhanced in rats given scop with EEAC at 100, 200, 400 mg/kg and donepezil at 5 mg/kg compared to group treated with scop ($p < 0.05$). These findings indicated that EEAC therapy protects from amnesia produced by scop in a dose-dependent way.

Y Maze

Y maze results are exhibited in Figures 4 and 5. Figure 4 displays the whole visits in all arm of the Y Maze in 10 minutes. According to the overall visits in the all arm, there were no variations in the number of visits across the treatment groups ($p < 0.05$). Figure 5 depicts the percentage memory index on the Y maze across the treatment groups. In the scop group, the %memory index was considerably less than the control group ($p < 0.05$). EEAC in all dose and donepezil 5 mg/kg considerably increased %memory index to the scop group ($p < 0.05$). These findings showed that EEAC in all dose prevented memory deficits produced by scop in a dose-dependent way.

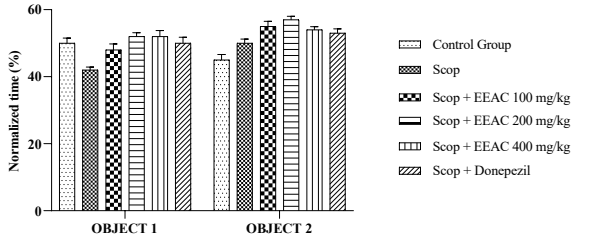

Figure 1: Effect of EEAC leaves in scop induced memory impaired rat at NORT on the time exploring objects during trial phase

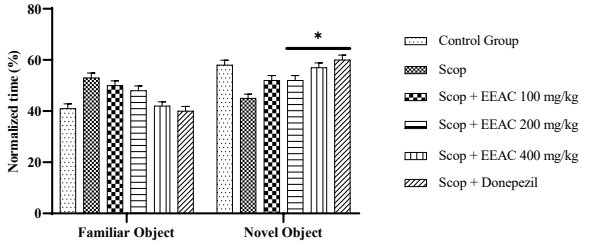

Figure 2: Effect of EEAC in scop-induced memory impaired rat at NORT on the time exploring objects during test phase

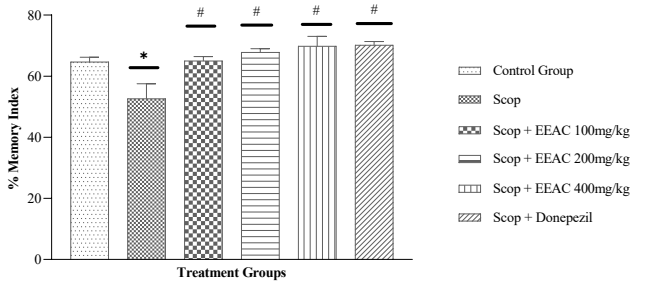

Figure 3: Effect of EEAC on NORT in Scop-induced memory impaired rats. Data expressed as mean ± SEM (n = 6). *$p < 0.05$ *vs* control group, # < 0.05 *vs* Scop group

Table 3: Phytochemical screening of EEAC leaves

S. No.	*Phytoconstituent category*	*Test name*	*Observations*	*Results*
1	Carbohydrates	Fehling's	brick red precipitate	+
2	Alkaloids	Dragendroff's	orange red color precipitate	+
3	Saponins	Foam test	foam formation	+
4	Tannins	Ferric chloride test	brownish green color	+
5	Flavonoids	Shinoda test	red to pink color	+

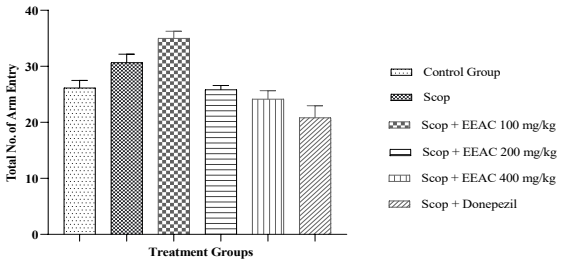

Figure 4: Effect on EEAC on total numbers of arm entries during 10-minute session on Y Maze in scop induced memory-impaired rats. Data s as mean ± SEM (n = 6)

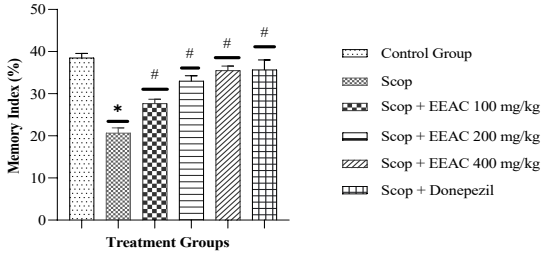

Figure 5: Effect of EEAC on memory index on Y maze in scop-induced memory impaired rats. Data represented as mean ± SEM (n = 6). *$p < 0.05$ *vs* control group, # < 0.05 vs scop group

Morris Water Maze

Figure 6 depicts the findings of the escape latency in the morris water maze (MWM) test. A statistically significant difference in escape delay ($p < 0.05$) was observed amomg the treatment groups. The comparison of days among the treatment groups revealed that the mean escape latency on day 5 in the control group was considerably lower compared to the first day ($p < 0.05$). EEAC in all dose and donepezil 5 mg/kg, the mean escape latency on the fifth day was considerably lower compared to the first day ($p < 0.05$).

Figure 7 displayed the time spent by rats of different groups in target quadrant during the probe trial. In target quadrant, the scop group spend less time than the control group ($p < 0.05$). After comparing results among the group, EEAC in all doses and donepezil 5 mg/kg spent considerably more time in the target quadrant than scop group ($p < 0.05$). These findings showed that all doses of EEAC prevented memory deficits produced by scop in a dose-dependent way.

Biochemical Parameters

The level of biochemical parameters was measured in rat brain tissue homogenates. Scop treated group, showed the significantly increased level of AchE (Figure 8), MDA (Figure 9) and NO (Figure 10), while the decreased level of SOD (Figure 11), catalase (Figure 12) and GSH (Figure 13) as compared to control group ($p < 0.05$). EEAC in all dose and donepezil 5 mg/kg showed the significantly decreased level of AchE, MDA and NO while increased level of SOD, Catalase and GSH as compared to Scop treated group ($p < 0.05$). These findings showed that all doses of EEAC prevented memory deficits produced by scop in a dose-dependent way.

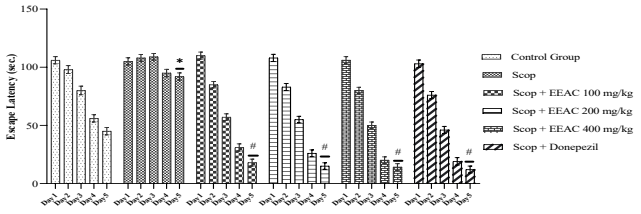

Figure 6: The effect of EEAC on escape latency on MWM test in scop-induced memory-impaired rats. Data represented as mean ± SEM (n = 6). *$p < 0.05$ vs control group, #$p < 0.05$ vs Scop group

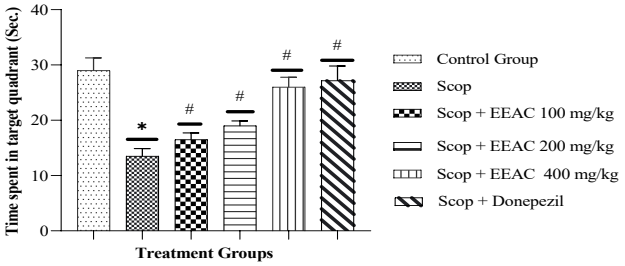

Figure 7: Effect of EEAC on time spent by rats of different groups in the target quadrant during probe trial session of MWM. Data are represented as Mean ± SEM (n = 6). *$p < 0.05$ vs Control group, #$p < 0.05$ vs Scop group

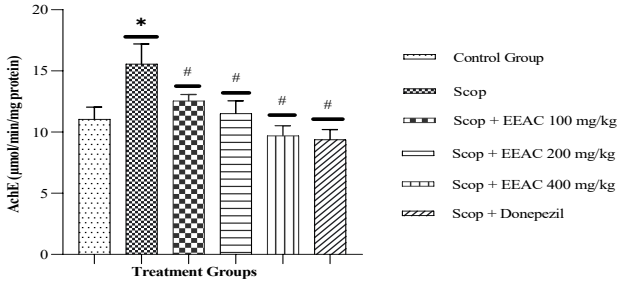

Figure 8:. Effect of EEAC on level of AchE in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *$p < 0.05$ vs control group, #$p < 0.05$ vs Scop group

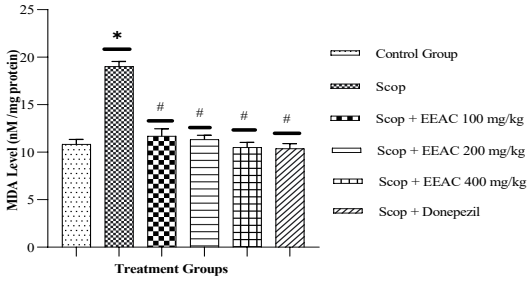

Figure 9: Effect of EEAC on level of MDA in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *$p < 0.05$ vs control group, #$p < 0.05$ vs Scop group

Histopathological Study

From Figure 14, it was clearly visible that in Figure (B) Scop treated group, hemorrhagic patches are more visible as compare

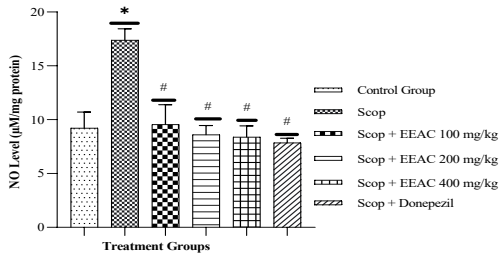

Figure 10: Effect of EEAC on level of NO in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *p < 0.05 vs control group, #p < 0.05 vs Scop group

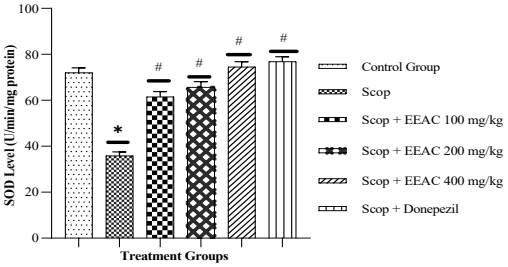

Figure 11: Effect of EEAC on level of SOD in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *p < 0.05 vs Control group, #p < 0.05 vs Scop group

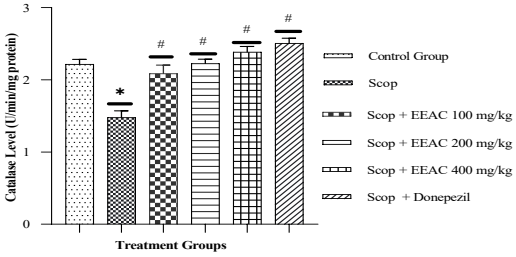

Figure 12: Effect of EEAC on level of Catalase in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *p < 0.05 vs Control group, #p < 0.05 vs Scop group

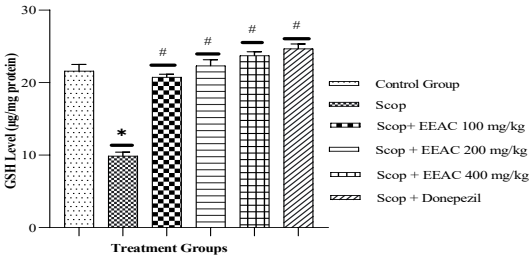

Figure 13: Effect of EEAC on level of GSH in Scop-induced memory-impaired rats. Values represented as Mean ± SEM (n = 6). *p < 0.05 vs Control group, #p < 0.05 vs Scop group

to other group's images. It means scop causes brain injury. As par the results, the treatment of rat given scop with EEAC at 100, 20 0, 400 and donepezil 5 mg/kg the hemorrhagic patches are less or very less as compare to scop-treated group. It means we can say that EEAC showed protective effect against scop-induced memory impairment.

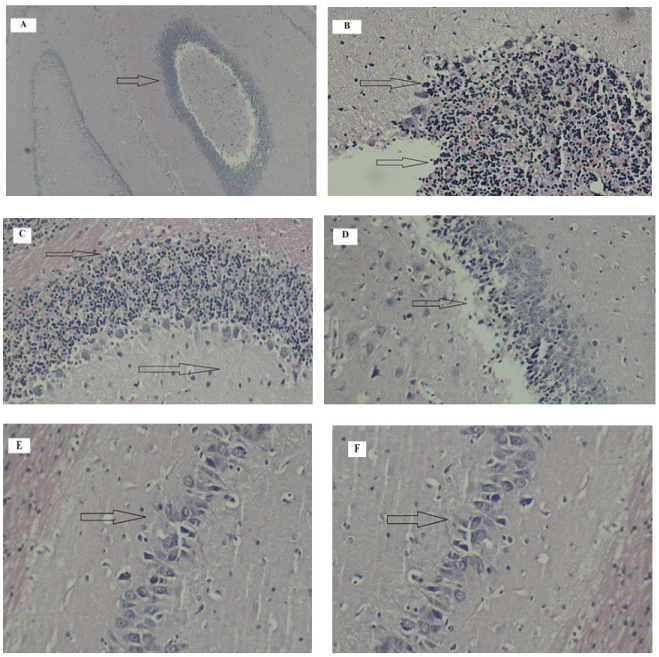

Figure 14: Histopathological figures of rat brain. Arrows showed the hemorrhagic patches in the hippocampus area. (A) Normal control- No Hemorrhagic patches, (B) Scop treated group shows more hemorrhagic patches, (C) Scop + EEAC 100 treated group shows less hemorrhagic patches as compare to Scop treated group, (D) Scop + EEAC 200 treated group shows less hemorrhagic patches as compare to Scop + 100 Treated group, (E) Scop + EEAC 400 treated group shows less hemorrhagic patches as compare to Scop + 200 Treated group, (F) Scop + Donepezil 5 treated group shows less hemorrhagic patches as compared to all other group except Normal control group

DISCUSSION

Neuroprotective and anti-amnesic activity in rat was investigated using various behavioral and biochemical estimations and histopathological studies. The NORT evaluated anti-amnesic activity in rat. Small laboratory animals prefer to look into novel items for a longer period of time than they do familiar objects.[28] The NORT contains two successive phases, trial stage and a test stage. During trial stage, the rat examined two same objects (Plastic ball). Investigated time for the two objects in the treatment groups was equal because both objects were novel to rats. During test stage, any one objects was changed to a new object (plastic Square). As a result, the exploring duration of the new object in the control group was much longer to the familiar object. This is the typical behavior of rodents. Scop treated group, there was no considerable change in the exploring time of the novel object to the familiar object. No considerable changes were found between new and familiar objects in the scop-treated group. Results tells scop produces memory impairment in rat. after treatment with EEAC in all doses, rats spent more time with novel objects than familiar objects. The results of %memory index also suggested that EEAC produces anti-amnesic effect in dose-dependent way.

The results of Y maze study are shown in Figures 4 and 5. Rats love to visit new locations that rat have never been to

before. Alteration behavior might, therefore, be utilized to study spatial memory in rats.[29,30] In the scop-treated group, percentage alteration significantly decreased compared to the control group, but after the scop with EEAC at a dose of 100, 200, 400 mg/kg, percentage alteration significantly increased compared to the scop-treated group. These results indicate that EEAC in all doses improves memory impairment produced by scop in a dose-dependent manner.

In the MWM test, the escape latency of the control group reduced slowly from day 1 to 5, indicating skills of rats. The escape latency of scop-group, remarkably increased from day 1 to 5, indicating memory impairment of rats. After treatment of scop with EEAC at dose of 100, 200 and 400 mg/kg, the escape latency significantly reduced slowly from day 1 to 5. This indicates that EEAC reverse memory impairment caused by scop in a dependent manner. To conform MWM result, we also go for analyze time spent in target quadrant by rat. A significant amount of time spent in the target quadrant showed improved recall.[31] According to our findings, time spent in the target quadrant was reduced in the scop-treated group. This result revealed that scop caused memory impairment. time compared to other groups. This result revealed that scop caused memory impairment. EEAC-treated rats stay longer in target quadrant, indicating improved memory. This demonstrated that EEAC effectively improves the cognitive loss produced with scop.

It has been observed that increasing AchE lower acetylcholine levels in the brain, resulting in memory loss. scop is a muscarinic antagonist that raises acetylcholinesterase (AchE) levels, and it has also been claimed that reduced muscarinic activity causes memory loss.[32,33]

Free radicals mediate oxidative stress, the main trigger in the pathogenesis of AD. In this research, the scop-treated group exhibited a substantial rise level of AchE, MDA, and NO while decreasing level of SOD, catalase and GSH. This is an indication of oxidative stress. Present research exhibited that EEAC decreased quantity of AchE, MDA & NO while increasing the level of catalase, SOD and GSH.[34]

Histopathological study results of the scop treated group showed the more hemorrhagic patches than another group. EEAC remarkably decrease the hemorrhage in dose dependent way than scop group, indicating that EEAC produce a protective effect against scop-induced amnesia in rats.

CONCLUSION

The outcomes of this research clearly indicated that phenolic and flavonoid-rich EEAC leaves have neuroprotective and anti-amnesic effect against scop induced memory impaired rat, as evidenced by NORT, Y Maze and MWM test data and biochemical data. Gallic acid and quercetin were found in rich amounts in EEAC leaves, which are responsible for their neuroprotective and anti-amnesic activity. According to the findings, the polyphenol-rich EEAC has a substantial potential for future use as a natural antioxidant and dietary source to prevent scop-induced oxidative stress-induced memory impairment.

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