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Evaluation of Neuropharmacological Effect of *Andropogon muricatus* Root Extract in Rats

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Abstract: The present study investigated the therapeutic capabilities of *Andropogon muricatus* root extract (AMRE) in combating Alzheimer's disease (AD)-like symptoms induced by scopolamine in rats. The study aimed to explore AMRE's neuroprotective properties using behavioural, biochemical, and histopathological analysis. AMRE, recognised for its abundant bioactive compounds such as saponins, flavonoids, tannins, phenols, and glycosides, exhibited antioxidant properties primarily mediated by polyphenols, which could potentially mitigate free radical formation associated with AD pathology. Behavioural assessments, including the Radial Arm Maze (RAM), Elevated Plus Maze (EPM), and Novel Object Recognition Test (NORT), demonstrated AMRE's effectiveness in reversing scopolamine-induced cognitive impairments, evidenced by reduced errors, decreased task completion time, and enhanced spatial and recognition memory. Biochemical analysis indicated that AMRE restored neurotransmitter levels and reduced oxidative stress markers, further supporting its neuroprotective mechanisms. Histopathological examination underscored AMRE's potential to mitigate neuronal degeneration and amyloid plaque deposition triggered by scopolamine, highlighting its neurorestorative effects crucial for arresting AD progression.Such outcomes highlight AMRE's encouraging efficacy as a sustainable treatment for AD, prompting further investigation into its precise molecular mechanisms and potential translation into clinical applications.

Keywords: *Andropogon muricatus*, Alzheimer, Rivastigmine, Scopolamine, Flavonoids

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Introduction

Alzheimer's dementia is a major obstacle within the landscape of neurodegenerative disorders, demanding rigorous investigation and innovative approaches for effective understanding and

intervention, casting a shadow of cognitive decline over the ageing population. With its insidious onset and progressive deterioration (DeTure and Dickson, 2019), AD imposes a significant burden on individuals, families, and healthcare systems worldwide. Despite decades of research, the precise aetiology of AD remains elusive, fueling an urgent quest for effective interventions (Trejo-Lopez *et al*., 2022). In these times, there has been a notable increase in the occurrence of dementia, correlating with the growing longevity of the worldwide population. The World Health Organization (WHO) estimates that fifty million people worldwide are currently living with cognitive impairment, and that about ten million new instances are diagnosed every year. Projections suggest a steep upward trajectory, with estimates foreseeing 82 million affected individuals by 2030 and a staggering 152 million by 2050, with Alzheimer's disease accounting for a substantial proportion of cases (Hoang *et al*., 2020).While the exact mechanisms driving AD progression remain incompletely understood (Jain *et al*., 2023), emerging evidence implicates oxidative stress and neurotransmitter imbalances as critical players in its pathogenesis. Increased concentrations of indicators of oxidative damage, such as substances produced by lipid per, have been detected in AD patients' brains, cerebro-spinal fluid, and plasma, underscoring the role of oxidative stress in neurodegeneration. Concurrently, disruptions in cholinergic neuro-transmission have been linked to the hallmark memory impairments of AD, emphasizing the importance of neurotransmitter homeostasis in disease pathology (Bai *et al*., 2022; Rummel and Butterfield, 2022).

In the quest to arrest AD progression, research endeavours have explored diverse avenues, including antioxidant interventions and presser-vation of cholinergic function. Among these, natural products have emerged as promising candidates for neuroprotection, offering potential avenues to maintain cellular integrity in the brain and mitigate neuronal loss under pathological conditions (Bagga and Kumar, 2023).

The widely utilised medicinal plant *Andropogon muricatus* holds popularity and significance in Ayurveda, an ancient holistic science practised in India for an extended period. *Andropogon muricatus* is known for its rich biochemical composition and therapeutic potential (Panda and Jhanji, 2020). This plant harbours many bioactive compounds, including alkaloids, flavonoids, tannins, phenols, terpenoids, and saponins, which collectively confer neuro-protective properties (Chen *et al*., 2021). Notably, the ethanolic extract of *Andropogon muricatus* roots has been associated with calming effects on stress, anxiety, and insomnia while exhibiting the potential for cooling the brain and aiding in treating ulcers. *Andropogon muricatus* roots extract demonstrates antihypertensive and antispasmodic (Gilani *et al*., 2007), Calcium Channel Blocking and Phosphodiesterase inhibitory activitiesin airway disorders (Shah *et al*., 2012), analgesic, antipyretic and anti-inflammatory activities (Sharma *et al*., 2010) and hepatoprotective effect (Harshad and Mohan, 2013). Harnessing the bioactive constituents of *Andropogon muricatus* and similar natural products holds promise for developing novel therapeutic strategies to combat AD's multifaceted pathogenesis. By targeting oxidative stress, neurotransmitter imbalances, and associated neurodegenerative processes, these interventions offer hope in the relentless pursuit of effective treatments in relation to dementia.

The present study investigated the therapeutic capabilities of *Andropogon muricatus* root extract (AMRE) in combating Alzheimer's disease (AD)-like symptoms induced by scopolamine in rats. The study aimed to explore AMRE's neuro-protective properties using behavioural, biochemical, and histopathological analysis.

Materials and Methods

Plant Collection and Authentication:

Dried roots of *Andropogon muricatus* were purchased from the local market of Wardha, India. Dr. Vishal N. Patil, Department of Botany,

Vidyabharti College, Seloo, Wardha, India, identified and authenticated the plant. The plant was identified and authenticated as *Andropogon muricatus* of the family Poaceae; Voucher specimen number is 06/rgbacscbotany/2022-2023.

Preparation of extracts:

Dried roots of *Andropogon muricatus* were crushed into powder and then passed into a size twenty-two mesh sieve. Prior to usage, the finely ground roots were stored individually in a sealed box. 200 g of powder endured maceration for 7-8 days at the ambient temperature with periodic shaking after being soaked in 1 L of aqueous ethanol. The concentrate was allowed to air dry after filtration and reduced at 45 °C using a rotating evaporator. The extract was semi-solid, dark brown and sticky. Dried extract was stored in an airtight, light-resistant container. The yield of the AMRE was 12 g, and % yield was 6 %.

Phytochemical screening:

Details regarding the phytoconstituents contained in the basic medication are provided by the initial phytochemical investigation and it is essential for ethno pharmacological screening of drug. Hence, the chemical tests were performed by using the standard procedure to identify the phyto-constituents of *Andropogon muricatus* (Gokhale *et al*., 2016).

Experimental animals:

The rats of White Wistar of either sex, weighing

200-250 g were procured from the livestock home of the Institute of Pharmaceutical Education and Research, Borgaon (Meghe), Wardha (Reg. no.535/PO/ReRcBt/S/02/CPCSEA/IPER/IAEC/2022-23/09). Under typical laboratory circums-tances, the livestock were kept in cages made of polypropylene with six cells each, and they had free access to drinking water and commercial pellet food. The experimental protocol was authorised by the Institutional Animal Ethical Committee of IPER, Wardha with protocol No. 535/PO/ReRcBt/S/02/CPCSEA/IPER/IAEC/2022-23/09.

Study design:

All animals had been divided in 5 experimental groupings. Each group contains six animals. Group I: Normal animals, Group II: Control animals received only Scopolamine, Group III: Standard drug Rivastigmine receiving animals, Group IV and V: Test drug *Andropogon muricatus* roots extract receiving animals. These groups were treated with varying doses of *Andropogon muricatus* roots extract, based on the prior researches, we determined the dose of *Andropogon muricatus* roots extract (Table 1).

Behavioural parameters:

Radial arm maze (*RAM*): A wooden maze with 8 arms was used for the test. The maze's arms measured 15 x 15 x 70 cm. The eight-sided central area, measuring 30 cm in size and at exactly the identical level as the arms, was where the arms stretched. With numerous constant extra-maze indicators, the maze was positioned 100 cm from the ground on a table in the laboratory. Flavoured grains were inserted into grooves 2 cm above each end of Four immovable arms to serve as bait. Rats were moulded for running to the ends of the spreading arms prior to the real training. Eventually, the baits were limited to grooves. Each trial's timing began when the rat was placed in the center of the maze and given full reign to move about. Once the rat had occupied at least half of the arm, arm selections were noted. When the rat selected all of the bait arms or spent 10 min, the trial was said to be over. After the testing started, the arms were not rebaited. For an entire of two and a half weeks-- that is, six days a week--each rat underwent two trials every day, or thirty trials in all. The number of entrances to unbaited arms or re-entry into a baited arm was used to determine errors. The number of times an entry was made to baited arms was used to measure working memory, and the number of times an entry was made to unbaited arms was used to

Table 1: Treatment, dose, and route of administration

S. No.	Groups	Treatment, dose and route of administration
1.	Normal	Saline
2.	Control	Scopolamine (1 mg/kg intraperitoneally)
3.	Standard	Scopolamine (1 mg/kg intraperitoneally) + Rivastigmine (2.5 mg/kg per oral)
4.	Test I	Scopolamine (1 mg/kg intraperitoneally) + AMRE (200 mg/kg per oral)
5.	Test II	Scopolamine (1 mg/kg intraperitoneally) + AMRE (400 mg/kg per oral)

measure reference memory. The average number of reference and working memory errors for every category, calculated over five blocks and six trials everyone was used to calculate the score. It was also determined how long it took on average for each trial to finish the task. (Afrin *et al*., 2022)

Elevated plus maze (*EPM*): The EPM is a widely used behavioral experiment in the study of various facets of animal retention and recall. The building is made up of 2 open arms that measure 50x10 cm and two close arms that have 40 cm-tall walls. By linkingthese arms featuring a 10x10 cm center square, gadget had given the appearance of a plus sign. Additionally, the EPM was set 50 cm above the ground. This makes it simple to move this labyrinth within or out of the testing space as needed and to relocate it right away. All behavioral assessments had been finished from 9 in morning to 6 in the evening in the evening light. EPM was used in two distinct instances to evaluate memory. During the training timing, animals are set up at the end of an EPM arm that is extended. Time it took to every rat for travelling completely in one of the two closed arms was determined using a stopwatch, and this is known as the "transfer latency (s)." To lessen the odor that lingered after each pass, the labyrinth was disinfected with 70% ethanol. Over the course of the two training sessions, the intricate maze was visited by each rat for an entire of 5 min. Reduction in amount of time needed data transfer throughout the experiment were considered to indicate improvement in memory. (Siddique *et al*., 2022)

Novel object recognition (*NOR*) *test*: The NOR assessment is a behavior-based activity that may be completed rapidly in just one learning session and has the benefit of not requiring any sort of consequence or reward. When an unfamiliar thing is placed next to a known one, rodents explore the unfamiliar one more thoroughly than they do the previous object. The NOR challenge, which is used to research how mice memories function, is based on this seeming unrestricted preference for an unfamiliar object, which suggests that an illustration of the known thing is present in memory. The apparatus consisted of a small black-painted chamber with dimensions 50 x 50x 50 cm. Rats were acclimated for 10 min before starting the task. Although there are other variations of the NORT test, it usually consists of two trials. The animal is exposed to one or two identical objects (sample objects) during the first trial (TI). Following the animal's exposure to the sample object, his home cage is given back to him for a period of retention. The animal is brought back to the arena in the second trial (T2), which happens after the retention period, and is shown a new and familiar object (the sample). The subject will investigate the novel object far more than the familiar one when its 'recalls' the prior exposure to the familiar thing. Each trial's effectiveness for a particular animal was evaluated using a factor. (Martin *et al*., 2020).

Biochemical Estimation:

Preparation of The Brain Homogenate: On the 29th day, the animals were euthanised, and their cerebral cortex was taken out. After being cleaned with regular aqueous solution the brain tissues were ground up in a tissue homogenizer using 0.1 M phosphate buffer (pH 7.4). There were ten additions of buffers (w/v) and a 10 min centrifugation at 10,000 rpm. Several portions of the supernatant were taken for the metabolic test (Jha *et al*., 2018).

Estimation of Reduced Glutathione (*GSH*): Glutathione was assessed by spectrophotometry**.** 130 µl of 0.04% 5,5-dithiobis(2-nitrobenzoate), 1.1 ml of 0.25 M buffered sodium phosphate (pH 7.4), and 50 µl of the its supernatant were combined to create a chemical reaction solution. Adding purified water, the solution's ultimate volume was increased to 1.5 ml, and a spectrophotometer was used to measure the absorbance at 412 nm. The findings are presented as µmol/mg (Vasava *et al*., 2022).

Estimation of Catalase (*CAT*): The catalase activity was determined using the previously published methodology. 0.1 ml of the supernatant and 2.9 ml of 10 mM H_2O_2 in 50 mM potassium phosphate buffer (pH 7) made up the reaction solution. The frequency of fluorescence decline was measured for 3 min by spectrophotometer at the wavelength of 240 nm. The findings are presented as µmol/mg (Vasava *et al*., 2022).

Estimation of Malondialdehyde (*MDA*): Malondialdehyde, is the final by-product of peroxidation of lipids. 1.5 ml of 20% (v/v) acetic acid (pH level reduced to 3.5), 0.2 ml of 8.1% (w/v) SDS, and 1.5 ml of 0.8% (w/v) TBA (aqueous solution) were combined with the tissue homogenization (0.2 ml). After adding purified water to bring the end result down to 4.0 ml, the mixture was incubated at 95 °C for 60 min. Additionally, upon climate control, 5 ml of a 15:1 v/v mixture of n-butanol and pyridine was introduced along with 1 ml of purified water. After centrifuging the mixture for 10 min at 4000 rpm, a spectrophotometer was used to measure the chemical layer's thickness at 532 nm. Concentrated activity of malondialdehyde is given as nmol/mg (Shunan *et al*., 2021)

Estimation of the Nitrite Content (*NO*): 0.2 ml of the supernatant was mixed with a Griess reagent fluid, and the mixture was then measured. When nitrite is converted from nitrate, a purple azo molecule is created that may be measured spectrophotometrically at 546 nm. The results are expressed in nM/mg (Gunalan *et al*., 2023).

Estimation of Acetylcholine Esterase: AChE activity was analysed by Ellman method. The brain homogenate (50 µl), 3 ml of 0.1 M phosphate buffer (pH 8), 0.1 ml of 14 mM acetylcholine iodide, and 0.1 ml of 10 mM 0.15,5-dithiobis(2-nitrobenzoate) were mixed. For 5 min, entire solution was incubated. Using a spectrophotometer, the rise in absorbance was recorded every 2 min for a total of thirty seconds at 412 nm. AChE's effectiveness is measured in units of mg (Injamuri *et al*., 2022).

Histopathological Examination:

One rat from each group was euthanised, and the cortex was removed; the brain regions were cleaned to eliminate excess tissue and fats. The clean brains were placed in a container with a 10% formalin solution. The Kanere Histopathology Laboratory, Nagpur, India processed the tissue specimens,

Statistical Analysis:

For evaluation of data, Graph Pad Prism 9.5.1 for Windows was used. The findings are presented as Mean±S.E.M. The degree of importance of the variations in the variables in every group was tested using One-way and Two-way ANOVA, as well as the Bonferroni test. Less than 0.05 P values are regarded as statistically significant.

Results

Phytochemical analysis of plant extract: The *Andropogon muricatus* root extract contains various chemical components including flavonoids

Table 2: Phytochemistry of *Andropogon muricatus* root extract

S. No.	Phytochemical Constituents	Tests	*Andropogon muricatus* root extract
1.	**Flavonoid**	NaOH test $FeCl_3$ test Lead acetate test	+ve +ve +ve
2.	**Alkaloids**	Dragendroff's test Mayer test Hager test	+ve +ve +ve
3.	**Phenol**	$FeCl_3$ test	+ve
4.	**Sterol**	Salkowski test	+ve
5.	**Saponin**	Froth test Foam test	+ve +ve
6.	**Tanin**	Gelatin test	+ve
7.	**Glycosides**	Keller Killiani test Bromine test	+ve +ve

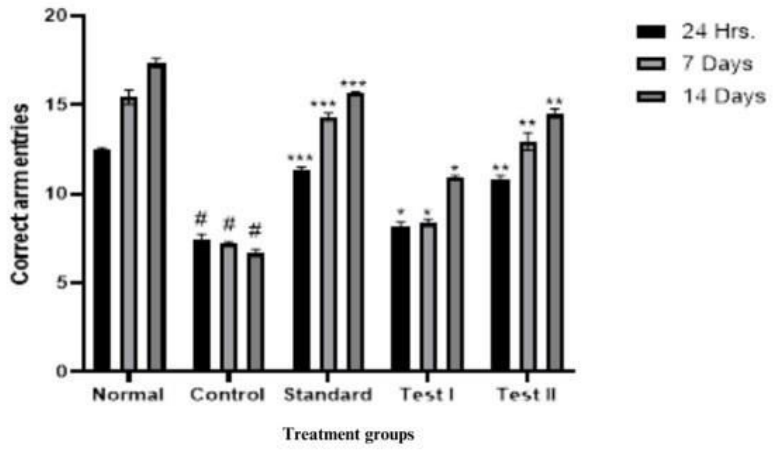


Fig. 1: Effect of AMRE on Correct Arm Entries: The six animal values (n = 6) in each group are presented as a mean ±S.E.M. #P<0.001 in comparison to the saline group. In comparison to the Scopolamine groups, *P<0.05, **P<0.01, and ***P<0.001 were found.

alkaloids, phenols, sterols, saponins, tannins, glycosides and carbohydrates (Table 2).

Determination of Behavioural Parameters

(A) *Radial arm maze*

Determination of Correct Arm Entries:

Correct arm entry determination is required to check the working memory process of the experimental animal. The experimental result shows the effect of *Andropogon muricatus* root extract against chemically induced Alzheimer's diseases in rats. Correct arm entries decrease after receiving scopolamine in all group animals except the normal group. The percentage of accurate arm entries increased considerably in 24 h, 7 days and 14 days after treatment with AMRE both 200 and400 mg/kg (Fig. 1).

Determination of Total Number of Errors:

A number of errors indicate the correct functioning of the brain. If errors increase, it means brain functioning is affected, which can be

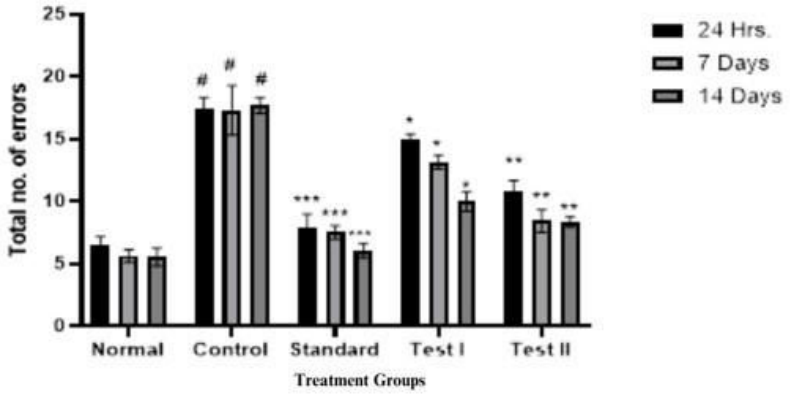


Fig. 2: Effects of AMRE on the Radial Arm Maze's Overall Error Number: In comparison to the Saline group, each group's six animal values (n = 6) are presented as a mean ±S.E.M. #P<0.001. *P<0.05, **P<0.01, ***P<0.001 in comparison to the groups that received Scopolamine.

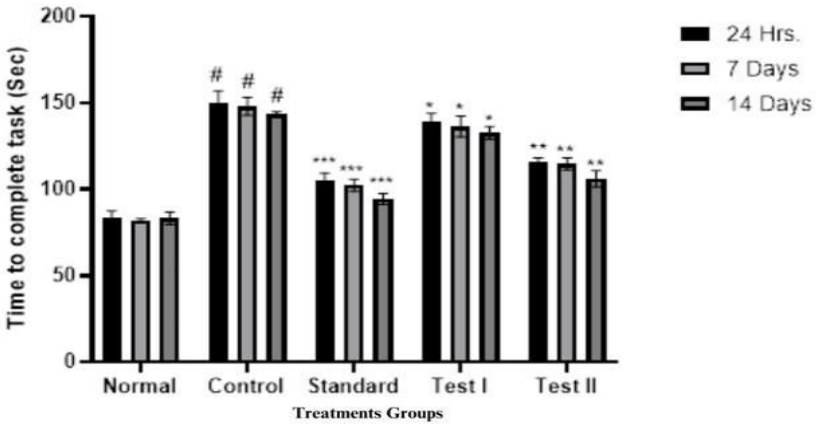


Fig. 3: Effects of AMRE on Radial Arm Maze Task Completion Time: Six animals (n = 6) make up each group, and the values are expressed as a mean ± S.E.M. with a #P<0.001 relative to the saline group. In comparison to the Scopolamine groups, *P<0.05, **P<0.01, and ***P<0.001 were found.

used to determine the functioning of the brain. If the errors decrease, it indicates the memory is retained. The study shows that the total number of errors increases after induction of AD with scopolamine. In contrast, it decreases by the treatment with the standard drug and tests AMRE both 200 and 400 mg/kg (Fig. 2).

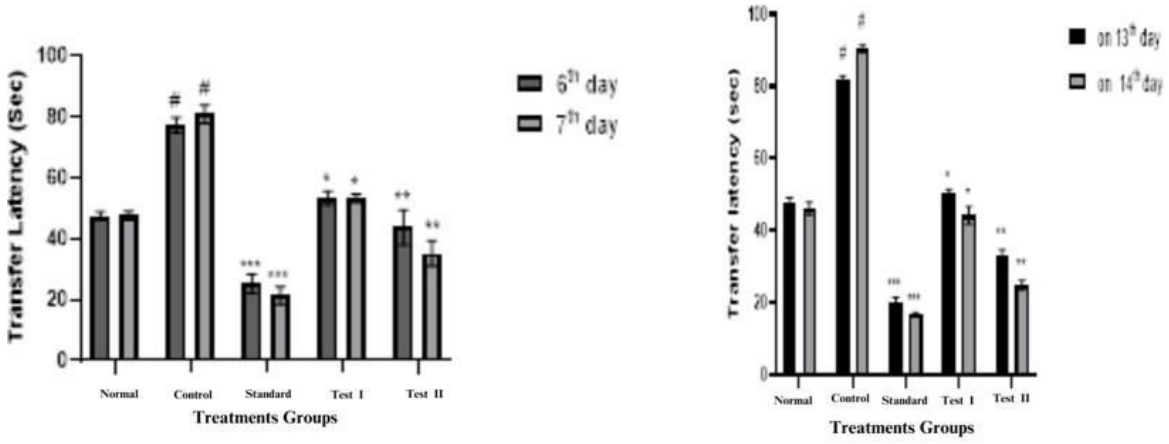
Determination of Time to Complete Task:

Calculating how long a task will take to finish provides the idea about improvement in memory performance in experimental animals. The outcome demonstrates that, when compared with the animals of normal group, the control group's animals took longer to finish the task. Task completion time decreases after receiving the standard drug and 200 mg/kg and 400 mg/kg AMRE within 24 h, 7 days and 14 days in contrast to the group under authority (Fig. 3).

(B) *Elevated Plus Maze* (*EPM*):

The time (seconds) that a rat takes to go from an

Fig. 4:



Effects of AMRE on Elevated Plus Maze Transfer Latency. Six animals (n = 6) make up each group in the Saline group, and the values are given as a mean ± S.E.M. #P<0.001. We observed *P<0.05, **P<0.01, and ***P<0.001 relative to the Scopolamine groups.

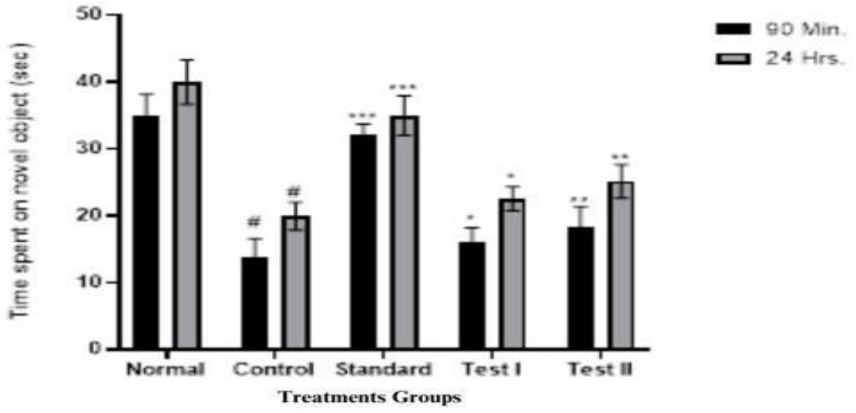


Fig. 5: Impact of AMRE on Time Expended on Novel Object in NORT. Each group consists of six animals (n = 6); the values are expressed as a mean ± S.E.M. with a statistical significance of P<0.001 in relation to the saline group. Plots of *P<0.05, **P<0.01, and ***P<0.001 indicate differences from the groups given Scopolamine.

open arm to an enclosed arm is known as transfer latency (TL). It could be related to memory in the retention phase, which gives an idea about memory performance in experimental animals. The experimental result shows the effect of *Andropogon muricatus* root extract against chemically induced Alzheimer's diseases in rats. Scopolamine treatment significantly increases the transfer latency in contrast to the average group. The treatment resulted in a notable decrease in transfer latency with standard and AMRE 200 and 400 mg/kg, in that order, in contrast to scopolamine-induced group (Fig. 4).

(C) *Novel Object Recognition Test* (*NORT*):

NOR test is a fast-paced, a one-phase learning assignment. Whenever an unfamiliar item is placed next to a known one, rodents explore the unfamiliar one more thoroughly than they do the familiar object. The control group's item identification time was significantly shorter than that of the normal group after 90 min and 24 h of NORT training. On the other hand, an increase in object recognition time following normal and AMRE between 200 and 400 mg/kg treatments (Fig. 5).

Table 3: Effects of AMRE on glutathione levels

S. No.	Groups	Treatment	GSH (µmol/mg)
1	Normal	Saline	0.5417 ± 0.02960
2	Control	Scopolamine (1 mg/kg)	0.3850 ± 0.03731###
3	Standard	Scop +Rivastigmine (2.5 mg/kg)	0.4717 ± 0.04936***
4	Test 1	Scop +AMRE 200 mg/kg	0.4267 ± 0.05051*
5	Test 2	Scop +AMRE 400 mg/kg	0.4700 ± 0.02757**

The mean value is expressed as ± S.E.M. and #P<0.001 when compared to the saline groups. * P<0.05, ** P<0.01, *** P<0.001 in comparison to the scopolamine-taking group. N=6.

Table 4: Effect of AMRE on catalase.

S. No.	Groups	Treatment	Catalase (µmol/mg)
1	Normal	Saline	1.243±0.04835
2	Control	Scopolamine (1mg/kg)	0.6517±0.04729###
3	Standard	Scop+Rivastigmine(2.5mg/kg)	1.132±0.05455***
4	Test 1	Scop +AMRE 200mg/kg	0.7867±0.04680*
5	Test 2	Scop +AMRE 400mg/kg	0.9583±0.04020**

When compared to the saline groups, the mean values of each group, comprising six animals (n = 6), were expressed as ± S.E.M. #P<0.001. * P<0.05, ** P<0.01, *** P<0.001 in comparison to the scopolamine-taking group.

Determination of biochemical parameters:

(A) *Determination of Reduced Glutathione* (*GSH*):

Glutathione level decreases in animals after treatment with Scopolamine compared to the normal group. Whereas there is notable rise in the reduced glutathione level after treatment with Standard drugs and with AMRE 200 and 400 mg/kg, respectively (Table 3).

(B) *Determination of Catalase*:

It was observed that the level of catalase decreases after receiving scopolamine, whereas there is increase in catalase level after treatment with standard drug and 200 mg/kg and 400 mg/kg AMRE (Table 4).

(C) *Determination of Malondialdehyde* (*MDA*):

MDA is a measure of the production of free radicals and a sign of lipid peroxidation. Research has shown that it is essential for memory retention. The result shows significant decreases in the MDA level by treatment with Standard and AMRE both 400 and 200 mg/kg in contrast to the control group, which increases after receiving scopolamine (Table 5).

(D)*Determination of Nitric Oxide* (*NO*):

In comparison to the normal group, rats in groups 2,3,4 and 5 experience higher levels of oxidative damage. When animals in groups 3,4 and 5 are induced with the usual medication and AMRE 200 mg/kg and 400 mg/kg, the concentrations of nitric oxide considerably fall, much like in the regular group (Table 6).

(E) *Determination of acetylcholinesterase* (*AChE*):

The level of AChE increases in animals after

Table 5: The effects of AMRE on malondialdehyde

S. No.	Groups	Treatment	MDA (nM/mg)
1	Normal	Saline	9.058±0.2550
2	Control	Scopolamine (1mg/kg)	26.67±0.8324###
3	Standard	Scop+Rivastigmine(2.5mg/kg)	10.98±1.262***
4	Test 1	Scop +AMRE 200mg/kg	14.83±0.6540*
5	Test 2	Scop +AMRE 400mg/kg	11.71±0.8008**

Values are expressed as a mean, ± S.E.M. #P<0.001, with each group consisting of six animals (n = 6). * P<0.05, ** P<0.01, *** P<0.001 in comparison to the scopolamine-taking group.

Table 6: Effects of AMRE on Nitric Oxide

S. No.	Groups	Treatment	NO (nM/mg)
1	Normal	Saline	4.320±0.2436
2	Control	Scopolamine (1mg/kg)	7.488±0.6533###
3	Standard	Scop +Rivastigmine (2.5mg/kg)	3.955±0.5610***
4	Test 1	Scop +AMRE 200mg/kg	4.487±0.5434*
5	Test 2	Scop +AMRE 400mg/kg	4.098±0.5310**

P<0.001 in comparison to Saline groups; mean values were provided as a mean, ± S.E.M. for each group of six animals. * P<0.05, ** P<0.01, *** P<0.001 in comparison to the scopolamine-taking group.

Table 7: AMRE effects on AChE level

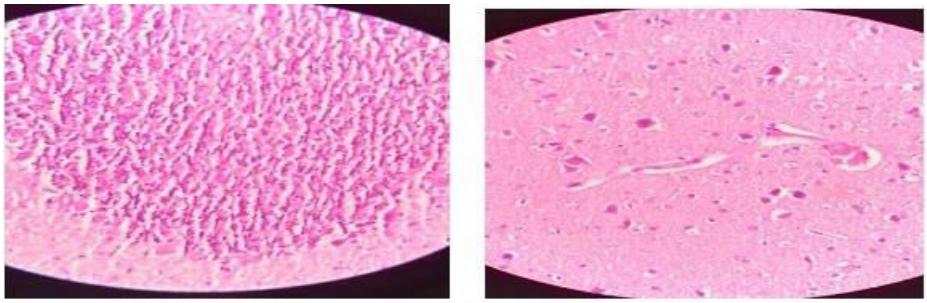
S. No.	Groups	Treatment	AChE Level (U/mg)
1	Normal	Saline	8.475 ± 0.4490
2	Control	Scopolamine (1mg/kg)	18.06 ± 0.9823###
3	Standard	Scop +Rivastigmine (2.5mg/kg)	10.02 ± 0.6299***
4	Test 1	Scop +AMRE 200mg/kg	12.90 ± 0.4900*
5	Test 2	Scop +AMRE 400mg/kg	11.15 ± 0.2527**

Six animals (n = 6) per group; means, ± S.E.M., and #P<0.001 relative to saline groups are reported. * P<0.05, ** P<0.01, *** P<0.001 in comparison to the scopolamine-taking group

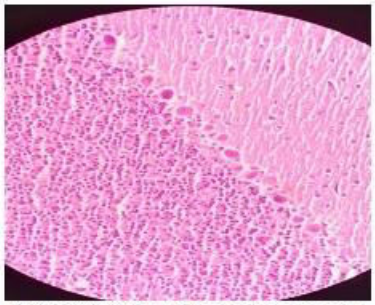
receiving scopolamine in comparison with the normal group. Whereas AChE level decreases in animals receiving standard drug and test AMRE (200 and 400 mg/kg) (Table 7).

Histopathological Study:

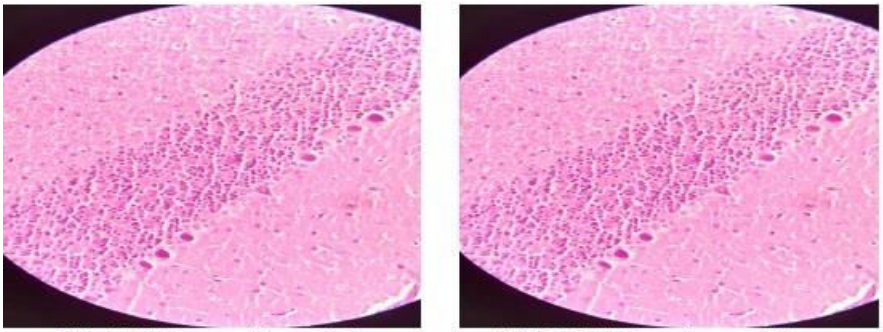
Normal group animal brain tissue exhibits normal cellular organization (Fig. 6A). Animals in the control group that were given scopolamine demonstrated neuron cell death, amyloid plaque (AP) accumulation, and neurofibrillary tangles (Fig. 6 B). Animals given the conventional medication rivastigmine 2.5 mg/kg showed reductions in neurofibrillary tangles and Amyloid plaque (AP) as well as improvement in brain regions with low neuronal degeneration (ND). (NFT) as shown in Figure 6C. Test-treated animals administered 200 mg/kg and 400 mg/kg of *Andropogon muricatus* root extract exhibited normal histology, with mild neuronal cell (NC)



(A) Normal group (B) Control group



(C) Standard drug receiving group



(D) AMRE 200 mg/kg receiving group (E) AMRE 400 mg/kg receiving group

degeneration and the disappearance of amyloid plaque (AP) and neurofibrillary tangles (NFT) (Figs. 6 D, E).

Discussion

Alzheimer's dementia (AD) continues to pose a serious medical problem due to its progressive cognitive decline and limited treatment options with potential adverse effects. Traditional herbal formulations have gained attention as potential alternatives, aiming to mitigate adverse reactions associated with conventional medications. This study investigated the memory-enhancing activity of *Andropogon muricatus* root extract (AMRE) in a rat model of AD produced by scopolamine.

Thus, observed neuroprotective impact of AMRE can be attributed to its rich composition of biologically active substances such as saponins, glycosides, phenols, tannins, and flavonoids. Particularly, ethanolic extract of AMRE demonstrates antioxidant properties, primarily mediated by polyphenols, which counteract free radical formation. Oxidative stress is implicated in AD pathology, leading to neuronal damage and cognitive impairment. AMRE's antioxidative activity suggests a potential therapeutic mechanism in combating AD progression.

Scopolamine, a muscarinic receptor antagonist, induces cognitive impairment in

animals, mimicking AD neuropathological changes. It disrupts cholinergic and noradrenergic neurotransmission, promotes amyloidogenesis, and enhances acetylcholinesterase (AChE) activity, resulting in cognitive deficits. The observed decrease in AChE activity following AMRE treatment indicates its potential to modulate cholinergic neurotransmission, crucial for learning and memory processes.

Behavioural tests, including the Radial Arm Maze (RAM), Elevated Plus Maze (EPM), and Novel Object Recognition Test (NORT), corroborate cognitive-enhancing effects of AMRE. Decreased errors, reduced time to complete tasks, and improved spatial and recognition memory in AMRE-treated rats suggest its efficacy in reversing scopolamine-induced cognitive deficits.

Biochemical assays further elucidate AMRE's neuroprotective mechanisms by restoring neurotransmitter levels and reducing oxidative stress markers. AMRE effectively mitigates scopolamine-induced increases in nitric oxide (NO) and malondialdehyde (MDA) levels, while enhancing reduced glutathione and catalase activity. These findings underscore AMRE's multifaceted neuroprotective properties, encompassing antioxidant, anti-inflammatory, and cholinesterase inhibitory activities.

Histopathological examination reveals AMRE's potential to ameliorate neuronal degeneration and amyloid plaque deposition induced by scopolamine. Preservation of neuronal integrity and reduction in amyloid plaques and neuro-fibrillary tangles signify AMRE's neuro-restorative effects, crucial for halting AD progression.

Conclusion

This study indicates that AMRE has promising therapeutic potential in mitigating scopolamine-induced AD-like pathology in rats. Its multifaceted pharmacological profile, including antioxidative, cholinesterase inhibitory, and neuroprotective properties, underscores its candidacy as a natural remedy for AD. The goal of future studies should be to clarify the exact molecular pathways. underlying AMRE's therapeutic effects and its translational potential in clinical settings.

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Conflict of Interest

Regarding this article, the authors have disclosed no relevant conflicts of interest.

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