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RESEARCH ARTICLE

COGNITIVE ENHANCING ACTIVITY OF SEMECARPUS ANACARDIUM IN SCOPOLAMINE INDUCED MEMORY IMPAIRMENT IN MICE.

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Abstract:

The present study was undertaken to investigate the effect of stem bark of Semecarpus anacardium (SA) on cognitive functions in scopolamine induced memory impairment with behavioral models Morris water maze, Object recognition test, Passive avoidance test. Memory impairment is induced by administration of scopolamine (1 mg/kg) in to the grps of mice (25-30 gm). Mice treated with extract of stem bark of Semecarpus anacardium (25, 50, 75 mg/kg) . Qualitative phytochemical screening of aqueous extract of SA shows a Alkaloids, tannins, flavonoids, phenolics compounds, terpenoids and steroids. Mean escape latency in Morris water maze, Discrimination index in object recognition test, were also decreased.

Keywords: Semecarpus anacardium, Memory impairment, Behavioral study, stem bark, Phytochemical study.

Introduction

Alzheimer disease is a neurodegenerative disorder produces impairment of cognitive abilities that is gradual in onset but relentness in progression ..AD refers to dementia that does not have an antecedent cause, such as stroke, brain trauma or alcohol. Its prevalence rises sharply with age, from about 5% at 65 to 90% or more at 95. Until recently, age-related dementia was considered to result from the steady loss of neurons that normally goes on throughout life, possibly accelerated by a failing blood supply associated with atherosclerosis.[2] Impairment of short-term memory usually is the first clinical feature, whereas retrieval of distant memories is preserved relatively well into the course of the disease. As the condition progresses, additional cognitive abilities are impaired, among them the ability to calculate, exercise visuospatial skills, and use common objects and tools (ideomotor apraxia). The level of arousal or alertness of the patient is not affected until the condition is very advanced, nor is there motor weakness, although muscular contractures are an almost universal feature of advanced stages of the disease. Death, most often from a complication of immobility such as pneumonia or pulmonary embolism, usually ensues within 6 to 12 years of onset.[3]

Semecarpus anacardium Linn. (Family: Anacardiaceae) is distributed in sub-Himalayan region, tropical and central parts of India. The nut is commonly known as 'marking nut' and in the vernacular as 'Ballataka' or 'Bhilwa'. It has high priority and applicability in indigenous system of medicine. The nut, fruit, and stem bark having a various medicinal activity.[4-6]

Materials and methods:

Instruments and equipments

Digital balance (Type AW 220, Shimadzu corporation, Japan), Hot air oven (Lab hospital corporation, India), Morris water maze, Object recognition test apparatus, Passive Avoidance test apparatus (step down).

Drugs and Chemicals

All drugs and chemicals required for the experiments were purchased from S.D.Fine Chemical. Scopolamine is obtained from Sigma Aldrich, USA.and Piracetam nootropic drug from medical stores Amravati.

Preparation of Methanolic Extract

The fresh stem bark of plant material was shade dried and powdered mechanically and subjected to soxhlet extraction methanol as a solvent system for 48 hr. The extract were filtered and concentrated in vacuums under reduced pressure using rota rod flash evaporator. Allowing complete evaporation of solvent on a water bath and then finally vacuum dried. The yield of methanolic crude extract (maroon pasty in nature) for 1 kg. Of powder was 35 gm. [7]

Phytochemical Screening [8]

For the qualitative phytochemical investigation 1% aqueous solution of extract was used .following methods for phytochemical screening were applied on methanolic extract.

1) Alkaloids: The extract was evaporated to dryness and the residue was heated on on a boiling water bath with 2 N hydrochloric acid (5 ml.). After cooling, the mixture was filtered and the filtrate was divided into two equal portions. One portion was divided into two equal portions. One portion was treated with a few drops of Mayer's reagent and the equal amounts of Wagner's reagent. The samples were then observed for the presence of turbidity or precipitation.

2) Saponins: The extract was shaken vigorously in a test tube to froth and was then allowed to stand for 15-20 minutes and determined for saponin content.

3) Tannins: A few drops of 0.1% of ferric chloride was added to solution of extract.

4)Flavonoids: On addition of increasing amount of sodium hydroxide, the extract showed yellow coloration, this decolorized after addition of dilute hydrochloric acid.

5) Phenolic compounds: The extract was diluted to 5 ml. with distilled water. To this a few drops of neutral of 5% of ferric chloride solution was added.

6) Terpenoids and steroids : 1 ml. of chloroform added to extract and filter. 1 ml. of acetic acid was added to the filtrate. Then few drops of sulphuric acid were pour down the side of test tube.

7) Carbohydrates: Molisch's test: Add few drops of alcoholic α- naphthol then add few drops of conc. Sulphuric acid through sides of the test tube.

8) Protein: Biuret test : Extract treated with sodium hydroxide and copper sulphate solution added dropwise and mixed, violet colour.

Millons's test : Extract treated with Millons reagent (Mercuric nitrate in nitric acid), red colour observed.

Nin hydrin test : Extract treated with Ninhydrin reagent and ammonia, heated, violet colour observed.

Behavioral Study:

Treatment schedule

Treatment schedule for the water morris maze, Passive avoidance (Step down) method, Object recognition model are as follows. For the above three model, animals divided into six group and each group containing six animal.

Group I : Vehicle only (water for injection)

Group II : Scopolamine and vehicle

Group III: Standard and Scopolamine

Group IV : Test drug (25 mg/kg) and scopolamine

Group V : Test drug (50 mg/kg) and scopolamine

Group VI ;Test drug (75 mg/kg) and scopolamine

Object Recognition Model [9]

Object recognition apparatus consist open white colored plywood box (70×60×30 cm.) with a well furnished floor. The box is illuminated by 60 w lamp suspended above the box. The object to be discriminated made of plywood in two different shape of 8 cm.and colored black and white.

The object recognition test is a behavioral test that is widely used to examine animal's memory performance. Memory performance in the ORT is based on the natural tendency of animals to explore novel objects.

The day before the test, mice was given habituation session where they were left to freely exploring the box for 2 min. No object was placed in the box during the habituation trial. On the day of test, two identical objects were presented in two opposite corner of the box during the first trial(T_1.). and the amount of time taken by each mouse to complete 20 s of object and/or touching it with nose or forepaw. Turning around or sitting on the object was not considered as an exploratory behavior. During the second trial (T_2, 90 min. after T_1) one of the objects presented in the T_1 (i.e. familiar objects) was replaced by new object and mice was left in box

for 5 min. The time spent (s) for exploration of the familiar (F) and new (N) object was recorded and discrimination index was calculated.

Discrimination Index= N-F/ N+F

Where, N= Exploration of the new object

F= Exploration of the familiar object

Scopolamine (1mg/kg) was injected i.p. after 45 min of administration of Semecarpus anacardium (25, 50, 75 mg/kg) or piracetam (200mg/kg)or vehicle in mice and trial was given 45 min after injection of scopolamine.

Passive Avoidance Test [10]

Passive avoidance test apparatus consist of (27×27×27) having three walls of wood and one wall of plexiglass.feauturing a grid floor (3 mm. of stainless steel rods set 8 mm apart) with a wooden platform (10×7×1.7) in the center of grid floor. The box is illuminated with 15 w during the experimental period. Electric shock (20 v AC) was delivered to the grid floor.

Passive avoidance behavior based on negative reinforcement was used to examine the long term memory. Training was carried out in two similar sessions. Each mouse gently placed on the wooden platform set in the center of the grid floor. When the mouse stepped down and placed all its paw's on the grid floor. Shock was delivered for 15 s and step down latency was recorded. SDL was defined as the time taken by the mouse to step down from wood platform to grid floor with with all its paws. Animals showing SDL in the range (2-15 s) during the test were used for the second session and the retention test. The second session was carried out after 90 min. of first test. When the animals stepped down before 60 s. electric shock was delivered for 15 sec. During the second test, the animals removed from the shock free zone if they did not step down for 60 s. Retention was tested after 24 h in a similar manner, except that the electric shocks were not applied to the grid floor. Each mouse again placed on the platform and the SDL was recorded with upper cut off time 300s.

Semecarpus anacardium extract (25, 50, 75 mg/kg orally), piracetam (200 mg/kg i.p.) or vehicle were administered orally for 8 days and SDl was recorded after 45 min. of administration of last dose on eight day and again after 24 h i.e. on ninth day, in the scopolamine treated group, Scopolamine(1mg/kg) was injected i.p. after 45 min of administration of extract or piracetam or

vehicle and SDL was recorded after of injection of scopolamine on eighth day and after 24 h. i.e. on ninth day .

Morris Water maze [11]

The Morris water maze is a circular pool (100 cm. in diameter and 45 cm. in height) with a featureless inner surface. The circular pool was filled with a water in which 500ml. of milk had been mixed to a height of 30 cm. (20±1 c). The pool was divided into four quadrants of equal area. A white platform (6 cm. in diameter and 29 cm. in height) was centered in one of the four quadrants of the pool and submerged 1 cm below the water surface so it was invisible at water level.

The spatial memory test was performed by the method of Morris .in the water maze experiments, the day prior to experiment was dedicated to swim training for 60 s in the absence of the platform, in the days following the mice were given two trial sessions each day for consecutive days, during each trial, the escape latencies of mice were recorded . This parameter was averaged for each session of trials and for each mouse. Once the mouse located the platform, it was permitted to remain on it for 10s. if the mouse did not locate the platform within 120s, it was placed on the platform for 10s. and then removed from the pool by experimenter. The mouse was given two daily trials for four consecutive days with an inter-trial interval of 20 min. The of entry of mouse in to the pool and location of the platform for escape remained unchanged between trials 1 and 2 but was changed each day thereafter. The in escape latency from day to day in trial 1 represents long term memory or reference memory while that from trial 1 to trial 2 represents short term memory or working memory. Mice were treated with water for injection and test extract preparation before the training trial. After 90 min, amnesia was induced mice with scopolamine given intraperitonially.

Result:

Extraction

Percentage yield of methanolic extract of Semecarpus anacardium was found to be 3.5 %

Qualitative Phytochemical Screening

Table I : **Qualitative phytochemical screening of aqueous extract of Semecarpus anacardium.**

Sr.No.	Phytochemical Screening	Aqueous Extract	
		Observation	**Inference**
1	Alkaloid	Colored precipitate	Presence of alkaloid
2	Saponin	No froth formation	Absence of saponin
3	Tannins	Blue black precipitate	Presence of tannins
4	Flavonoids	Flavonoids dissolve giving yellow color	Presence of flavonoids
5	Phenolics	Dark green	Presence of phenolics
6	Terpenoids and steroids	Reddish brown color and blue color	Presence of terpenoids and steroids
7	carbohydrates	Green color	Presence of carbohydrates
8	Proteins	No Violet color	Absence of proteins

Pharmacological method

Object Recognition method : The results of cognitive enhancing activity of Semecarpus anacardium by Object recognition method are presented in a table. The values presented in the table represent average discrimination index. Discrimination index was significantly decreased ($P<0.05$; $p<0.01$) in the scopolamine grp. as compare to the control grp. While pretreatment with Semecarpus anacardium extract (25, 50, and 75 mg/kg) significantly increased as compare to the

scopolamine treated grp. Result of the test extract is nearer to the standard extract (200mg/kg), indicating improvement in short term memory and reversal of amnesia induced by scopolamine.

Table II : Effects of extract of stembark of Semecarpus anacardium in object recognition model.

Group	Treatment and doses	Discrimination index	%Discrimination index
I	Control (oral)	0.041033±0.002884	100
II	Scopolamine (1 mg/kg in water for inj. i.p.) + vehicle	0.002053±0.002025*	5.004102
III	Standard (piracetam, 200mg/kg, i.p.)+ Scopolamine (1mg/kg, i.p.)	0.039766±0.001347	96.91134
IV	SAE. (25 mg/kg, orally) + Scopolamine (1mg/kg, i.p.)	0.02066±0.00656	50.34972
V	SAE. (50 mg/kg, orally) + Scopolamine (1mg/kg, i.p.)	0.030053±0.000252*	73.24196
VI	SAE. (75 mg/kg, orally) + Scopolamine (1mg/kg, i.p.)	0.035771±0.00931	87.17687

SAE : Semecarpus anacardium extract I.P. : intraperitoneal

Passive Avoidance Test: The effects of extract of Semecarpus anacardium on the scopolamine induced memory deficit were further evaluated using passive avoidance test. All the observation is showed in table

Step down latency of second day (ninth day treatment) indicated the long term memory of animals. The step down latency (SDL) was shortened in mice treated with scopolamine (1mg/kg, i.p.) as compared to that of normal control mice. Extract of Semecarpus anacardium (25, 50 and 75 mg/kg) administered to the mice for 8 days showed increase ($P<0.05$,$P<0.01$)in SDL .Administration of extract of Semecarpus anacardium for 8 days reversed memory deficits due to scopolamine induced amnesia.Piracetam also showed improvement in memory in mice. Treatment of amnesic mice with Semecarpus anacardium extract significantly increased the latency to a level of 70% of normal control mice.

Table III:The enhancing effects of extract of Semecarpus anacardium on memory impairment induced by scopolamine in mice.

Group	Mean& S.E.M.(step down latency)	% of control
Control	226.1667± 1.222719	100
Scopolamine	34.5±0.99183	15.25
Std.(piracetam)+Scopolamine	169±1.183453*	74.72
SEM (25mg/kg)+scopolamine	154±1.238526	68.09
SEM (50mg/kg)+scopolamine	159.8333±0.477356	70.67
SEM (75mg/kg)+scopolamine	164±0.730443*	72.51

6.3.3 Morris Water Maze: The efficacy of Semecarpus anacardium in enhancing cognition after impairment of spatial memory via scopolamine was evaluated through Morris water maze. Control mice rapidly learned the location of the platform from their first training day and reached stable latencies by day 2. By contrast, mice treated with scopolamine (1 mg/kg body weight) failed to find the given platform until given the maximum time limit, 120 s. on days 1 and 2. Amnesic mice treated with Semecarpus anacardium demonstrated a significantly shortened ($p<0.05$,$P<0.01$) interval to find the platform as compared to mice given scopolamine alone.

Table IV: The effects of Semecarpus anacardium extract on memory impairment induced by scopolamine in mice through Morris water maze.

Group	Day	Mean& S.E.M (Escape latency)	Group	Day	Mean& S.E.M. (Escape latency)
Control	1	33.66667±0.4945	scopolamine	1	119±0.4473
	2	24.33333±0.4217		2	118.1667±0.5427
	3	23.5±0.6192		3	117±0.7304
	4	17.66667±0.4945		4	116±0.4473

Table V:The effects of Piracetam on memory impairment induced by scopolamine in mice through Morris water maze.

Group	Day	Mean& S.E.M (Escape latency)	Group	Day	Mean& S.E.M. (Escape latency)
Piracetam + scopolamine	1	51.33333±0.4945	SAE(25mg/kg) Scopolamine	1	67.83333±0.7033
	2	49.33333±0.4217		2	59.66667±0.8820
	3	45.16667±0.6541		3	54.16667±0.4014
	4	42.83333±0.3073		4	51.83333±0.3873

Table VI: The effect of semecarpous anacardium (50mg/kg, 75mg/kg)

Group	Day	Mean& S.E.M (Escape latency)	Group	Day	Mean& S.E.M (Escape latency)
SAE (50mg/kg) Scopolamine.	1	59.83333±0.7924	SAE(75mg/kg) Scopolamine	1	57±0.5775
	2	53.66667±0.4945		2	50.33333±0.4945
	3	54±0.9662		3	48.83333±0.3073
	4	49.83333±0.4773		4	47.16667±0.4773

Above table shows a Mean and SEM of escape latency of all treatment groups. Escape latency of SAE pretreatment grps. decreases on successive days. Mean and Escape latency of SAE on 1st day is 57.833±0.703, 59.833±0.792, and 57±0.5774 and forth day is 51.833±0.307, 49.833±0.4773,and 47.1666±0.477 of a dose 25mg/kg, 50 mg/kg, and 75 mg/kg respectively. So this value indicates the Escape latency decreases on successive days of SAE treatment group.

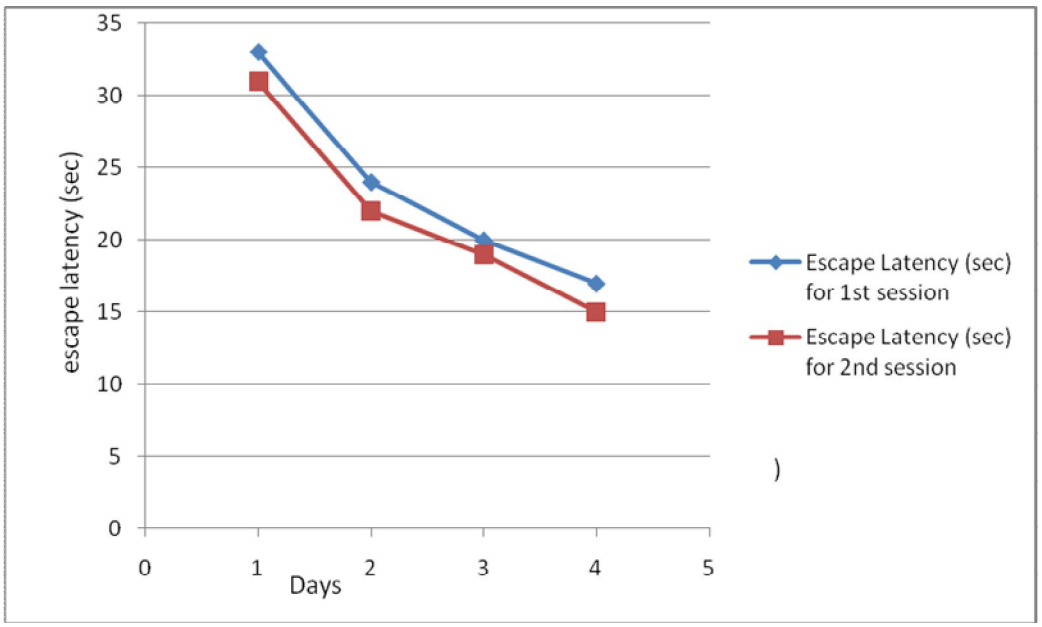

Figure 1.1: Graph representation of escape latency A) Control

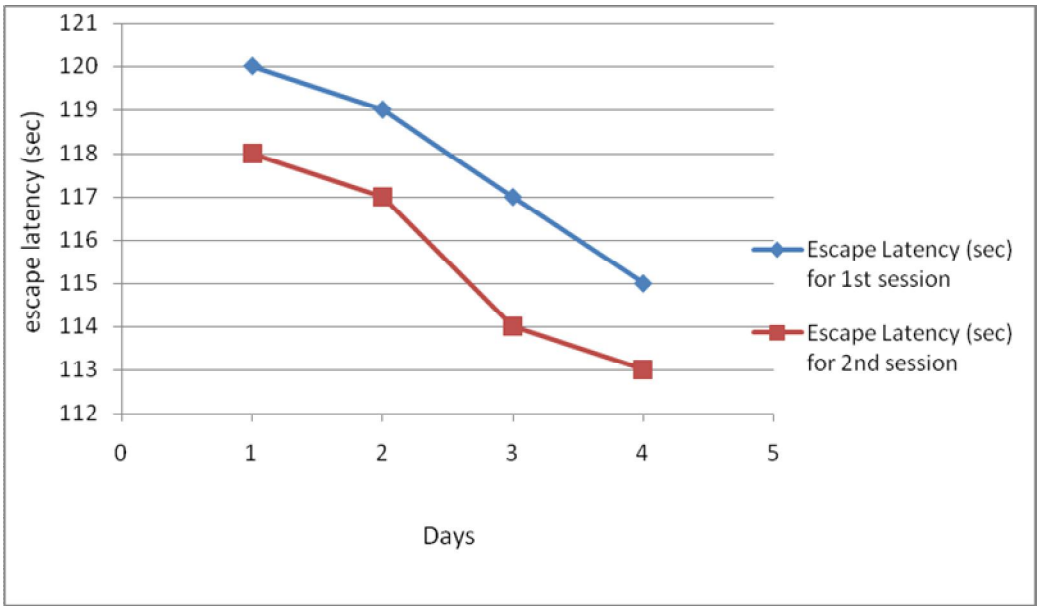

Figure 1.2: Graph representation of escape latency B) Scopalamine

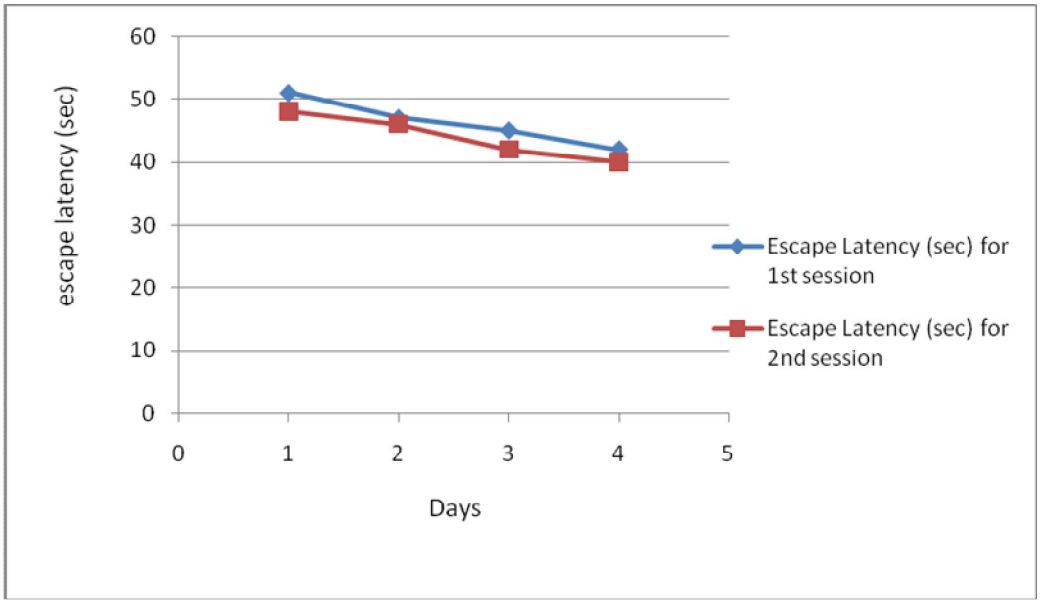

Figure 1.3: Graph representation of escape latency C) Piracetam (std.) + Scopolamine

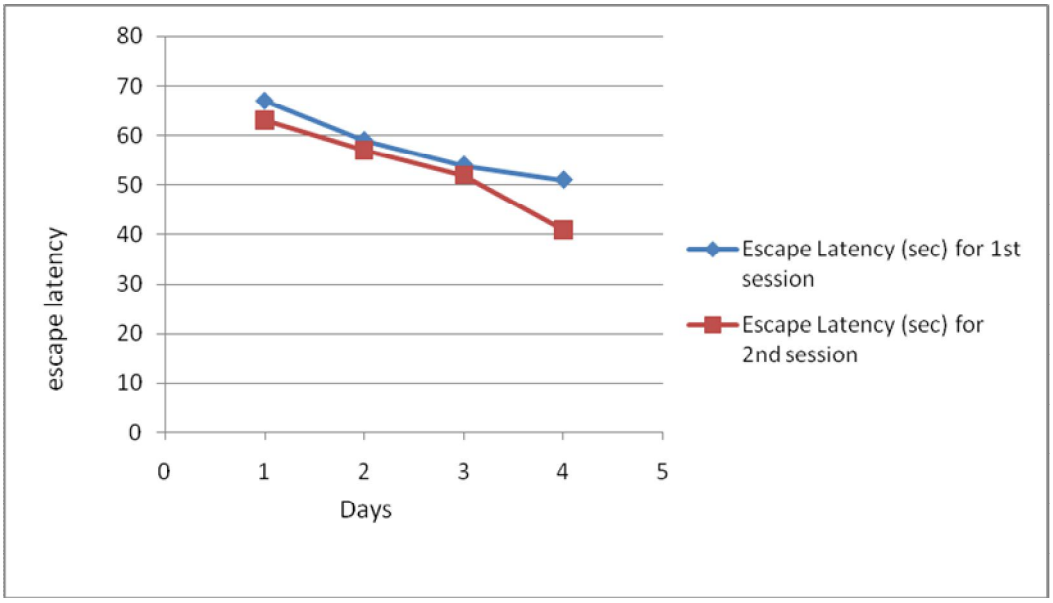

Figure 1.4: Graph representation of escape latency D) S.Anacardium extract

(25 mg/kg.) + Scopolamine

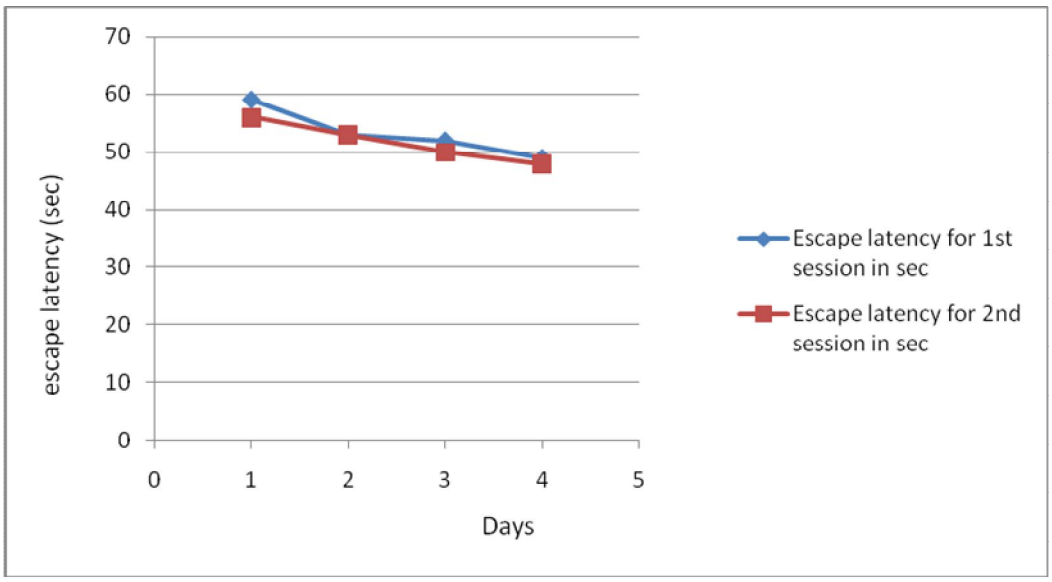

Figure 1.5: Graph representation of escape latency E) S.Anacardium extract (50 mg/kg) + Scopolamine

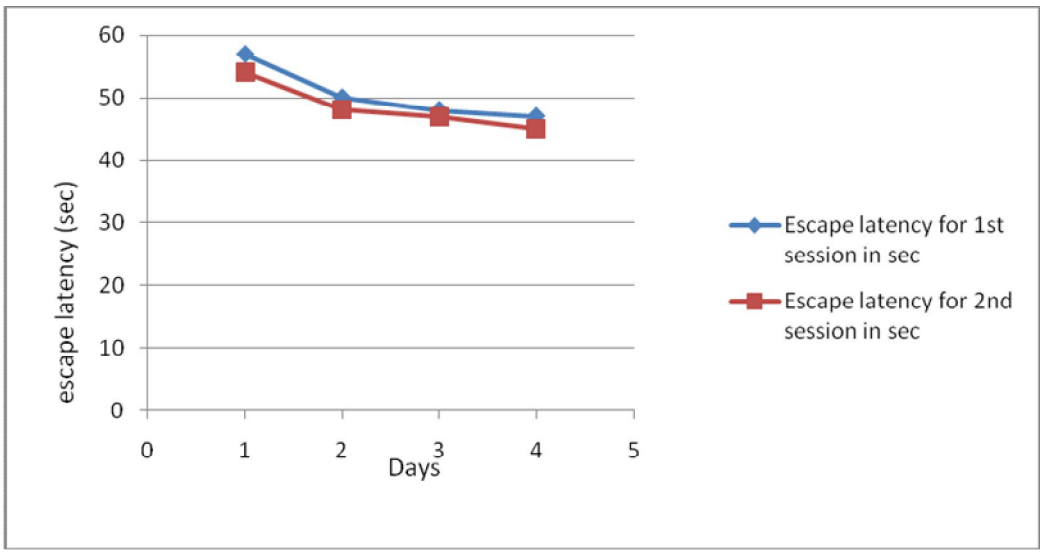

Figure 1.6: Graph representation of F) S. Anacardium extract (75 mg/kg) + Scopolamine

The cognitive enhancing effects of Semecerpus anacardium on spatial memory impairment induced by scopolamine in mice. Mice were given two sessions of trials each day for 4

consecutive days. The swimming time recorded for mouse to the platform was recorded in each day. Each day, mice were treated with SAE (25, 50, 75 mg/kg) . After 90 min. of the treatment , amnesia was induced by scopolamine (1 mg/kg, i.p.) All mice were tested for spatial memory 30 min. after the injection of scopolamine. The values shown are the mean escape latency ± S.E.M. Each grp represents six animals.A) Normal control grp. B) Scopolamine treated grp. (1 mg/kg, i.p.) c) Piracetam (200mg/kg, i.p.) ± Scopolamine (1mg/kg) D) SAE (25 mg/kg) ± Scopolamine E) SAE (50mg/kg) ± Scopolamine F) SAE (75 mg/kg ± Scopolamine)

Discusion:

It is estimated that there are currently about 18 million people worldwide with Alzheimer's disease. This figure is projected to nearly double by 2025 to 34 million. Much of this increase will be in the developing countries, and will be due to the ageing population. Currently, more than 50% of people with Alzheimer's disease live in developing countries and by 2025, this will be over 70%. It is estimated that there are currently about 18 million people worldwide with Alzheimer's disease. This figure is projected to nearly double by 2025 to 34 million. Much of this increase will be in the developing countries, and will be due to the ageing population.

Free radicals are chemical species possessing an unpaired electron. The radical derivatives of oxygen (O2) are the most important free radicals in the biological systems. These radicals are involved in the pathogenesis of many inflammatory diseases like Alzheimer's disease.

The clinical features of Alzheimer's disease (AD) are coupled with a progressive loss of neurones in several different regions of the brain. One theory on the pathogenesis of AD postulates that neurodegeneration is the result of oxidative stress and damage to vulnerable cerebral tissues.Scientists have known for some time that certain proteins accumulate in the brains of Alzheimers patients, leading to nerve cell damage. Exactly what causes the toxic plaques to form has not been established, but researchers positive that so-called free radicals highly reactive, naturally occurring molecules that damage cellular structures play a role. If so, it would stand to reason that antioxidants, which have the ability to bind and inactivate these destructive radicals, can combat the plaques. Under normal circumstances, the brain is protected from such damage by a careful balance between pro-oxidant and antioxidant mechanisms which include antioxidant enzymes A diet rich in certain antioxidants, such as

vitamins C and E, may help prevent Alzheimers disease, according to the results of new studies.[12]

In the present study, extract of Sememecarpus anacardium(25,50 and 75 mg/kg) improved learning and memory of mice significantly in interoceptive behavioral models employed. The simultaneous analysis or a distinction between reference and working memory is well established through ORT, MWM and PAT. Scopolamine a nonselective muscarinic antagonist blocks cholinergic signaling and produce memory deficit that are similar to those found in age related senile CNS dysfunction. The impairment of memory in scopolamine induced animal model is also associated with altered status of brain oxidative stress.[12] Scopolamine interferes with memory and cognitive function and subsequently causes impairment of reference (long term) and working (short term) memories. Mice were given scopolamine to induce memory impairment at a dose of 1 mg/kg.

In the present study, the effect of Semecarpus anacardium (25, 50, 75 mg/kg) was examined on the performance of mice in an object recognition task that has been considered to be a pure working memory task. Mice are able to discriminate between a familiar object and new object 1 h. or less, but not 24 h. after the presentation of familiar task. The effect of Extract of Semecarpus anacardium (25, 50, 75 mg/kg) was investigated on the acquisition of the information and on the consolidation of memory that takes place shortly after the acquisition of the information and on the consolidation of memory that takes place shortly after the acquisition and on the restitution of the information. The result indicated that the mice spend more time in exploring a new object than a familiar object in the scopolamine treated grp., when pretreated with extract of Semecarpus anacardium. The Discriminition index decreased in the scopolamine treated group. Pretreatment with extract of Semecarpus anacardium significantly increased the DI when compared with respective control. 50 mg/kg and 75 mg/kg of Semecarpus anacardium extract was found to be effective to enhance memory task.

Thus result demonstrates that improved retention in mice subjected to object recognition task in the scopolamine treated group. Extract of Semecarpus anacardium improves the consolidation and possibly the acquisition phase of working memory that is altered in interoceptive memory deficit models, i.e. scopolamine treated group. Piracetam(200mg/kg) established nootropic agents used as a standard in the present study also significantly improved the DI.

The ameliorative effects of extract of Semecarpus anacardium on learning and memory were investigated in the passive avoidance task. Scopolamine treated mice significantly shorter step down latencies. Extract of Semecarpus anacardium (25,50,and 75 mg/kg) treatment showed a significant increase in SDL in mice. Pretreatment with extract of semecarpus anacardium significantly decreased SDL in the scopolamine treated group. Piracetam used as the positive control also increased the step down latency. 25, 50 and 75 mg/kg dose of S.Anacardium was found to be effective to reverse the amnesia induced scopolamine.

The simultaneous analysis for a distinction between reference and working memory is well established through the Morris water maze test. In our experiments, normal control mice exhibited well formed reference memory and working memory. By contrast, mice given scopolamine exhibited neither reference nor working memory. Extracts of Semecarpus anacardium (25, 50, and 75 mg/kg) improved the amnesic deficits in reference memory and working memory. The prolongation in escape latency induced by scopolamine was significantly and gradually decreased over the four testing days. Mean escape latency and S.E.M. of extract on first day 67.833±0.703, 59.833±0.792, and 57 and on forth day, 51.833±0.307, 49.833±0.477, and 47.1666±0.4773 of dose 25, 50 and 75 mg/kg resp. This value indicates escape latency was decreased on successive days. Graph 6.4, 6.5,and 6.6 depicts a escape latency of SAE (25, 50, 75mg/kg) .and it shows a decrease in escape latency on a successive days.

The above behavioral results suggests that extract of Semecarpus anacardium has the ability to improve and ameliorate spatial long and working memory.

Many clinical studies have reported strong evidence that oxidative stress is involved in the pathogenesis of AD. Oxidative stress refers to the undue oxidation of biomolecules leading to cellular damage, and it occurs by reactive oxygen species. The histopathological and experimental evidence support the impact of oxidation on the pathogenesis of Alzheimer's a disease Cell in the brains of AD patients exhibit abnormally high amounts of oxidatively modified proteins, lipids and DNA; such free radicall-mediated molecular damage is particularly prominent in the environment of senile plaques and in neurofibrillary tangle bearing neurons, suggesting roles for ROS in amyloid mediated neuronal damage and neurofibrillary pathologies. Several sources of ROS in AD have been proposed, with amyloid beta protein and redox –active metals such as Fe^{2+} or Cu^{+} being two such sources.

The drugs with antioxidant effects might be beneficial for processing brain function. Antioxidant enzymes such as superoxide dismutase (SOD), glutathione peroxidase and catalase as well as glutathione reductase and ascorbate are involved in the reduction of oxidative stress. Antioxidant enzymes display the reduced activities in the affected brain region of patients of Alzheimer's disese, moreover the reduction in the level of intracellular oxidized protein under these conditions has been associated with the improvement of cognitive and or psychomotor functions. Augmentation of endogenous antioxidants by therapeutic substance has recently evoked scientific interest because any such property of a therapeutic agent can be expected to cause significant improvement in the endogenous defense against oxidative stress. These agent also reduce the oxidative damage and promote a functional recovery in neurodegenerative disorder. In the course of searching natural products with memory enhancing activity using scopolamine induced amnesic mouse as an experimental model for Alzheimer's disease. It was found that extract of Semecarpus anacardium showed a significant memory enhancing activity in ORT, MWM and PAT.

The observed beneficial effects of extracts of semecarpus anacardium may be attributed to it's diversified chemical components namely Flavonoids, Alkaloids, and Phenolic compounds like tannins,. There are evidences which shows that flavonoids, alkaloids and tannins exhibit potential antioxidant property and free radical scavenging activity. Phytochemical screening of S. Anacardium extract showed presence of flavonoid, tannins, and alkaloids. Thus S. Anacardium plant may exhibit a antioxidant property. Acetylcholine is neurotransmitter involved in memory and cognitive function. There are also evidences found about nuts of this plant that it showed a anticholinesterase activity, so may be chances a stem bark also pussses a anticholinesterase activity. Superoxide dismutase and glutathione peroxidase are the endogenous antioxidant which decreases the oxidative stress. There is a evidences plant may exhibit a neuroprotective action and may help in the the release of endogenous antioxidant.

Conclusion:

From the result of present investigation, it can be concluded that stem bark of Semecarpus anacrdium enhances the cognitive activity of brain.

It could improve the short term memory and long term memory.

Antioxidant, anticholinesterase and neuroprotective role could be responsible for a cognitive enhancing effect.

Hence, Semecarpus anacardium may be useful in the treatment or prevention of various cognitive disorders.

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