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Research

Phytochemical screening and pharmacological evaluation of anti-ulcer activity of asparagus racemosus willd

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Abstract

The cause of ulceration in patients is mainly due to hyper secretion of gastric juice and also due to hyper secretion of pepsin. In traditional system of medicine a number of herbal preparations have been used for the treatment of peptic ulcers. There are various medicinal plants has been used for the treatment of gastrointestinal disorders. In view of this, in present study we have to evaluate the anti-ulcer activity of *Asparagus racemosus*. Study was carried out, by using three methods i.e., alcohol, paracetamol and stress induced ulcers in rats pretreated with the doses of 250 mg/kg AQAR and ALAR, 10mg/kg Omeprazole and 50 mg/kg Ranitidine.

To evaluate the antiulcer activity of aqueous and alcoholic extracts of *Asparagus racemosus* leaves (AQAR and ALAR) at 250 doses using different experimentally induced gastric ulcer models in rats.

Gastric ulcers were induced in rats by 80% alcohol, paracetamol and forced immersion stress induced methods. In alcohol induced ulcer model, paracetamol induced ulcer model and stress induced model the ulcer index was determined. Where as in stress induced ulcers stress plays an important role in ulcerogenesis.

In alcohol-induced ulcers, AQAR and ALAR were effective in reducing lesion index and increasing the gastric mucus content. It was also effective in decreasing ulcer index in paracetamol-induced ulcers. All the results obtained with *Asparagus racemosus* were dose dependent. The results suggest that AQAR and ALAR possesses significant and dose dependent antiulcer activity. The antiulcer activity of AQAR and ALAR can be attributed to its cytoprotective and antisecretory action.

Keywords: *Asparagus racemosus*, antisecretory, cytoprotective, gastric ulcer, alcohol induced ulcers, paracetamol-induced ulcers and stress induced ulcers.

INTRODUCTION

Peptic ulcer and other acidic symptom affect up to ten percentages of the humans with sufficient severity to prompt victims to seek medical attention. The more significant disease condition requiring medical fuscous is ulcer and gastro esophageal disease. In the US, approximately 4 million people have peptic ulcer

(duodenal and gastric types), and 350 thousand new patient are diagnosed in each year, around 180 thousand peoples are admitted to hospital and treated with drugs yearly, and about five thousand patient from this case die each year as a result of ulcer condition. The lifetime of human being developing a peptic ulcer is about 10 percentages for Americans males and four percentages for female population.

Peptic ulcers is wound in the lesions that are most often affected in younger to older adults population, but this may diagnosed in young adult life. They often appear without obvious sign and symptom, after a period of days to months of active phase of disease, it may heal with or without drug treatment. It also affects because of bacterial infections with H. Pylori.

Danger of ulcer

Bleeding: Upper gastrointestinal (UGI) bleeding is the secondary common medical condition that effect high mortality in peptic ulcer. UGI bleeding commonly present along with hematemesis (vomiting with digested food and blood or coffee-ground like substance) and black, tarry stools (melana). Clinical diagnosis of UGI done by nasogastric tube lavage shows blood or coffee-ground like material presence. However, this diagnosis may be negative when the bleeding arises beyond a closed pylorus region. Most of the patient's having bleeding ulcers can be treated with fluid and blood resuscitation, drug therapy, and endoscopic surgery.

Perforation: This ulcer may be spread to small intestine, oesophagus and large intestine ulcers account for 60, 20 and 20 percent of perforations.

Penetration: Ulcer penetration called due to the permeation of the ulcer among the bowel part without free perforation and filtration of whole contents inside the peritoneal cavity. Surgical treatment regimen recommended that permeation affect in twenty percentage of ulcers, but little proportion of penetrating ulcers become clinically important. The common symptom these complications include acidic irritation, weight reduction and diarrhea: watery vomiting is an uncommon, but diagnostic symptom. No evident clinical data is available in the treatment regimen and guidance for the curing of penetrating ulcers.

Obstruction: Gastric wall obstruction among the frequent ulcer symptoms. Most of the cases are related with duodenal or pyloric part ulceration is 5 percent of the patient populations. Changes in lifestyle and dietary:

Aspirin and related drugs (non-steroidal anti-inflammatory drugs), alcohol, coffee (even decaf)9 and tea can interfere with the curing of the peptic ulcers. Smoking may also low the ulcer healing process. People with ulcer symptom have been evaluated to had more carbohydrate than people with no ulcers, from this route may occur with a genetic susceptibility for the ulcer pathogenies.

Sugar has also been reported to increase stomach pH14. Salt may cause the stomach and intestine irritation. Large uptakes of salt have been linked to higher risk of stomach ulcer. One of the amino acids Known as Glutamine, is the important source in the energy in cells which cover the stomach and intestine. It is also preventing the stress ulcer related by large burns of the preliminary study about the pathogenesis of ulcers.

Digestive system

The function of the digestive system is to digest and absorb food. It consists of a tubular gastrointestinal tract and accessory organs that aid in digestion and absorption.

All organisms require food to sustain life. The cells of the body require nutrients for the chemical reactions of enzyme synthesis, cell division, growth and repair and also for the production of heat energy. Most of the food we eat requires considerable processing before it can be used by the cells. It must be broken down mechanically and chemically before it is transported by the blood to the cells.

The activities that are performed by the digestive system include the following activities: Ingestion: the taking of food into the mouth.

Mastication: chewing food which pulverizes it and mixes it with saliva

Deglutination: Swallowing; moving food from the mouth to the pharynx and into the esophagus.

Digestion: The mechanical and chemical breakdown of food to prepare it for absorption.

Absorption: the passage molecules of food through the mucous membrane of the small intestine and into the blood and lymph for distribution to the cells.

Peristalsis: the rhythmic wavelike contractions of the smooth muscle of the intestines that¬ move food through the GI tract.

Defecation

The discharge of indigestible wastes (feces) from the GI tract. Anatomically and functionally the digestive system can be divided into a tubular gastrointestinal (GI) tract and accessory digestive organs. The GI tract which extends from the mouth to the anus is a continuous tube approximately 30 feet (9m) long. It goes through the thoracic cavity and enters the abdominal cavity through the diaphragm. The organs of the digestive system include the oral cavity (mouth), pharynx, esophagus, stomach, small intestine and large intestine. The accessory organs include teeth, salivary glands, liver, gall bladder and pancreas. It usually takes about 24-48 hours for food to travel the length of the GI tract. Food travels in an assembly line manner through the tract

where it is broken down to the molecular level and transported to the cells. Each region of the GI tract has a specific function in the process.

MATERIALS AND METHODS

The designing of methodology involves a series of steps taken in a systematic way in order to achieve the set goal(s) under the prescribed guidelines and recommendations. It includes in it all the steps from field trip to the observation including selection and collection of the medicinal plant, selection of dose value, standardization of protocol, usage of instruments, preparation of reagents, selection of specific solvents for extraction, formation of protocols and final execution of the standardized protocol. All this requires good build of mind and a good and soft technical hand to handle the materials and procedure in a true scientific manner.

Preliminary qualitative test
Preliminary Phytochemical Screening
Preliminary phytochemical screening of the plant extract was carried out for the analysis of Alkaloids, Carbohydrates, Tannins, Saponins, Steroids, Phenols, Flavonoids .as per the standard methods.

Detection of Alkaloids: Extracts were dissolved individually in dilute Hydrochloric acid and filtered.
a) Mayer's Test: Filtrates were treated with Mayer's reagent (Potassium Mercuric Iodide). Formation of a yellow-colored precipitate indicates the presence of alkaloids.
b). Wagner's Test: Filtrates were treated with Wagner's reagent (Iodine in Potassium Iodide). Formation of brown/reddish precipitate indicates the presence of alkaloids.
c). Dragendroff's Test: Filtrates were treated with Dragendroff's reagent (solution of Potassium Bismuth Iodide). Formation of red precipitate indicates the presence of alkaloids.
d). Hager's Test: Filtrates were treated with Hager's reagent (saturated picric acid solution). Presence of alkaloids confirmed by the formation of yellow coloured precipitate.

Detection of Carbohydrates: Extracts were dissolved individually in 5ml distilled water and filtered. The filtrates were used to test for the presence of carbohydrates.
a). Molisch's Test: Filtrates were treated with 2 drops of alcoholic α-naphthol solution in a test tube. Formation of the violet ring at the junction indicates the presence of Carbohydrates.
b). Benedict's Test: Filtrates were treated with Benedict's reagent and heated gently. Orange red precipitate indicates the presence of reducing sugars.
c). Fehling's Test: Filtrates were hydrolyzed with dil. HCl, neutralized with alkali and heated with Fehling's A&B solutions. Formation of red precipitate indicates the presence of reducing sugars.

Detection of saponins
a). Froth Test: Extracts were diluted with distilled water to 20ml and this was shaken in a graduated cylinder for15 minutes. Formation of 1cm layer off a am indicates the presence of saponins.
b). FoamTest:0.5gm of extract was shaken with 2ml of water. If foam produced persists forten minutes it indicates the presence of saponins.

Detection of steroids.
a). Salkowski's Test: Extracts were treated with chloroform and filtered. The filtrates were treated with few drops of Conc. Sulphuric acid, shaken and allowed to stand. Appearance of golden yellow colour indicates the presence of triterpenes.
b). Libermann Burchard's test: Extracts were treated with chloroform and filtered. The filtrates were treated with few drops of acetic anhydride, boiled and cooled. Conc. Sulphuric acid was added. Formation of brown ring at the junction indicates the presence of phytosterols.

Detection of Phenols
Ferric Chloride Test: Extracts were treated with 3-4 drops of ferric chloride solution. Formation of bluish black colour indicates the presence of phenols.

Detection of Tannins
Gelatin Test: To the extract,1 %gelatin solution containing sodium chloride was added. Formation of white precipitate indicates the presence of tannins.

Detection of Flavonoids

Alkaline Reagent Test: Extracts were treated with few drops of sodium hydroxide solution. Formation of intense yellow colour, which becomes colourless on addition of dilute acid, indicates the presence of flavonoids.

Lead acetate Test: Extracts were treated with few drops of lead acetate solution. Formation of yellow colour precipitate indicates the presence of flavonoids.

Experimental animals

Wistar rats (150-200 g) and were procured from Animals were housed in appropriate cages in uniform hygienic conditions and fed with standard pellet diet (Amrul Laboratory Animal Diet) and water ad libitum. All the animals were maintained under standard conditions, that is room temperature 26 ± 1°C, relative humidity 45 - 55% and12:12 h light – dark cycle. The animals were housed in large spacious hygienic cages during the course of the experimental period. Animal studies had approval of IAEC.

Plant Material Collection

The leaf of *Asparagus racemosus* was collected from the Botanical Garden and was identified and authenticated from Department. The plant material was cleaned, reduced to small fragments, air dried under shade at room temperature and coarsely powdered in a mixer. The powdered material was stored or taken up for extraction process.

Preparation of plant extracts

Preparation of Aqueous Extract

Fresh leaves of *Asparagus racemosus* were collected and washed under tap water. The leaves extract used was prepared by taking 20gms of finely cut leaves into 250ml beaker containing 200ml of water. The contents were mixed well and then the mixture was boiled up to 80-100^{0}C for 4-5hrs. Further the extract was filtered with whatmann filter paper. The filtrate was boiled until the concentrated residue is formed. The concentrated product was sealed in sample covers and stored under room temperature and used for further experiment to check the activities.

Preparation of Alcoholic Extract

Fresh leaves of *Asparagus racemosus* were collected and washed under tap water. The leaves extract used was prepared by taking 20gms of finely cut leaves into 250ml beaker containing 200ml of alcohol. The contents were mixed well and then the mixture was boiled up to 50-60^{0}C for 4-5hrs. Further the extract was filtered with whatmann filter paper. The filtrate was boiled until the concentrated residue is formed. The concentrated product was sealed in sample covers and stored under room temperature and used for further experiment to check the activities.

Selection of dose for animal study

The dose considered for the experiment on rats was obtained from conversion of human dose of *Asparagus racemosus* (3-5 g/kg). The conversion factor of human dose (per 200 g body weight) is 0.018 for rats. Hence the calculated dose for the rats (considering human dose 3 and 5 g/kg) is 200 mg/kg. Acute toxicity was done at dose of 2000mg/kg body weight.

Pharmacological evaluation

Preparation of extracts:

The aqueous and alcoholic extracts of *Asparagus racemosus* suspended in water in presence of 3%v/v Tween-80 solution.

All the drugs were administered orally for experimental purpose. Each time preparations of the extracts were prepared when required. The drugs were administered at a constant volume of 10ml/kg for each animal.

Acute oral toxicity

The acute oral toxicity of aqueous and alcoholic extracts of *Asparagus racemosus* was determined by using rats which were maintained under standard conditions. The animals were fasted 12 hours prior to the experiment, up and down procedure OECD guideline no. 425 were adopted for toxicity studies. Animals were administered with single dose of individual extract up to 2000mg/kg and observed for its mortality during 7days and 21days study period (long term) toxicity and observed up to 7days for their mortality, behavioral and neurological profiles.

Screening for anti-ulcer activity

The aqueous and alcoholic extracts of *Asparagus racemosus* leaves were tested for antiulcer activity using various methods like Acetic acid induced, alcohol induced, paracetamol induced and pyloric ligation method.

Acute stress-induced ulcer

The rats were deprived of food for 24 h, although water was allowed. Albino rats weighing between 160 - 180 g were divided into 12 groups consisting of six animals each. Experimental design and dosing schedule was as follows.

Animals were divided into four (I-V) groups.

Group I - Control group received distilled water (1ml, p.o).

Group II- Ulcer control

Group III - Standard group received Cimetidine (32mg/kg i.p).

Group IV - Test group received aqueous extract of *Asparagus racemosus* 250mg/kg p.o).

Group V - Test group received alcoholic extract of *Asparagus racemosus* (250mg/kg p.o).

Immediately after each procedure, the animals were killed and their stomachs removed, opened, and the inner lining examined. The gastric lesions were counted, and an ulcerative index (UI) was calculated for each animal as follows:

$$UI = (n \text{ lesion I}) + (n \text{ lesion II})\ 2 + (n \text{ lesion III})\ 3$$

Where:

I = presence of edema, hyperemia and single, submucosal, punctiform hemorrages;

II = presence of submucosal, hemorrhagic lesions with small erosions;

III = presence of deep ulcer with erosions and invasive lesions.

Acute, gastric lesions were induced by stress according to the model. After oral administration of 0.9% Nacl, Cimetidine and different doses of *Asparagus racemosus* extract, each rat was immobilized in a cylindrical cage and vertically immersed in water to the level of the xiphoid process for 17 h at 23°-25°C. After this, the animals were immediately killed, their stomachs removed, and the gastric lesions were counted.

Alcohol Induced Ulcers in Rats

Alcohol induced ulcer model, in rats was studied for all extractives of both plants to determine the ulcer index and ulcer inhibition. Albino rats weighing between 160 - 180 g were divided into 12 groups consisting of six animals each. Experimental design and dosing schedule was as follows.

Animals were divided into four (I-V) groups.

Group I - Control group received distilled water (1ml, p.o).

Group II- Ulcer control

Group III - Standard group received Omeprazole for seven days (2mg/kg i.p).

Group IV - Test group received aqueous extract of *Asparagus racemosus* (250mg/kg p.o) for seven days.

Group V - Test group received alcoholic extract of *Asparagus racemosus* (250mg/kg p.o) for seven days.

On the final day of dosing, the animals also received extractives and the standard drug thirty minutes before administration of 1ml of ethanol. Animals were sacrificed after one hour and the contents of the gastric juice in the stomach were aspirated. Later the stomachs were removed and kept immersed in saline for 5 min. Incisions of the stomach were performed along the greater curvature and linear haemorrhagic lesions in the glandular regions were observed. A pair of dividers was used to measure the length of all the lesions in the stomachs. The length (mm) of each lesion was determined at 10 x magnification and summed up per stomach. Ulcer index was the sum of length of all lesions for each stomach. Stomachs were immersed in 10% formalin for 24 h to study the histopathological changes in treated and ulcerated rats. Photographs of the opened stomachs were taken. The percentage ulcer inhibition was calculated by the following formula and the results were tabulated.

$$\% \text{ Ulcer protection} = \frac{\text{Ulcer Index in Control} - \text{Ulcer index in Test}}{\text{Ulcer Index in Control}} \times 100$$

Histopathological Evaluation of Alcohol induced Ulcers

The stomachs of the all groups of animals were immersed in 10% formalin to study the histopathological changes. After the standard processing the wet ulcerated tissues were embedded in paraffin and cut into thick sections. Parameters used to study histopathological changes included shedding of gastric epithelium, gastric erosions, infiltration of neutrophils, edema and inflammation.

Alcohol induced ulcer model was carried out with the different extractives of *Asparagus racemosus* based on the previous protocol to select the extractives with anti-ulcer activity for further evaluation on other anti-ulcer models.

Paracetamol Induced Modified Pylorus Ligated Model

The selected extractives of both plants were subjected to anti-ulcer studies using Aspirin induced model. Adult Wistar albino rats of either sex weighing 180-250 g were fasted for 48h with free access to water and divided into six groups of six animals each. They were placed in cages with grating floor to avoid coprophagy. The experimental design and dosing schedule was carried out as follows.

Group I: Normal control
Group II: Ulcer control (Solvent) (10 ml/kg) + Paracetamol (200 mg/kg)
Group III: Ranitidine (50 mg/kg)
Group IV: AQAR (250 mg/kg)
Group V: ALAR (250 mg/kg)

In Paracetamol induced ulcer model, one hour before pyloric ligation, aspirin at a dose of 200 mg/kg was administered orally as a suspension in 0.1% CMC. The animals were orally treated with the extractives at doses of 100 and 200 mg/kg once daily for seven days and 1 hour before administration of aspirin. The standard group of animals was also treated in the same way.

Pyloric ligations were performed under ether anaesthesia taking care to avoid damage to the pylorus and the blood vessels. After ligation the stomachs were replaced and abdominal wall sutured. Food and water was restricted during the post-operative period of 4 h. The animals were sacrificed at the end of four hours using excess ether anaesthesia. Thereafter the stomachs were opened and the contents of the gastric juice were collected. The contents were centrifuged and various biochemical estimations were carried out in the collected samples of control and treated groups of animals. The stomach samples were soaked in saline for five minutes and fixed to boards for morphological examinations of ulcer indices. Photographs were taken for further reference.

Evaluation of Ulcer Index and Inhibition

The ulcer index was calculated by counting the lesions with the aid of hand lens (10 X) and graded as follows.

0 = Normal coloured stomach
0.5 = Red colouration
1 = Spot ulcer
1.5 = Haemorrhagic streaks
2.0 = ulcers > 3 but < 5
3.0 = ulcers > 5

Mean ulcer score for each animal was expressed as ulcer index. Ulcer protection was calculated according to the standard formula.

$$\% \text{ Ulcer protection} = \frac{\text{Ulcer Index in Control} - \text{Ulcer index in Test}}{\text{Ulcer Index in Control}} \times 100$$

The volume and pH of the collected gastric juice was recorded. Free acidity and total acidity were calculated. Various bio-chemical estimations like total proteins, total hexoses, hexosamine, fucose, sialic acid, total carbohydrate and carbohydrate/protein ratio of the gastric juice were performed using standard methods.

Acetic Acid Induced Ulcer Model in Rats

The selected plant extractives of both plants were subjected to Acetic acid induced Chronic Ulcer Model in rats. Adult Wistar albino rats weighing 160-220 g of both sexes were selected. Five groups of ten animals each were formed with these rats. To avoid coprophagy they were placed in cages with grating floor. The rats were fasted for 24 h, but allowed free access of water.

Group I - Control (Non-ulcerated) (10 ml/kg, 0.1% CMC *p.o.*)
Group II - Solvent control (Ulcerated) (10 ml/kg, 0.1% CMC *p.o.*)
Group III - Animals received Omeprazole (10 mg/kg *p.o.*)
Group IV - Animals received AQAR (250 mg/kg *p.o.*)
Group V - Animals received ALAR (250 mg/kg *p.o.*)

Albino rats weighing 160-220 g were fasted for 24 h and abdomen was opened under light ether anaesthesia. A cylindrical plastic mould was placed near the region of the lesser curvature of the stomach and 50

µl of 50% glacial acetic acid was administered upon the wall of the stomach corpus. The stomach wall was wiped with cotton wool soaked in saline. Povidone iodine was applied to the abdominal stitches for the next few days to avoid infection. Thereafter the animals were fed with normal diet and access to water. Group I served as nonulcerated control and received only the vehicle while Group II served as the ulcerated control. Rats of Group III served as standard and was administered Omeprazole at 10 mg/kg while groups IV and V were treated with 250 mg/kg of AQAR and ALAR respectively as a suspension in 0.1% CMC. The treatment with the standard drug and the extractives were carried out for 21 days. During the remaining length of the experimental period of 21 days the amount of food and water consumed by the animals were noted. On the final day of the experiment, blood was withdrawn and the blood cell count of all group of animals were estimated by standard methods. The animals were sacrificed by excess anesthesia and Ulcer area of all groups of animals was calculated using the standard formula. Ulcer area was calculated as the product of length and width of ulcer (mm^2). The stomach samples of the treated and the control group of animals were stored in formalin for histopathological studies. The rate of healing of ulcers was calculated by comparing the ulcer index of extractives and Omeprazole treated rats with those of the ulcerated controls.

Statistical analysis

The values were expressed as mean ± SEM data was analyzed using one-way ANOVA followed by T-test. Two sets of comparison had made. i.e. Normal control Vs All treated groups. Differences between groups were considered significant at $P<0.001$ and $P < 0.05$ levels.

RESULTS

Phytochemical screening test

The freshly prepared extract of the leaves of *Asparagus racemosus* was subjected to phytochemical screening tests for the detection of various active constituents. The extract showed the presence of alkaloids, tannins, steroids, phenolic and flavonoids, carbohydrates, and glycosides in crude extract of *Asparagus racemosus* leaves.

Table 1: Result of chemical group tests of the Aqueous and Alcoholic Extract of *Asparagus racemosus leaves*.

Test	Aqueous Extract	Alcoholic Extract
Carbohydrates	-	+
Tannins	++	+
Flavonoid	+	++
Saponins	++	+
Phenols	++	+++
Steroids	-	+
Alkaloids	++	+
Glycosides	++	+

Aqueous and Alcoholic extract; (+): Present; (-): Absent; (+++); Reaction intensity is high; (++): Reaction intensity is medium; (+): Reaction intensity is normal;

Acute toxicity study

Administration of the *Asparagus racemosus* extracts *in* rats at doses of 250 mg/kg by oral gavage did not reveal any adverse effects or signs of toxicity.

Observations twice daily for fourteen days also did not reveal any drug related observable changes or mortality. Accordingly, the acute oral LD50 of the extractives was concluded to exceed 2000 mg/kg body weight, the highest dose tested in the study.

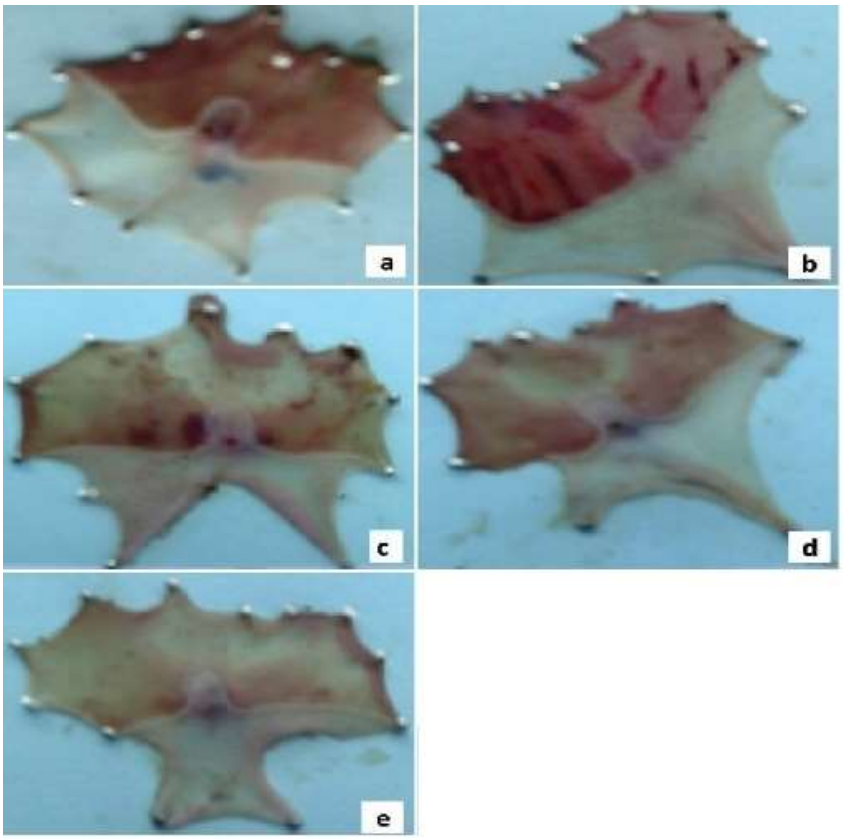
Effect on alcohol induced gastric ulcers

Oral administration of 80% alcohol produced haemorrhagic gastric lesions in glandular portion of stomach. Pretreatment with AQAR and ALAR at the dose of 250 mg/kg and Omeprazole (10 mg/ kg) significantly ($p<0.001$) protected the gastric mucosa as shown by reduced values of lesion index (18.3 ± 1.10 and 28.31± 2.43 respectively) against alcohol challenge as compared to solvent control (36.23±1.43).

Table 2: Effect of *Asparagus racemosus* at various doses on alcohol induced gastric ulcer in rats.

Treatment (n=6)	Dose mg/kg (p.o.)	Lesion index	% Inhibition of ulcer	Mucus content (μg Alcian blue/g wet tissue)
1% CMC	-	30.16 ± 0.47	-	0.50 ± 0.29
Ulcer control	-	36.23±1.43	-	0.61±0.13
Omeprazole	10	28.31± 2.43	21.05	0.70 ± 3.19
AQAR	250	33.15 ± 0.26	9.61	0.28 ± 1.14
ALAR	250	18.3 ± 1.10	40.43	0.849± 2.09

Values are mean ± S.E.M. n=number of animals in each group. Significant differences with respect to solvent control group were evaluated by Student's t – test. ($p<0.05$, $p<0.01$ and $p<0.001$).



(a) Normal Control (b) Ulcer Control (c) *AQAR* (250 mg/kg) treated (d) *ALAR* (250 mg/kg) treated (e) Omeprazole (10 mg/kg treated).

Fig 1: Effect of *Asparagus racemosus* on alcohol induced ulcers in the rats in the study

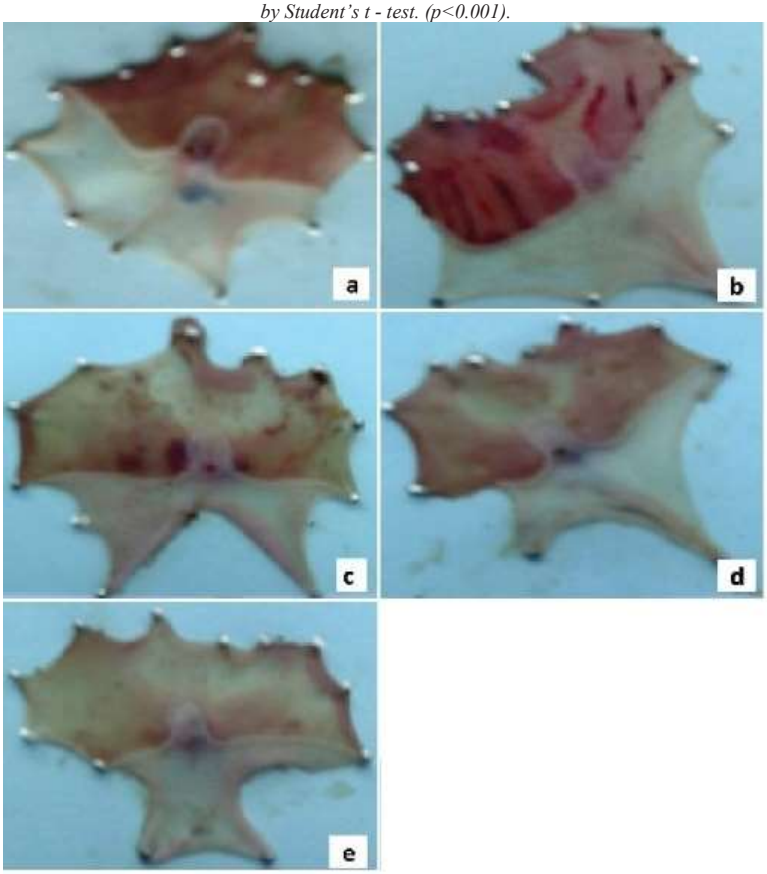
Effect on Paracetamol induced gastric ulcers

In *Asparagus racemosus* treated groups (250 mg/kg), the ulcer index values (0.48 ± 0.01 respectively) were significantly reduced ($p<0.001$) when compared to solvent control (0.73 ± 0.14), while the ulcer index for ranitidine treated group was 0.24 ± 1.12 ($p<0.001$). The %inhibition of ulcer showed by AQAR and ALAR (250mg/kg) and ranitidine was 53.0 %, 35.3% and 54.6 % respectively.

Table 3: Effect of *Asparagus racemosus* at various dose levels on paracetamol induced gastric ulcer in rats

Treatment(n=6)	Dose mg/kg(p.o.)	Ulcerindex	% Inhibitionof ulcer
1% CMC	-	0.73 ± 0.14	-
Ulcer control	-	0.81±0.34	--
Ranitidine	50	0.24 ± 1.12	53.0
AQAR	250	0.48 ± 0.01	35.3
ALAR	250	0.33 ± 0.06	54.6

Values are mean ± S.E.M. n=number of animals in each group; Significant differences with respect to solvent control group were evaluated by Student's t - test. (p<0.001).



(a) Normal Control (b) Ulcer Control (c) AQAR *(250 mg/kg) treated (d)* ALAR *(250 mg/kg) treated (e) Ranitidine (50 mg/kg treated)*

Fig 2: Effect of *Asparagus racemosus* on paracetamol induced ulcers in the rats in the study

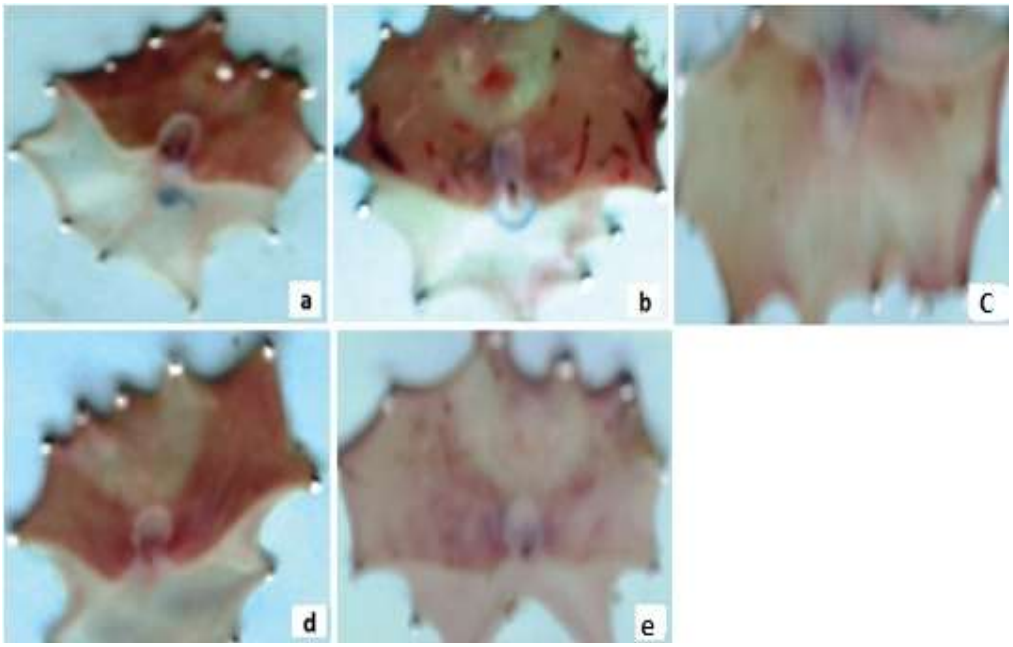
Stress-induced ulcers

In water immersion stress induced ulcers, the mean score value of ulcer inhibition was found to be significant (P<0.001) for 250 mg/kg of the extract. The percentage ulcer inhibition was 78.43 and 84.41 for 250 mg/kg for both aqueous and alcoholic extracts, and that of the standard was found to be 89.31.

Table 4: Effect of *Asparagus racemosus* at various dose levels on Stress induced gastric ulcer in rats.

Group	Dose mg/kg (p.o.)	Ulcer index	Percentage inhibition
Normal Control	-	00.00±0.00	-----
Ulcer control	-	22.32±1.42	------
Standard	50	5.45±0.19	89.31
AQAR	250	6.62±0.23	78.43
ALAR	250	6.42±0.51	84.41

Values are mean ± S.E.M. n=number of animals in each group; Significant differences with respect to solvent control group were evaluated by Student's t - test. (p<0.001).



(a) Normal Control (b) Ulcer Control (c) AQAR *(250 mg/kg) treated (d)* ALAR *(250 mg/kg) treated (e) Omeprazole (10mg/kg treated)*

Fig 3: Effect of *Asparagus racemosus* on stress induced ulcers in the rats in the study

CONCLUSION

The anti-ulcer activity of the plant *Asparagus racemosus* was evaluated by employing paracetamol, alcohol and stress induced ulcer models. These models represent some of the most common causes of gastric ulcer in humans. Many factors and mechanisms are implicated in the ulcerogenesis and gastric mucosal damage induced by different models employed in the present study involving, depletion of gastric wall, mucin mucosal damage induced by nonsteroidal anti-inflammatory drugs and free radical production.

NSAID's like aspirin and paracetamol causes gastric mucosal damage by decreasing prostaglandin levels through inhibition of PG synthesis. Alcohol and Aqueous extract of the plant of *Asparagus racemosus* was significantly effective in protecting gastric mucosa against paracetamol induced ulcers at all the dose level studied.

Alcohol induced gastric injury is associated with significant production of oxygen free radicals leading to increased lipid peroxidation, which causes damage to cell and cell membrane. The extracts of the *Asparagus racemosus* has significantly protected the gastric mucosa against alcohol challenge as shown by reduced values of lesion index as compared to control group suggesting its potent cytoprotective effect. It has been proposed that in pyloric ligation, the digestive effect of accumulated gastric juice and interference of gastric blood circulation are responsible for induction of ulceration.

The antiulcer activity of *Asparagus racemosus* extracts in stress induced model is evident from its significant reduction in gastric volume, ulcer index and increase in pH of gastric juice. Because of animals treated with *Asparagus racemosus* extracts significantly inhibited the formation of ulcer in the stomach and also decreased both acid concentration, gastric volume and increased the pH values.

It is suggested that *Asparagus racemosus* extracts can suppress gastric damage induced by aggressive factors. It is generally accepted that gastric ulcers result from an imbalance between aggressive factors and the maintenance of the mucosal integrity through endogenous defence mechanisms. The excess gastric acid formation by prostaglandin (PG) includes both increases in mucosal resistance as well as a decrease in aggressive factors, mainly acid and pepsin. Inhibitions of PG synthesis by aspirin coincide with the earlier stages of damage to the cell membrane of mucosal, parietal and endothelial cells.

The preliminary phytochemical studies revealed the presence of flavonoids in aqueous and alcoholic extracts of *Asparagus racemosus* various flavonoids have been reported for its anti-ulcerogenic activity with good level of gastric protection. So the possible mechanism of antiulcer action of *Asparagus racemosus* may be due to its flavonoid content. In this study we observed that *Asparagus racemosus* provides significant anti-ulcer activity against gastric ulcers in rats.

On the basis of the present results and available reports, it can be concluded that the anti-ulcer activity elucidated by *Asparagus racemosus* could be mainly due to the modulation of defensive factors through an improvement of gastric cytoprotection and partly due to acid inhibition.

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