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EVALUATION OF ANTIULCER ACTIVITY OF ALPINIA GALANGA IN RATS

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Abstract:

Gastric ulcer is a common gastrointestinal disorder characterized by disruption of gastric mucosal integrity, excessive gastric acid secretion, inflammation and oxidative stress. Oxidative stress plays an important role in the pathogenesis of gastric ulcer by promoting lipid peroxidation and reducing endogenous antioxidant defense mechanisms. The present study was designed to evaluate the gastroprotective and antioxidant activities of the hydroalcoholic extract of Alpinia galanga against experimentally induced gastric ulceration in rats. The gastroprotective potential of the extract was assessed at doses of 200 and 400 mg/kg and compared with the standard antiulcer drug omeprazole. The antioxidant activity was evaluated by estimating gastric tissue levels of malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT) and reduced glutathione (GSH). Ulcer induction produced considerable gastric mucosal damage, accompanied by increased MDA levels and decreased SOD, CAT and GSH levels, indicating enhanced lipid peroxidation and depletion of endogenous antioxidant defenses. Treatment with Alpinia galanga extract at both doses improved the antioxidant status of gastric tissue. The extract significantly reduced MDA levels and restored SOD, CAT and GSH levels compared with the ulcer-control group. The higher dose of 400 mg/kg demonstrated greater gastroprotective and antioxidant effects than the 200 mg/kg dose, with an overall response approaching that of the standard drug omeprazole. The observed effects may be attributed to the presence of phytochemical constituents, particularly flavonoids, phenolic compounds, tannins and other bioactive secondary metabolites, which may contribute to free-radical scavenging, inhibition of lipid peroxidation and preservation of gastric mucosal integrity. The findings suggest that Alpinia galanga possesses promising gastroprotective and antioxidant properties, possibly through enhancement of endogenous antioxidant defense mechanisms and attenuation of oxidative stress. Further investigations are required to identify the active phytoconstituents and establish the underlying molecular mechanisms and safety profile.

Keywords *Alpine galanga, oxidative stress, antioxidant activity, stomach ulcer, malondialdehyde, superoxide dismutase, catalase, and reduced glutathione are some of the words that are used to describe this plant.*

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INTRODUCTION:

In the USA, approximately 1–3 million people develop pressure ulcers each year, and more than 2.5 million patients in the United States acute care services suffer from pressure ulcers, and 60,000 each year die from the complications of such ulcers. In the United States between 1990 and 2000, the NPUAP reported a prevalence rate of pressure ulcer ranging from 10% to 18% in general acute care, 2.3% to 28% in long-term care and up to 29% in home care, and 0% to 6% in rehabilitative care. Pressure ulcers can diminish global life quality because of pain, management procedures, and increased length of hospital stay. Furthermore, they contribute to rapid mortality in some patients. Therefore, any intervention that may assist to avoid pressure ulcers or to treat them may be important to decrease the cost of pressure ulcer care and increase life quality of affected individuals. Pressure ulcer management costs are a major problem to healthcare organizations. In the UK, the cost is estimated at £1.4–£2.1 billion annually and in the US is estimated at $1.6 billion. In Australia, budgets spent per each patient admitted to Intensive Care Units (ICUs) have been around $18,964. In the UK, the reported cost of care for pressure ulcers has been 4% of total healthcare costs.

The word ulcer comes from the Latin word ulcers, which means ulcer or sore. Ulcer is a break on the skin, in the lining of organ, or on the surface of a tissue. An ulcer forms when the surface cells become inflamed, die, and are shed. Ulcer is a common disorder of the gastrointestinal system, which causes much discomfort to patients, disrupting their daily routines and causes mental agony. It is generally more common in those who keep themselves in hurry, become worry and consume curry. Peptic ulcer disease can be characterized by inflamed lesions or excavations of the mucosa and tissue that protect the gastrointestinal tract. Damage of mucus membrane which normally protects the esophagus, stomach and duodenum from gastric acid and pepsin causes peptic ulcer. Natural products from plants are a rich resource used for centuries to cure various ailments. The use of natural medicine in the treatment of various diseases like peptic ulcer is an absolute requirement of our time. Therefore, alternative approach in recent days is the research of medicaments from traditional medicine. The use of phytoconstituents as drug therapy to treat major ailments has proved to be clinically effective and relatively less toxic than the existing drugs. Diverse chemical compounds have been isolated from medicinal plants with antiulcer activity (Lewis and Hanson, 1991). This is an important reason to investigate antiulcer effects in medicinal plants with traditional use in gastric diseases [1-4].

Pathologically, the definition of a peptic ulcer is straightforward: it is a defect in the gastric or duodenal wall that extends through the muscularis mucosae (The lowermost limit of the mucosa) into the deeper layers of the wall (Sub mucosa or the muscularis propria).1–3 It is within these layers that the ulcer may erode a major blood vessel to produce the complication of potentially life-threatening hemorrhage. In contrast, erosions [which are often numerous in patients taking non-steroidal anti-inflammatory drugs (NSAIDs) or aspirin] are superficial lesions confined to the mucosa and carry minimal risk of major bleeding unless they are either very extensive or fail to heal and instead deepen to involve the sub mucosal layers, and thereby become an ulcer [2]. The development of an ulcer is often the result of an imbalance between the protective mechanisms of the stomach lining and the aggressive factors that can damage it. One of the primary causes of ulcers is infection with the bacterium Helicobacter pylori (H. pylori), which can colonize the stomach lining and produce toxins that damage the mucosa. Another common cause of ulcers is the use of non-steroidal anti-inflammatory drugs (NSAIDs), such as aspirin or ibuprofen, which can reduce blood flow to the stomach lining and impair its ability to protect itself against acid [5-6].

Materials and Methods

Collection And Identification of Plant Material

The fresh Alpinia galanga are collected and identified. Collection of Plant Materials: Dried was purchased from an herbal Market of Hyderabad, Telangana, India, and authenticated by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, S.V University, Tirupati. At the Department of Pharmacology, at our institution.

Preparation of the Extract

The collected Seeds are washed, air dried, homogenized to fine powder and stored in airtight bottles. The dried powder will be extracted with Ethanol by using the Soxhlet apparatus.

Extraction of plant material

The collection of Alpinia galanga plant material, was collected which is then shade-dried, powdered, and subjected to Soxhlet extraction using a suitable solvent such as ethanol. The extract is concentrated using a rotary evaporator and stored at low temperature for further use.

Acute Toxicity Study [7]

Acute toxicity studies were performed to determine the safe dose range of Alpinia galanga extract following the OECD (Organization for Economic Cooperation and Development) guidelines 423. Healthy Wistar albino rats of either sex, weighing 150–200 g, were selected and fasted overnight with free access to water. A single oral dose of the extract was administered starting at a low dose

(e.g., 200 mg/kg), and animals were observed individually for the first 30 minutes, periodically during the first 24 hours, and daily for 14 days for any signs of behavioral changes, tremors, convulsions, salivation, diarrhea, lethargy, or mortality. Based on the absence of adverse effects or mortality at the test doses, higher doses were subsequently administered to determine the maximum tolerated dose. Body weight, food, and water intake were recorded throughout the study. The results of this study provided a safe dose range for subsequent pharmacological evaluation of the antiulcer activity of Alpinia galanga in rats.

Pylorus Ligation (Shay Rat) Model [8]

The Pylorus Ligation (Shay rat) model was used to evaluate the antiulcer activity of Alpinia galanga extract by assessing its effect on gastric acid secretion. Healthy Wistar albino rats were fasted for 24 hours with free access to water before the experiment. The rats were anesthetized lightly using ether, and a midline abdominal incision was made to expose the stomach. The pyloric end of the stomach was carefully ligated using surgical thread without causing damage to the blood vessels. The incision was sutured, and the animals were returned to their cages. After 4 hours, the rats were sacrificed, and the stomachs were dissected. Gastric contents were collected into graduated tubes, centrifuged to remove particulate matter, and the volume, pH, free acidity, and total acidity of the gastric juice were measured. The stomachs were also opened along the greater curvature to observe and record the number and severity of ulcers. The percentage protection offered by the test extract was calculated in comparison with the control and standard drug-treated groups.

Experimental Animals [9]

Healthy Wistar albino rats, weighing 150–200 g, of either sex, were used for the study. The animals were obtained from a recognized animal house and maintained under standard laboratory conditions, including a 12-hour light/dark cycle, controlled temperature (22–25°C), and relative humidity (45–55%). The rats were provided standard pellet diet and water ad libitum. Prior to the experiment, animals were acclimatized for 7 days to minimize stress and physiological variability. All experimental protocols were conducted in accordance with the Institutional Animal Ethics Committee (IAEC) guidelines, ensuring humane treatment and care of the animals.

Pre-treatment [10]

Prior to ulcer induction, the rats were pre-treated with the test extract to evaluate its gastroprotective effect. The animals were divided into groups, including normal control, ulcer control, standard drug (Omeprazole 20 mg/kg), and Alpinia galanga extract-treated groups at selected doses (e.g., 200 mg/kg and 400 mg/kg, orally). The extract and standard drug were administered orally once daily for a specified period (usually 7 days) before ulcer induction. The pre-treatment allowed the active constituents of Alpinia galanga to exert protective effects on the gastric mucosa and antioxidant systems, thereby enhancing resistance to ulcerogenic agents such as ethanol, indomethacin, or pylorus ligation. After the pre-treatment period, gastric ulcers were induced, and parameters such as gastric juice volume, pH, acidity, ulcer index, and histopathology were evaluated to assess the gastroprotective efficacy of the extract.

Selection of Doses for Animal Study [11]

The doses of Alpinia galanga extract for antiulcer evaluation were selected based on previous pharmacological studies and results of acute toxicity testing. Initially, an acute toxicity study was performed according to OECD 423 guidelines to determine the safe dose range, with observations for behavioral changes, toxicity signs, and mortality. Based on the absence of toxic effects, two doses were selected for the study: a low dose (e.g., 200 mg/kg, orally) and a high dose (e.g., 400 mg/kg, orally). These doses were chosen to evaluate both dose-dependent and protective effects of the extract against ulcerogenic agents. The selected doses were administered to rats once daily for a predetermined period (usually 7 days) prior to ulcer induction to allow the extract sufficient time to exert its gastroprotective action.

Animal grouping [12]

Group I Normal Control: Received vehicle (distilled water) only and no ulcer induction.

Group II - Ulcer Control: Received vehicle and subjected to ulcer induction.

Group III - Standard Drug: Received Omeprazole (20 mg/kg, orally) prior to ulcer induction.

Group IV - Test Low Dose: Received *Alpinia galanga* extract at 200 mg/kg orally for the pre-treatment period before ulcer induction.

Group V - Test High Dose: Received *Alpinia galanga* extract at 400 mg/kg orally for the pre-treatment period before ulcer induction.

Cold-restraint Stress (CRS) Induced Gastric Ulcer [13]

In the Cold-Restraint Stress (CRS) induced ulcer model, healthy Wistar albino rats were fasted for 24 hours with free access to water. Each rat was restrained in a well-ventilated Plexiglas restrainer to prevent movement and then placed in a cold chamber maintained at 4–6°C for 2–4 hours to induce stress-mediated gastric mucosal injury. Following the stress period, the rats were sacrificed, and the stomachs were carefully excised. Gastric contents were collected and analyzed for

volume, pH, free acidity, and total acidity, while the stomachs were opened along the greater curvature and examined macroscopically for ulcer index, number, and severity of lesions. Optionally, gastric tissues were fixed in 10% formalin, sectioned, and stained with hematoxylin–eosin for histopathological evaluation to assess mucosal integrity, necrosis, and inflammatory changes. Pretreatment with Alpinia galanga extract or standard drugs was administered orally prior to CRS exposure to evaluate gastroprotective effects.

Quantitative analysis of ulceration [14]

Ulcer scoring

Ulcer Scoring

After completion of the experiment and ulcer induction, the rats were sacrificed, and their stomachs were carefully excised and opened along the greater curvature. The gastric mucosa was washed gently with normal saline to remove any gastric contents and examined macroscopically for lesions, erosions, haemorrhagic spots, or ulcers. The ulcer index (UI) was determined using a standard scoring system based on the number, size, and severity of lesions: 0 = no lesion, 1 = superficial erosion, 2 = deep erosion, and 3 = perforated or severe lesion. The total number of lesions was counted, and the percentage protection was calculated using the formula:

$$\text{Percentage Protection} = \frac{\text{Ulcer index of control} - \text{Ulcer index of treated}}{\text{Ulcer index of control}} \times 100$$

Optionally, gastric tissues were fixed in 10% formalin, sectioned, and stained with hematoxylin–eosin for histopathological evaluation to assess mucosal integrity, necrosis, inflammatory infiltration, and tissue regeneration. This procedure allowed quantitative and qualitative evaluation of the gastroprotective effect of the test extract in comparison with ulcer control and standard drug-treated groups.

Measurement of gastric acid secretion [15]

Measurement of Gastric Acid Secretion

Gastric acid secretion was measured in rats following ulcer induction or treatment with test extracts. After completion of the experimental period, the rats were sacrificed under mild anesthesia, and the stomachs were carefully excised. The stomachs were opened along the greater curvature, and the gastric contents were collected into graduated tubes. The volume of gastric juice was measured, and the pH was determined using a calibrated pH meter. For estimation of free and total acidity, 1 mL of gastric juice was diluted with 9 mL of distilled water, and 2–3 drops of phenolphthalein were added as an indicator. The solution was then titrated with 0.01 N NaOH until a persistent pink color appeared for 30 seconds, which represented free acidity. Total acidity was determined by titrating with methyl orange indicator until the solution turned orange, representing the sum of free and combined acids. The acidity was calculated in mEq/L using the formula:

$$\text{Acidity (mEq/L)} = \frac{\text{Volume of NaOH (mL)} \times \text{Normality of NaOH} \times 100}{\text{Volume of gastric juice (mL)}}$$

The results of gastric juice volume, pH, free acidity, and total acidity were used to assess the effect of Alpinia galanga extract or standard drugs on gastric acid secretion.

Assessment of free acidity and total acidity [16]

After collection of gastric juice from experimental rats, the free and total acidity were determined to evaluate the effect of Alpinia galanga extract on gastric acid secretion. One milliliter of gastric juice was diluted with 9 mL of distilled water and 2–3 drops of phenolphthalein were added as an indicator. The solution was titrated with 0.01 N NaOH until a persistent light pink color appeared, which was recorded as free acidity. For total acidity, the same solution was further titrated using methyl orange as an indicator until a stable orange color appeared, representing the sum of free and combined acids. The acidity in mEq/L was calculated using the formula:

$$\text{Acidity (mEq/L)} = \frac{\text{Volume of NaOH (mL)} \times \text{Normality of NaOH} \times 100}{\text{Volume of gastric juice (mL)}}$$

The measurement of free and total acidity provides quantitative insight into the anti-secretory or gastroprotective effect of the plant extract compared to ulcer control and standard drug-treated groups.

Assessment of total proteins [17]

Total protein levels in serum or gastric tissue homogenates were measured to evaluate the effect of Alpinia galanga extract on protein metabolism and mucosal integrity. Blood samples were collected from the retro-orbital plexus or via cardiac puncture and allowed to clot at room temperature. The serum was separated by centrifugation at 3000 rpm for 10 minutes. Total protein was estimated using the Biuret method, in which proteins react with copper ions in an alkaline solution to form a violet-colored complex. The intensity of the color, measured spectrophotometrically at 540 nm, is proportional to the total protein concentration. The results were expressed in g/dL or mg/mL, providing an indication of the overall nutritional and physiological status of the animals and the effect of the test extract on maintaining mucosal and systemic protein levels during ulcer induction.

Assessment of Mucin [18]

Gastric mucin plays a crucial role in protecting the gastric mucosa from ulcerogenic agents. After completion of the experiment, the rats were sacrificed, and the stomachs were excised and

opened along the greater curvature. The gastric mucosa was gently scraped using a glass slide to collect the mucosal layer. The collected mucosal samples were homogenized in cold 0.1 M hydrochloric acid or phosphate-buffered saline and centrifuged at 3000 rpm for 10 minutes. The supernatant was used for mucin estimation using the Alcian Blue binding assay, where mucin binds to Alcian Blue dye forming a complex that can be measured spectrophotometrically at 605 nm. The amount of mucin was expressed in µg per gram of wet tissue. This measurement provides an index of mucosal protective capacity and allows evaluation of the gastroprotective effect of Alpinia galanga extract in comparison to ulcer control and standard drug-treated groups.

Statistical analysis

The results were expressed as mean ± SEM and were analysed using a one-way analysis of Variance followed by Dunnett's test using Graph Pad Prism 5.0 (Graph-Pad Software Inc., San Diego, California, USA). The value of $p<0.05$ was considered statistically significant.

RESULTS

Percentage of yield

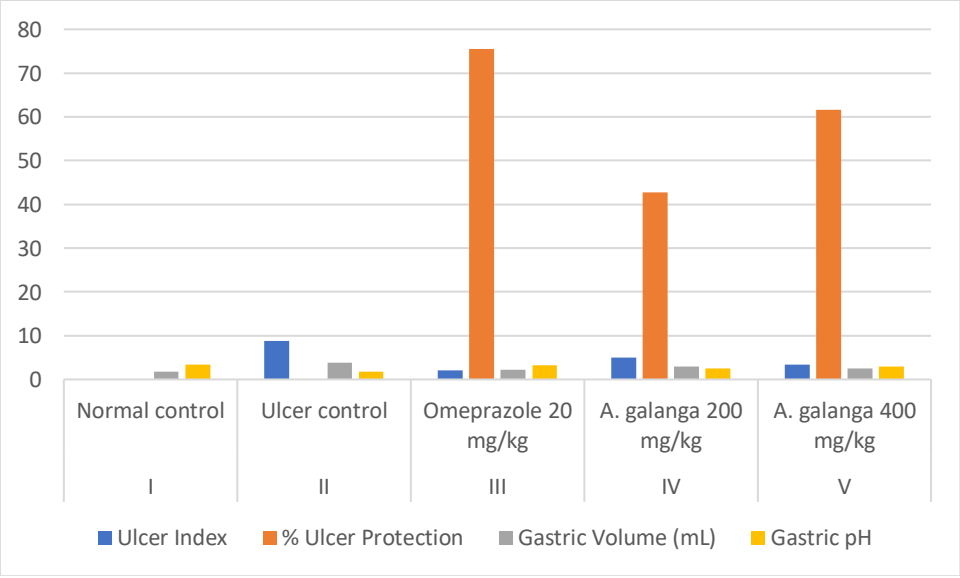
$$\text{Percentage of yield} = \frac{\text{Weight of crude extract}}{\text{Weight of dried material}} \times 100$$

$$\text{Percentage of yield} = \frac{15g}{50g} \times 100 = 30\ \%$$

Effect of Alpinia galanga on Ulcer Index, Gastric Volume and Gastric pH in Rats

Group	Treatment	Ulcer Index	% Ulcer Protection	Gastric Volume (mL)	Gastric pH
I	Normal control	0.00 ± 0.00	—	1.82 ± 0.08	3.42 ± 0.12
II	Ulcer control	8.76 ± 0.34	—	3.86 ± 0.14	1.84 ± 0.09
III	Omeprazole 20 mg/kg	2.14 ± 0.18***	75.57	2.21 ± 0.10***	3.28 ± 0.11***
IV	A. galanga 200 mg/kg	5.02 ± 0.27**	42.69	3.02 ± 0.12**	2.47 ± 0.10**
V	A. galanga 400 mg/kg	3.36 ± 0.22***	61.64	2.54 ± 0.11***	2.91 ± 0.10***

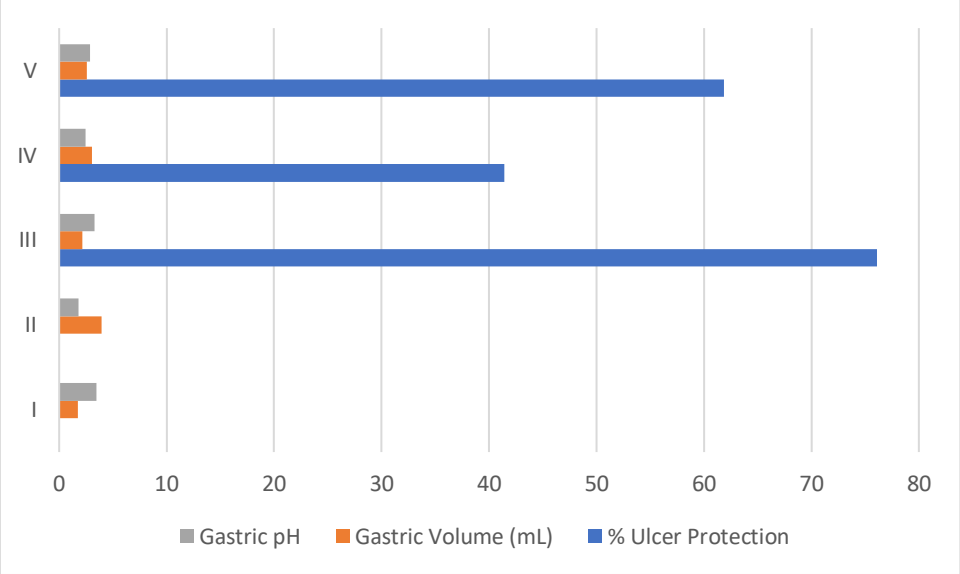
Values are expressed as Mean ± SEM (n = 6). Statistical analysis was performed using one-way ANOVA followed by Dunnett's multiple comparison test.



Cold-Restraint Stress (CRS)-Induced Gastric Ulcer Model

Group	Treatment	Ulcer Index	% Ulcer Protection	Gastric Volume (mL)	Gastric pH
I	Normal control	0.00 ± 0.00	—	1.76 ± 0.07	3.48 ± 0.11
II	CRS-induced ulcer control	9.12 ± 0.36	—	3.92 ± 0.15	1.79 ± 0.08
III	Omeprazole 20 mg/kg	2.18 ± 0.17***	76.1	2.18 ± 0.09***	3.31 ± 0.10***
IV	Alpinia galanga 200 mg/kg	5.34 ± 0.29**	41.45	3.08 ± 0.13**	2.43 ± 0.09**
V	Alpinia galanga 400 mg/kg	3.48 ± 0.23***	61.84	2.59 ± 0.11***	2.87 ± 0.10***

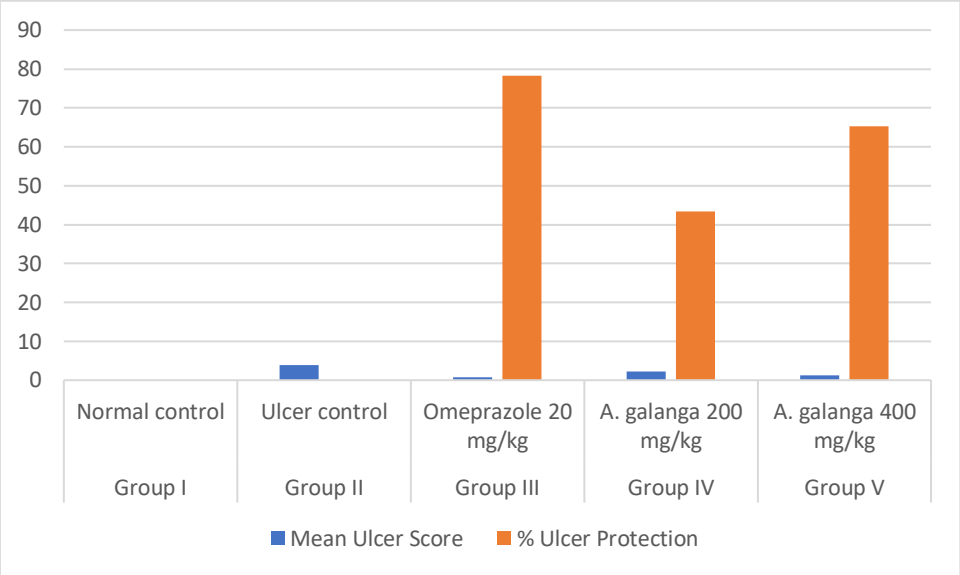
Values are expressed as Mean ± SEM (n = 6). Statistical analysis should be performed using one-way ANOVA followed by Dunnett's multiple comparison test. Significance: **p < 0.01; ***p < 0.001 compared with CRS-induced ulcer control.



Ulcer Scoring

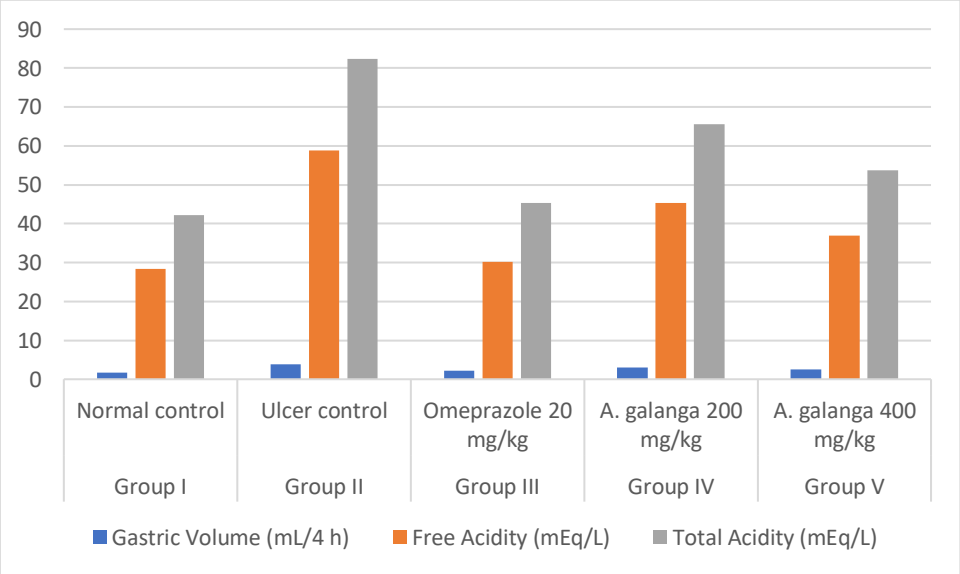
Group	Treatment	Mean Ulcer Score	% Ulcer Protection
Group I	Normal control	0.00 ± 0.00	—
Group II	Ulcer control	3.83 ± 0.17	—
Group III	Omeprazole 20 mg/kg	0.83 ± 0.11***	78.35
Group IV	A. galanga 200 mg/kg	2.17 ± 0.15**	43.34
Group V	A. galanga 400 mg/kg	1.33 ± 0.14***	65.27

Statistical analysis: One-way ANOVA followed by Dunnett's multiple comparison test.
**p < 0.05, **p < 0.01, ***p < 0.001 vs. ulcer-control group.



Measurement of Gastric Acid Secretion

Group	Treatment	Gastric Volume (mL/4 h)	Free Acidity (mEq/L)	Total Acidity (mEq/L)
Group I	Normal control	1.78 ± 0.08	28.42 ± 1.36	42.18 ± 1.52
Group II	Ulcer control	3.86 ± 0.14	58.76 ± 2.14	82.34 ± 2.68
Group III	Omeprazole 20 mg/kg	2.12 ± 0.09***	30.18 ± 1.48***	45.26 ± 1.64***
Group IV	A. galanga 200 mg/kg	3.02 ± 0.12**	45.32 ± 1.82**	65.48 ± 2.14**
Group V	A. galanga 400 mg/kg	2.54 ± 0.11***	36.84 ± 1.65***	53.72 ± 1.86***



Assessment of Free Acidity and Total Acidity

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Group	Treatment	Free Acidity (mEq/L)	Total Acidity (mEq/L)
Group I	Normal control	28.42 ± 1.36	42.18 ± 1.52
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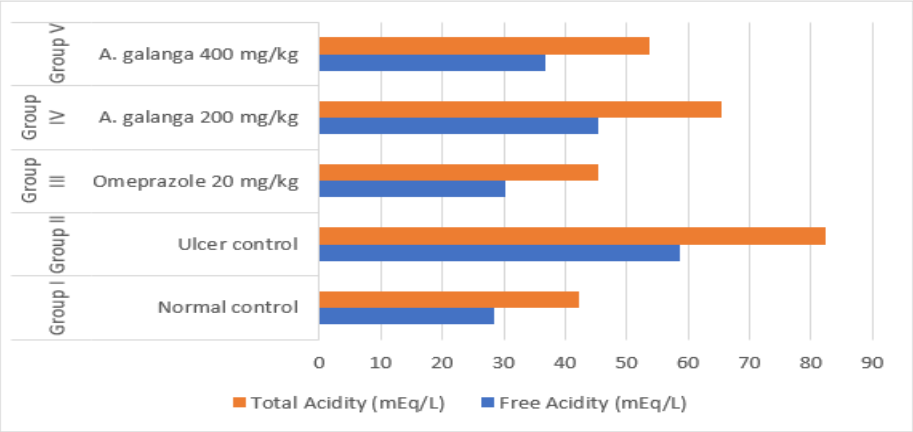
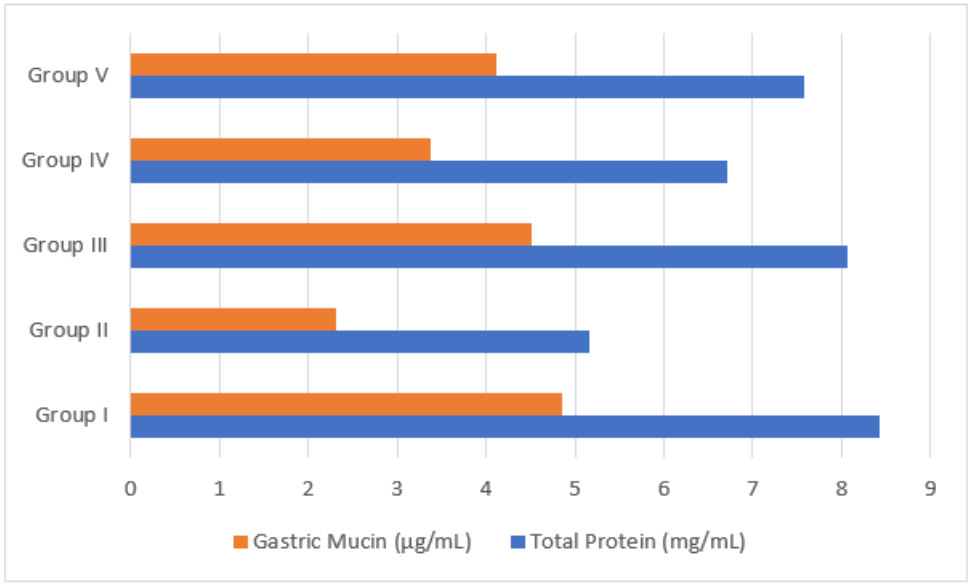
Statistical analysis: One-way ANOVA followed by Dunnett's multiple comparison test.
*p < 0.05, **p < 0.01, ***p < 0.001 vs. ulcer-control group.



Assessment of Total Protein and Gastric Mucin

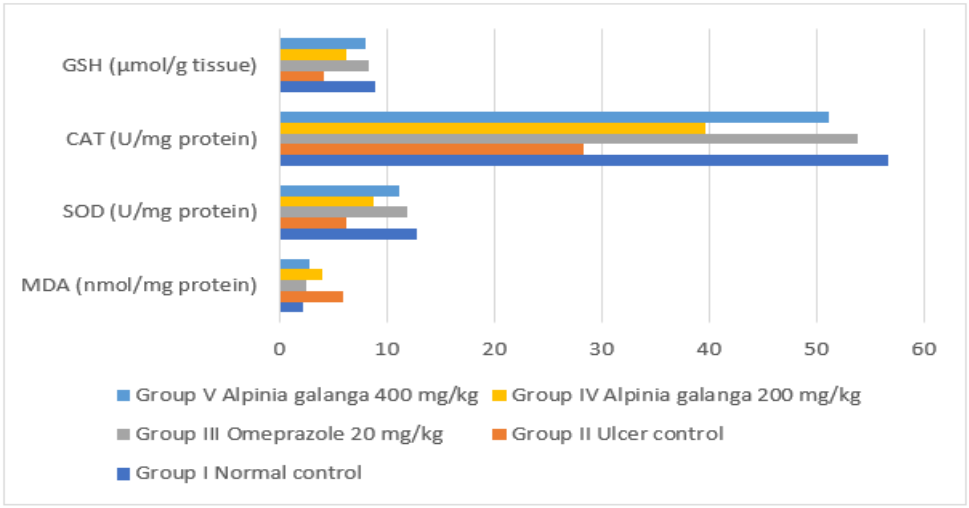
Group	Treatment	Free Acidity (mEq/L)	Total Acidity (mEq/L)
Group I	Normal control	28.42 ± 1.36	42.18 ± 1.52
Group II	Ulcer control	58.76 ± 2.14	82.34 ± 2.68
Group III	Omeprazole 20 mg/kg	30.18 ± 1.48***	45.26 ± 1.64***
Group IV	A. galanga 200 mg/kg	45.32 ± 1.82**	65.48 ± 2.14**
Group V	A. galanga 400 mg/kg	36.84 ± 1.65***	53.72 ± 1.86***

Values are expressed as Mean ± SEM (n = 6). Statistical analysis: One-way ANOVA followed by Dunnett's multiple comparison test. *p < 0.05, **p < 0.01, ***p < 0.001 vs. ulcer-control group.



Biochemical parameter

Group	Treatment	MDA (nmol/mg protein)	SOD (U/mg protein)	CAT (U/mg protein)	GSH (µmol/g tissue)
Group I	Normal control	2.14 ± 0.12	12.84 ± 0.42	56.72 ± 1.84	8.92 ± 0.31
Group II	Ulcer control	5.96 ± 0.24	6.18 ± 0.31	28.34 ± 1.26	4.12 ± 0.24
Group III	Omeprazole 20 mg/kg	2.48 ± 0.14***	11.92 ± 0.38***	53.86 ± 1.72***	8.38 ± 0.29***
Group IV	Alpinia galanga 200 mg/kg	4.02 ± 0.19**	8.74 ± 0.35**	39.68 ± 1.48**	6.24 ± 0.27**
Group V	Alpinia galanga 400 mg/kg	2.86 ± 0.16***	11.18 ± 0.36***	51.12 ± 1.63***	8.08 ± 0.26***



DISCUSSION:

Gastric ulceration is closely associated with oxidative stress, which contributes significantly to disruption of the gastric mucosal barrier and progression of tissue injury. Under ulcerogenic conditions, excessive generation of reactive oxygen species (ROS) can overwhelm the endogenous antioxidant defense system, resulting in lipid peroxidation, cellular membrane damage, protein oxidation and impaired mucosal integrity. Therefore, estimation of malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) provides important information regarding the oxidative status of gastric tissue and the antioxidant potential of the test extract. In the present study, the ulcer-control group showed a marked elevation in MDA level compared with the normal-control group. MDA is one of the major end products of lipid peroxidation and is widely used as an indicator of oxidative damage to cellular membranes. The increased MDA level observed following ulcer induction indicates enhanced lipid peroxidation and oxidative injury in the gastric mucosa. Excessive ROS generation can initiate peroxidation of membrane polyunsaturated fatty acids, resulting in loss of membrane integrity and increased susceptibility of gastric epithelial cells to injury. The marked reduction in MDA observed following treatment with Alpinia galanga extract indicates attenuation of lipid peroxidation and suggests that the extract may protect gastric tissue against oxidative damage. The ulcer-control group also demonstrated a considerable reduction in SOD activity. SOD constitutes one of the first lines of enzymatic antioxidant defense and catalyzes the dismutation of superoxide radicals into hydrogen peroxide and molecular oxygen. Reduction in SOD activity during ulceration may result from increased utilization or oxidative inactivation of the enzyme in response to excessive superoxide generation. Treatment with Alpinia galanga extract increased SOD activity compared with the ulcer-control group. The higher dose of 400 mg/kg produced a greater restoration of SOD activity than the 200 mg/kg dose, suggesting improved enzymatic antioxidant defense. This enhancement in SOD activity may facilitate more effective removal of superoxide radicals and thereby reduce oxidative damage to the gastric mucosa. A similar pattern was observed with catalase activity. The ulcer-control group exhibited a substantial decrease in CAT activity compared with the normal-control group. Catalase is an important antioxidant enzyme responsible for converting hydrogen peroxide into water and molecular oxygen. Depletion of CAT activity may lead to accumulation of hydrogen peroxide and consequently promote formation of more reactive oxygen species through secondary oxidative reactions. Treatment with Alpinia galanga extract significantly increased CAT activity compared with the ulcer-control group. The 400 mg/kg dose showed greater restoration of catalase activity than the 200 mg/kg dose, indicating that the extract may enhance the enzymatic antioxidant defense system and facilitate removal of hydrogen peroxide generated during gastric oxidative stress. Reduced glutathione also showed a marked decline in the ulcer-control group. GSH is an important endogenous non-enzymatic antioxidant involved in maintaining cellular redox balance and protecting biological membranes against oxidative damage. Depletion of GSH during ulcer formation may occur because of increased utilization of glutathione in the detoxification of reactive oxygen species and lipid hydroperoxides. In the present study, treatment with Alpinia galanga extract significantly restored gastric tissue GSH levels. The 400 mg/kg dose produced a greater increase than the 200 mg/kg dose and approached the values observed in the normal-control and omeprazole-treated groups. Restoration of GSH suggests that the extract may preserve endogenous antioxidant capacity and improve the ability of gastric tissue to counteract oxidative stress. The overall antioxidant profile indicates a clear relationship between oxidative injury and gastric mucosal damage. Ulcer induction increased MDA while simultaneously decreasing SOD, CAT and GSH, demonstrating enhanced oxidative stress and depletion of endogenous antioxidant defenses. Conversely, treatment with Alpinia galanga reversed these alterations, as evidenced by reduction in MDA and restoration of SOD, CAT and GSH. The stronger effect observed at 400 mg/kg compared with 200 mg/kg suggests a dose-related antioxidant response. The antioxidant effects of Alpinia galanga may be associated with its phytochemical constituents, particularly flavonoids, phenolic compounds, tannins and other secondary metabolites reported in the plant. Such compounds may contribute to free-radical scavenging, inhibition of lipid peroxidation and preservation of endogenous antioxidant enzymes. Flavonoid and phenolic constituents may also stabilize cellular membranes and reduce oxidative damage associated with ulcerogenic stress. These mechanisms may act together with other gastroprotective mechanisms, including reduction of gastric acid-related injury, preservation of mucus secretion and maintenance of gastric mucosal integrity. The findings obtained with the standard drug omeprazole further support the validity of the experimental model. Omeprazole markedly reduced MDA and restored SOD, CAT and GSH levels compared with the ulcer-control group. Although omeprazole is primarily recognized for its inhibition of gastric acid secretion, improvement

of the oxidative environment may accompany protection of the gastric mucosa. The comparable improvement produced by the higher dose of Alpinia galanga suggests that its gastroprotective effect may involve mechanisms extending beyond acid suppression, particularly enhancement of antioxidant defense. Taken together, the alterations in MDA, SOD, CAT and GSH demonstrate that oxidative stress plays an important role in the development of gastric mucosal injury in the present experimental models. Alpinia galanga extract, particularly at 400 mg/kg, substantially corrected these biochemical abnormalities. The reduction in lipid peroxidation together with restoration of enzymatic and non-enzymatic antioxidant defenses provides evidence that the gastroprotective activity of Alpinia galanga may be partly mediated through attenuation of oxidative stress and enhancement of the endogenous antioxidant system. These findings, when considered together with the ulcer index, gastric acidity, mucin, protein and histopathological observations, support the potential of Alpinia galanga as a promising natural source of gastroprotective phytoconstituents. Conclusion The present study demonstrated that the hydroalcoholic extract of Alpinia galanga possesses significant gastroprotective activity against experimentally induced gastric ulceration in rats. Ulcer induction produced marked gastric mucosal damage, increased ulcer index and gastric acid secretion, together with depletion of important antioxidant defenses. The ulcer-control group showed increased MDA levels and decreased SOD, CAT and GSH levels, confirming the involvement of oxidative stress in gastric mucosal injury. Pretreatment with Alpinia galanga extract at doses of 200 and 400 mg/kg produced a dose-dependent reduction in ulcer severity and improvement in gastric mucosal protection. The extract reduced lipid peroxidation, as indicated by decreased MDA levels, while simultaneously restoring the activities of SOD and CAT and the level of reduced glutathione. The higher dose of 400 mg/kg produced greater antioxidant and gastroprotective effects than the 200 mg/kg dose and showed an overall response approaching that of the standard drug omeprazole. The gastroprotective activity of Alpinia galanga may be attributed to its phytochemical constituents, particularly flavonoids, phenolic compounds, tannins and other bioactive secondary metabolites. These constituents may contribute to free-radical scavenging, inhibition of lipid peroxidation, preservation of endogenous antioxidant defenses and maintenance of gastric mucosal integrity. The overall findings suggest that the gastroprotective effect of Alpinia galanga may involve multiple mechanisms, including reduction of gastric acidity, enhancement of mucosal defensive factors and attenuation of oxidative stress. Thus, the present findings indicate that Alpinia galanga is a promising medicinal plant with potential antiulcer and antioxidant activity. The higher dose demonstrated particularly encouraging gastroprotective effects. Further studies involving isolation and characterization of the active phytoconstituents, detailed molecular investigations, dose optimization, chronic toxicity evaluation and clinical studies are warranted to establish its therapeutic potential and safety for the management of gastric ulcer disorders.

CONCLUSION:

The present study demonstrated that the hydroalcoholic extract of Alpinia galanga possesses promising gastroprotective and antioxidant activities against experimentally induced gastric ulceration in rats. Gastric ulcer induction resulted in considerable damage to the gastric mucosa, increased lipid peroxidation and depletion of endogenous antioxidant defense mechanisms, as evidenced by elevated malondialdehyde (MDA) levels and decreased superoxide dismutase (SOD), catalase (CAT) and reduced glutathione (GSH) levels. These biochemical alterations confirm the involvement of oxidative stress in the development and progression of gastric mucosal injury.

Treatment with Alpinia galanga extract at doses of 200 and 400 mg/kg produced appreciable gastroprotective effects by improving the antioxidant status of gastric tissue and reducing oxidative damage. The extract decreased MDA levels while restoring SOD and CAT activities and GSH concentrations towards normal values. Among the tested doses, 400 mg/kg demonstrated greater protective activity than 200 mg/kg, indicating a dose-related improvement in antioxidant defense and gastric mucosal protection. The effects observed with the higher dose were comparable to those produced by the standard drug omeprazole.

The gastroprotective potential of Alpinia galanga may be attributed to its phytochemical constituents, particularly flavonoids, phenolic compounds, tannins and other biologically active secondary metabolites. These compounds may exert protective effects through free-radical scavenging, inhibition of lipid peroxidation, enhancement of endogenous antioxidant mechanisms and preservation of gastric mucosal integrity. The overall findings suggest that the antiulcer activity of the extract may involve multiple protective mechanisms, including modulation of gastric acid-related injury and attenuation of oxidative stress.

In conclusion, Alpinia galanga represents a promising natural source of gastroprotective and antioxidant phytoconstituents and may have potential for further development as a supportive therapeutic agent in the management of gastric ulcer disorders. However, further investigations

involving bioactive compound isolation, pharmacological characterization, molecular mechanism studies, dose optimization, long-term toxicity evaluation and clinical trials are necessary to establish its efficacy, safety and therapeutic applicability.

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