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Evaluation Of Anti-Ulcer And Anti-Oxidant Effects Of *Erythrina Variegata* Linn (Var.Alba) Bark

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Abstract
Prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs) can cause gastric ulceration and other gastrointestinal complications. This study evaluated the anti-ulcer and antioxidant effects of a 70% methanolic bark extract of Erythrina variegata Linn (EV). The EV extracts (100 and 200 mg/kg, b.w., p.o.) was tested in rats using aspirin (200 mg/kg, b.w.) and ethanol induced (80%1 ml/rat) gastric ulcer models, with ranitidine (50 mg/kg, b.w) and omeprazole (20 mg/kg, b.w) as standards. Antioxidant potential was assessed in vitro methods using DPPH and hydrogen peroxide scavenging assays including quantitative estimation of phytoconstituents was also assessed.
EV bark extract significantly reduced ulcer indices (5.8±0.06, 4.6±0.04 in aspirin induced 6.6±0.04, 5.3±0.06 in ethanol induced) and increased ulcer inhibition (50.84±61.01% in aspirin; 51.1±160.74% in ethanol). Standard drugs showed greater inhibition (66.94% and 68.14%). In antioxidant assays, EV extract produced dose-dependent effects, with 60.13±0.21% and 59.41±0.32% scavenging at 100 µg/ml, and 77.24±0.14% and 81.21±0.18% at 200 µg/ml in DPPH and H_2O_2 models, respectively, compared to ascorbic acid (66.01±0.31% and 86.11±0.24%).
In conclusion, the 70% methanolic bark extract of Erythrina variegata demonstrated significant, dose-dependent anti-ulcer and antioxidant activities, due to presence of phenolic, flavonoid and alkaloids contents to supporting its potential as a natural gastroprotective agent.
Key words: *Erythrina variegata, gastric ulcer, ulcer index, antioxidant, free radicals.*

INTRODUCTION

Peptic ulcer disease (PUD) is characterized by the presence of damage or ulcers in the mucosal lining of the gastrointestinal tract (GI), which leads to the excessive secretion of gastric acid or pepsin typically these symptoms, appear within the gastric region and the proximal segment of the duodenum[1].

PUD has a global incidence of approximately 200–250 cases per 100,000 populations, with a higher prevalence observed in developing regions. The disorder affects both men and women similarly and can occur at any age, although it most commonly manifests during adolescence, around 10–15 years of age. Clinically, PUD presents with symptoms such as epigastric discomfort, bloating, nausea, gastrointestinal bleeding (manifested as hematemesis or melena), loss of appetite, and unintended weight loss [2].

NSAIDs are widely used to manage pain, inflammation, fever, and certain cardiovascular and rheumatic conditions. However, prolonged use can lead to serious gastrointestinal complications, including bleeding and ulceration. Drugs such as aspirin and indomethacin induce ulcers primarily by inhibiting cyclooxygenase, which reduces prostaglandin-mediated mucosal protection. Additionally, NSAIDs promote oxidative stress through free radical generation, enhance neutrophil activation and adhesion, decrease gastric blood flow, impair the mucus barrier, and increase acid-pepsin secretion [3].

Reactive oxygen species (ROS) contribute to gastric injury by disrupting physical, chemical, and physiological defenses, leading to ulcer formation in humans and experimental models. Oxidative stress, resulting from elevated ROS or reduced antioxidant levels, plays a key role in the development of gastric ulcers. Other risk factors for peptic ulcer disease include stress, diet, smoking, alcohol consumption, *Helicobacter pylori* infection, blood group, and gender [4].

Although several chemical agents are available for the treatment of PUD, many are associated with adverse effects. H2-receptor antagonists can cause headache, skin rash, and arrhythmias, while proton pump inhibitors may lead to hypergastrinemia and atrophic gastritis[5]. Antacids can result in bloating, belching, constipation, and an increased risk of ulcer perforation[6]. Other drugs, such as anticholinergics, may induce dry mouth, urinary retention, blurred vision, constipation, and exacerbate glaucoma. Ulcer-protective agents can cause diarrhea, dizziness, edema, and hypophosphatemia, whereas prostaglandin analogues may lead to abdominal cramps, uterine bleeding, or miscarriage[7,8].

In this modern era also 75-80% of the world population still use herbal medicine mainly in developing countries, for primary health care because of better cultural acceptability, better compatibility with the human body and least side effects, Materia medica provides lots of information about ethnomedicinal herbs, which are valuable as antiulcer agents[3,9].

Erythrina variegata (syn. *Erythrina indica*) is a thorny deciduous tree of the Fabaceae family, well known for its diverse phytoconstituents and broad pharmacological potential. Phytochemical investigations have revealed the presence of several bioactive classes, including alkaloids, flavonoids, triterpenoids, lectins, phenolic compounds, saponins, and coumarins[10,11]. Among these, alkaloids exhibit anti-inflammatory, analgesic, and neuromuscular blocking activities[10], while flavonoids such as alpinumisoflavone, auriculatin, osajin, and scandenone demonstrate antioxidant, antibacterial, and anthelmintic properties[12], with some showing affinity toward proteins implicated in Alzheimer's disease. Triterpenoids contribute to antioxidant and anti-inflammatory effects, and phenolic compounds enhance antimicrobial and free radical scavenging activity[10]. Isoflavonoids including eryvarin M, eryvarin H, and neobavaisoflavone have also shown promising enzyme inhibition relevant to diabetes management[13].

Pharmacologically, *E.variegata* has demonstrated potent antioxidant[14], anti-inflammatory[15], analgesic[10], antidiabetic[13,15], and neuroprotective properties[12], supporting its traditional use in the treatment of fever, pain, infections, and neurological disorders. In particular, its bark extract has been reported to improve memory and inhibit acetylcholinesterase, indicating therapeutic potential in neurodegenerative conditions[12].

The EV has been traditionally used for various ailments, its bark extract (var.alba) has not been extensively investigated for combined anti-ulcer and antioxidant effects in validated animal models. This study provides the first systematic evaluation of its dual gastroprotective and free radical scavenging activities, highlighting its potential as a natural alternative to conventional anti-ulcer drugs that are often associated with adverse effects.Considering these facts, the present study was aimed to evaluate the anti-ulcer activity of EV bark extract using in vivo models, and its antioxidant potential through in vitro assays.

MATERIALS AND METHODS

Collection and authentication of plant

The stem bark of *Erythrina variegata* was collected in March from Maraur village, Kalaburagi district, Karnataka. A herbarium specimen was prepared and submitted to the Department of Botany, H.K.E. Society's V.G. Women's Degree College, Kalaburagi for authentication.

Preparation of bark powder and extraction

The collected bark was thoroughly washed with water, shade-dried at temperatures below 40 °C for three days, and cut into small pieces. The dried material was pulverized using a cutter mill and passed through a 52 mm sieve to obtain a coarse, yellowish powder. A total of 250 g of this powder was subjected to Soxhlet extraction with 70% methanol. The extract was concentrated under reduced pressure and stored in a desiccator until further use for anti-ulcer and antioxidant studies[16].

Flavonoid content

The total flavonoid content of EV extracts was determined using the aluminum chloride colorimetric method. Briefly, 1 ml of EVME or EVAE at various concentrations was mixed with 3 ml methanol, 0.2 ml of 10% aluminum chloride, 0.2 ml of 1 M potassium acetate, and 5.6 ml of distilled water. The mixture was incubated at room temperature for 30 minutes, and absorbance was measured at 415 nm using a UV-VIS spectrophotometer (Shimadzu UV-1280), with methanol as the blank. Flavonoid content was calculated as quercetin equivalents using a standard calibration curve[17]. Results are expressed as mean ± SD of three independent determinations.

Phenolic content

The total phenolic content of EV methanolic (EVME) and aqueous (EVAE) extracts was determined using the Folin–Ciocalteu method. Briefly, 0.1 ml of extract solution (50 mg/ml) was mixed with 7.9 ml distilled water, 0.5 ml Folin–Ciocalteu reagent, and 1.5 ml of 20% sodium carbonate. After reacting at room temperature for 2 hours, absorbance was measured at 750 nm. A blank without extract was used as reference. Results were expressed as mg gallic acid equivalents per gram of dry extract, using a gallic acid calibration curve[18].Results are expressed as mean ± SD of three independent determinations.

Anti-oxidant capacity

The total antioxidant capacity of EV was evaluated using the phosphomolybdenum assay. For total antioxidant capacity, 0.3 ml of each extract or sub-fraction in methanol, along with ascorbic acid standards (5–200 µg/ml) and a blank (methanol and water), were mixed with 3 ml of phosphomolybdenum reagent. The mixtures were incubated at 95°C for 90 minutes, cooled to room temperature, and absorbance measured at 695 nm against the blank. Antioxidant activity was expressed as ascorbic acid equivalents and calculated from the standard calibration curve. Results are expressed as mean ± SD of three independent determinations[19].

Alkaloid content

The dried stem bark powder of EV was initially moistened with dilute ammonia and subjected to continuous extraction with 95% alcohol for 18 hours. The alcohol was removed via distillation, and the remaining residue was extracted repeatedly (five times) with 25 ml portions of 1N HCl until alkaloid extraction was complete. The resulting acidic solution was transferred to a separating funnel and washed with 5 ml of chloroform, which was discarded. The solution was then rendered alkaline with ammonia and repeatedly extracted (five times, 25 ml each) with chloroform. The combined chloroform extracts were washed twice with 5 ml portions of water, filtered, and evaporated to dryness. To ensure constant weight, 5 ml of alcohol was added to the residue and evaporated repeatedly in a vacuum desiccators [20]. The total alkaloid content was expressed as a percentage of the dried bark powder.

DPPH scavenging activity

The DPPH (α,α-diphenyl-β-picrylhydrazyl) assay was used to evaluate free radical scavenging activity. A 0.004% DPPH solution was prepared in methanol. Stock solutions of the plant extract and ascorbic acid (100µg/mL) were prepared in methanol, from which 0.1 mL of various concentrations was mixed with 3 mL of DPPH solution. The mixtures were incubated in the dark for 30 min, and absorbance was measured at 517 nm using a spectrophotometer. Methanol with DPPH served as the control. The percentage inhibition was calculated using the formula[21]:

$$[(A_0\text{-}A1) / A_0] \times 100$$

Where A_0 is the absorbance of the control and A_1 is the absorbance of the extract/standard. Antioxidant activity was expressed as IC_{50} values. All assays were performed in triplicate, and results are presented as mean ± standard deviation (SD).Linear regression analysis was used to calculate the IC_{50} value.

Hydrogen peroxide scavenging activity

Hydrogen peroxide (20mM) solution was prepared in phosphate buffer saline (pH 7.4). Various concentrations of the plant extract and ascorbic acid (standard) were prepared in methanol. Test solutions (1 mL) were mixed with 2 mL of hydrogen peroxide solution and incubated for 10 min at room temperature. Absorbance was recorded at 230 nm using a spectrophotometer. Phosphate buffer without hydrogen peroxide served as the blank[22]. Percent inhibition was calculated using formula 1. The scavenging potential of the extract was compared with that of ascorbic acid, and results were expressed as Mean ± SD from triplicate experiments. Linear regression analysis was used to calculate the IC_{50} value.

Experimental animals:

The study was carried out on Wistar albino rats weighing 150–200g. Animals were maintained under standard laboratory conditions at 23±1°C with a 12 h light/dark cycle and were provided with a standard diet and water *ad libitum*. Ethical approval for the experimental protocol was obtained from the Institutional Animal Ethics Committee (IAEC), with clearance dated 24-02-2020 (Registration No. 1948/PO/Re/S/17/CPCSEA-23-02-2017).

Acute toxicity study:

This study was performed as per the up-and-down-procedure of Organization for Economic Cooperation and Development (OECD) guidelines 425. A limit dose of 2000 mg/kg,bw of 70% methanolic and aqueous extracts of EV was used involving five mice (20-25g). Each mouse was treated with a single oral dose of 2000 mg/kg,bw of extract in sequence at 48 hrs intervals. Animals were observed individually at least once during the first 30 min after dosing, periodically during the first 24 hrs and daily thereafter, for a total of 14 days for any clinical signs of toxicity or mortality.

Experimental design:

Doses of 100 mg/kg and 200 mg/kg of the 70% methanolic bark extract of *Erythrina variegata* were selected for anti-ulcer evaluation. Separate experiments were conducted using aspirin- and ethanol-induced ulcer models. In

each model, thirty-six Wistar albino rats of either sex (150–200 g) were randomly divided into four groups (n = 6 per group) after fasting for 24 h with free access to water. [13]
Group I (Control): 1% CMC solution (vehicle, p.o.)
Group II (Standard):
Aspirin induced model: Ranitidine (50 mg/kg,p.o.)
Ethanol induced model: Omeprazole (20 mg/kg,p.o.)
Group III (Test 1): EV bark extract (100 mg/kg b.w., p.o.)
Group IV (Test II: EV bark extract (200 mg/kg b.w., p.o.)

Aspirin-induced gastric ulcer model:

All groups received their respective treatments orally, followed by aspirin (200 mg/kg,p.o.) after 1 h to induce gastric ulcer. Animals were sacrificed 4 h later, and the stomachs were excised, opened along the greater curvature, and examined[23]. The ulcer index and percentage inhibition of ulcer were calculated.

Ethanol-induced gastric ulcer model:

One hour after administration of test and standard drugs, gastric ulceration was induced with 1 mL of 80% ethanol (p.o.). Animals were sacrificed 1 h later, and the stomachs were removed, opened along the greater curvature, and examined[24]. The ulcer index and percentage inhibition of ulcer were determined.

Ulcer scoring and Ulcer index determination:

The stomach was cut open along the greater curvature, rinsed with saline to remove gastric content and blood clots and examined by a ×5 magnifier lenses to assess the formation of ulcers. The numbers of ulcers were counted. Ulcer scoring was undertaken.The score was:0= no ulcer, 1= superficial ulcer,2= deep ulcer,3=perforation.

Ulcer index was measured by using following formula.

$U_I=U_N+U_S+U_P\times10^{-1}$

U_I=Ulcer Index

U_N= Average number of ulcers per animal

U_S=Average number of severity score

U_P= Percentage of animals with ulcers

Percentage inhibition of ulcer was calculated as below:

% Inhibition of Ulcer = (Ulcer index $_{Control}$– Ulcer index $_{Test}$×100/ Ulcer index Control.[23, 24]

Statistical Analysis

Values represent mean ± standard error of the mean, n=6, analysis was performed by using (Graph Pad Prism 10.2.3 version) One-Way ANOVA followed by Tukey's multiple comparison test, *p* value less than 0.05 was considered as statistically significant.*p<0.05, ** p<0.01, and ***p<0.001, data of group II compared with Group I, and data of group III and IV compared with Group II.

RESULTS:

Quantitative Estimation of Phytoconstituents

The total flavonoid content of EV bark extracts was 1.98±0.12 mg/g (quercetin equivalents) in the 70% methanolic extract and 1.41±0.10 mg/g in the aqueous extract.The total phenolic content was 110±0.32 mg/g in the 70% methanolic extract and 94±0.91 mg/g in the aqueous extract, expressed as gallic acid equivalents.The total antioxidant capacity, expressed as ascorbic acid equivalents, was 1.74±0.39 mg/g for the methanolic extract and 1.21±0.14 mg/g for the aqueous extract.The total alkaloid content of *E. variegata* bark powder was found to be 0.72%.Across all parameters, the 70% methanolic extract consistently exhibited higher levels of phytoconstituents compared to the aqueous extract, suggesting a greater concentration of bioactive compounds that may contribute to its stronger antioxidant and anti-ulcer potential.

DPPH scavenging activity

The results of the DPPH assay are presented in Table 1 and Figure 1. Both the methanolic (EVME) and aqueous (EVAE) extracts of EV bark demonstrated notable antioxidant activity compared with the standard, ascorbic acid. The half-maximal inhibitory concentration (IC_{50}) of ascorbic acid was 19.78±0.59 µg/mL, whereas EVME and EVAE showed IC_{50} values of 74.44±0.89 µg/mL and 86.78 µg/mL, respectively. In both extracts, the percentage inhibition of DPPH radicals increased in a dose-dependent manner, confirming their free radical scavenging potential.

Hydrogen peroxide scavenging activity

The hydrogen peroxide scavenging assay revealed a significant, dose-dependent antioxidant response from both extracts. At a concentration of 200 µg/mL, the maximum percentage inhibition was 94.32±0.22% for EVME and 81.21±0.18% for EVAE. The IC_{50} values were calculated as 52.23±0.63 µg/mL (EVME) and 89.50±0.87 µg/mL (EVAE), indicating stronger activity of the methanolic extract compared with the aqueous extract. Ascorbic acid, used as the standard, exhibited superior activity.

From the above activities results it was found that EVME was more effective compared to EVAE. Hence further pharmacological investigation was conducted by EVME. The detailed results are presented in Table 1.

Table 1. Effect of EVME and EVAE in DPPH and hydrogen peroxide scavenging activity

Concentration in (µg/ml) (n=3)	% of Inhibition of EVAE and EVME				
	Standard (ascorbic acid)	DPPH Scavenging		H_2O_2 Scavenging	
		EVME	EVAE	EVME	EVAE
10	38.10 ±0.21	21.38±0.31	19.01±0.21	29.22±0.12	25.51±0.62
20	52.21±0.31	28.10±0.28	27.20±0.26	32.14±0.24	29.22±0.40
40	71.20±0.20	34.22±0.32	39.32±0.21	44.23±0.15	33.13±0.22
60	79.01±0.12	48.20±0.19	46.41±0.31	56.18±0.23	39.10±0.31
80	87.20±0.52	59.49±0.24	52.22±0.17	68.10±0.20	47.24±0.14
100	92.01±0.31	66.10±0.18	60.13±0.21	77.50±0.15	59.41±0.32
200	96.11±0.24	88.53±0.21	77.24±0.14	94.32±0.22	81.21±0.18
IC_{50}	**19.78±0.59µg/ml**	**74.44±0.89µg/ml**	**86.78±0.94µg/ml**	**52.23±0.63µg/ml**	**89.50±0.87µg/ml**

Each value represents mean±SD (n=3)

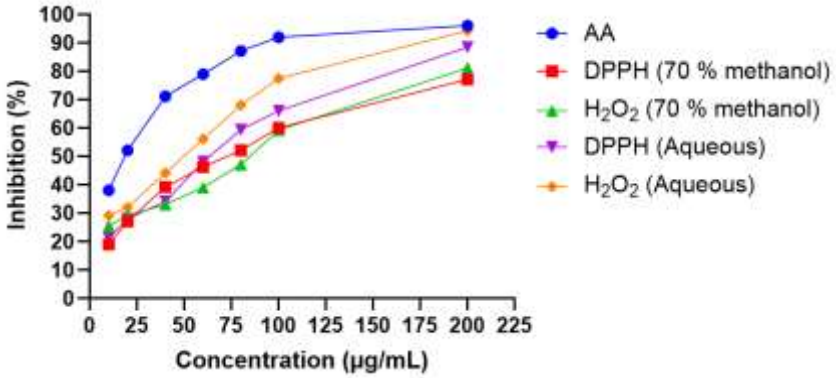

Figure 1.Effect of EV in DPPH and H_2O_2 scavenging activity

Aspirin induced gastric ulcer

The treatment with EV significantly reduced ulcer formation in aspirin-induced gastric ulceration. The ulcer index values were markedly decreased to 5.8±0.06 and 4.6±0.04 at doses of 100 and 200 mg/kg, respectively, compared with the control (11.8±0.01). The standard drug, ranitidine (50 mg/kg), produced an ulcer index of 3.9±0.08. The percentage inhibition of ulceration was 50.84% and 61.01% for the 100 and 200 mg/kg EVME-treated groups, respectively, while the standard drug showed 66.94% inhibition. These results demonstrate that EVME provides significant gastroprotection in a dose-dependent manner, approaching the effect of the standard treatment.

Ethanol induced gastric ulcer

The treatment with EV at doses level of 100 and 200 mg/kg, result a significant reduction in ulcer index compared to the control group. The ulcer index values were 6.6 ± 0.04 and 5.3±0.06 for the 100 and 200 mg/kg groups, respectively, versus 13.5±0.07 in the control. The standard drug, omeprazole (20 mg/kg), showed an ulcer index of 4.3±0.09. The percentage inhibition of ulceration was 51.11% and 61.74% for the EVME-treated groups (100 and 200 mg/kg), while omeprazole exhibited 68.14% inhibition. These findings indicate that EVME significantly protects against ethanol-induced gastric mucosal injury in a dose-dependent manner, with effects comparable to the standard treatment.

Table 2. Ulcer protective effect of 70% methanolic extract of EV in aspirin induced ulcer in rats

Treatment	Dose (mg/kg) (p.o.)	Ulcer index (Mean ± SEM)	% of inhibition
Group-I 1% CMC solution	20mg/kg	11.8±0.01	-
Group-II Ranitidine	50	3.9±0.08 ***	66.94
Group-III 70% methanolic extract of EV	100	5.8±0.06***	50.84
Group-IV 70% methanolic extract of EV	200	4.6±0.04***	61.01

Values represent mean ± standard error of the mean, n=6, analysis was performed by using (Graph Pad Prism 10.2.3 version) One-Way ANOVA followed by Tukey's multiple comparison test, *p* value less than 0.05 was considered as statistically significant. *p<0.05, ** p<0.01, and ***p<0.001, data of group II compared with Group I, and data of group III and IV compared with Group II.

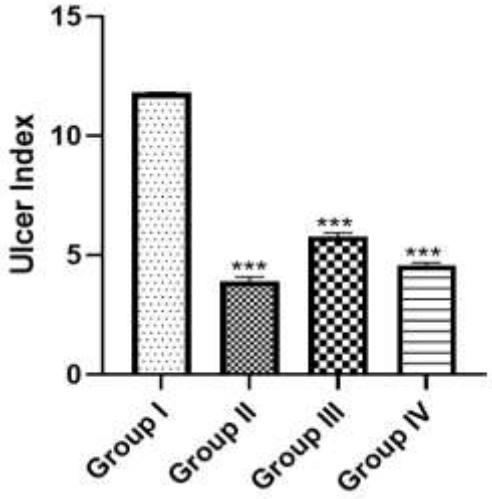

Fig. Effect of EV in aspirin induced ulcer in rats

Table 3. Ulcer protective effect of 70% methanolic extract of EV in ethanol induced gastric ulcer in rats

Treatment (n=6)	Dose (mg/kg) (p.o.)	Ulcer index (Mean ± SEM)	% of inhibition
Group-I 1% CMC solution	20ml/kg	13.5±0.07	-
Group-II Omeprazole	20	4.3±0.09***	68.14
Group-III 70% methanolic extract of EV	100	6.6±0.04***	51.11
Group-IV 70% methanolic extract of EV	200	5.3±0.06***	60.74

Values represent mean ± standard error of the mean, n=6, analysis was performed by using (Graph Pad Prism 10.2.3 version)One-Way ANOVA followed by Tukey's multiple comparison test, *p* value less than 0.05 was considered as statistically significant. *p<0.05, ** p<0.01, and ***p<0.001, data of group II compared with Group I, and data of group III and IV compared with Group II.

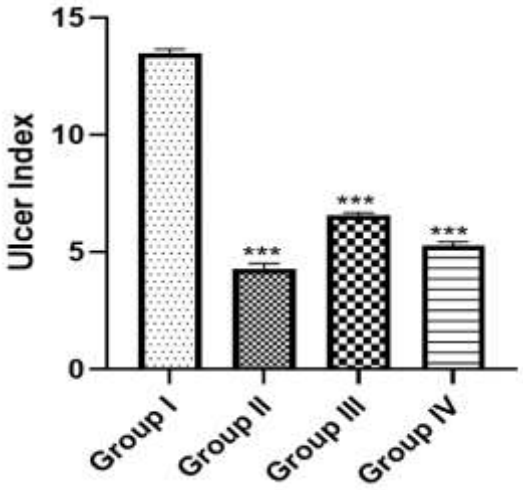

Fig. Effect of EV in ethanol induced gastric ulcer in rats.

DISCUSSION

PUD remains a major gastrointestinal disorder, with NSAID use and alcohol consumption among the leading causes. NSAID-induced ulcers are attributed to inhibition of cyclooxygenase (COX) enzymes, leading to reduced prostaglandin synthesis, diminished mucus secretion, and impaired mucosal defence. Ethanol-induced ulcers, on the other hand, are strongly associated with oxidative stress through the generation of reactive oxygen species (ROS), lipid peroxidation, and direct mucosal injury. Thus, agents possessing both antioxidant and anti-ulcer properties are of considerable therapeutic interest.

The aspirin-induced ulcer model is widely used to study NSAID-related gastric injury, which primarily involves the inhibition of cyclooxygenase enzymes and subsequent suppression of prostaglandin synthesis, leading to reduced mucus and bicarbonate secretion. In the aspirin-induced ulcer model, EVME markedly reduced ulcer index values and achieved ulcer inhibition of 50.84% and 61.01% at 100 and 200 mg/kg, respectively, compared with 66.94% for ranitidine.These results indicate that EVME may preserve mucosal integrity, potentially by stimulating prostaglandin pathways or enhancing mucosal defence mechanisms. The standard drug ranitidine produced 66.94% inhibition, showing that EVME possesses considerable gastroprotective potential though slightly less potent than the reference drug.

In the ethanol-induced ulcer model, gastric damage is primarily mediated by oxidative stress and lipid peroxidation, resulting in mucosal necrosis and haemorrhage. EVME significantly reduced ulcer index values at both test doses (6.6±0.04 and 5.3±0.06) compared to the control (13.5±0.07), corresponding to ulcer inhibition of 51.11% and 61.74%, respectively. Omeprazole, the standard, produced 68.14% inhibition. The close comparability of EVME with omeprazole underscores its effectiveness in attenuating ethanol-induced oxidative mucosal damage.

The antioxidant findings strongly support the anti-ulcer activity of EVME. The extract demonstrated dose-dependent scavenging of DPPH and hydrogen peroxide radicals, with IC_{50} values of 74.44±0.89 µg/mL and 52.23±0.63µg/mL, respectively, confirming its free radical–neutralizing potential. Phytochemical analysis revealed high levels of phenolics (110±0.32 mg/g) and flavonoids (1.98±0.12 mg/g), which are known contributors to antioxidant and cytoprotective mechanisms. These constituents likely underlie the gastroprotective effects by mitigating oxidative stress and maintaining mucosal defence.

Notably, EVME exhibited stronger antioxidant activity and higher phytochemical content than the EVAE, aligning with its superior performance in both ulcer models. The presence of phenolic and flavonoid compounds, recognized for their ROS-scavenging and mucosal-protective properties, provides a plausible explanation for the observed efficacy of EVME in reducing gastric lesions.

Our results align with earlier reports on the pharmacological properties of *Erythrina* species, including antioxidant, anti-inflammatory, and cytoprotective activities. The observed gastroprotection can thus be attributed to the synergistic actions of multiple phytoconstituents, particularly phenolics, flavonoids, and alkaloids, which are abundant in the methanolic extract.

The findings are consistent with earlier reports on the pharmacological activities of *Erythrina* species, which include anti-inflammatory, antimicrobial, and antioxidant effects. This study extends current knowledge by providing in vivo evidence for the anti-ulcer efficacy of EV bark and its mechanistic link to antioxidant defence. Taken together, the findings suggest that EV bark possesses significant gastroprotective and antioxidant properties, supporting its traditional use in managing gastrointestinal disorders. The results also highlight the therapeutic potential of EVME as a natural alternative to synthetic anti-ulcer agents, which are often associated with adverse effects. However, further studies are required to isolate and characterize the active constituents, elucidate their precise mechanisms of action, and confirm efficacy through chronic ulcer models and clinical evaluations.

CONCLUSION

The present study demonstrated that the EV possesses significant gastro-protective effects in aspirin- and ethanol-induced ulcer models, along with potent antioxidant activity confirmed by DPPH and hydrogen peroxide scavenging assays. The higher phenolic and flavonoid contents of EVME likely contribute to its strong free radical scavenging and anti-ulcer effects. These findings provide scientific support for the use of EV and highlight its potential as a natural therapeutic agent against gastric ulcers and oxidative stress–related disorders. Further phytochemical and pharmacological investigations are recommended to isolate the active constituents and elucidate their mechanisms of action.

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