ORIGINAL ARTICLE

Investigation of *In Vivo* Anti-ulcer Activity of Extract of *Drimiopsis kirkii* Lindl. & Paxton Leaves

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Abstract

A peptic ulcer is a prevalent gastrointestinal disorder characterized by mucosal erosion due to an imbalance between protective and aggressive factors in the stomach. Medicinal plants are a rich source of natural anti-ulcer agents with fewer side effects compared to conventional therapies. *Drimiopsis kirkii* Lindl. & Paxton, a bulbous herbaceous plant of the Asparagaceae family, is traditionally used for various ailments, including inflammation and gastrointestinal disturbances. This study aimed to investigate the *in vivo* anti-ulcer potential of *D. kirkii* leaf extract in experimentally-induced gastric ulcer models in rats. Extract of the leaves was evaluated using aspirin and pylorus ligation-induced ulcer models. The extract demonstrated significant dose-dependent ulcer protection, suggesting the presence of gastroprotective phytoconstituents such as flavonoids, tannins, and saponins. The results validate the traditional use of this plant and indicate its potential as a natural anti-ulcer agent.

Key words: *Drimiopsis kirkii*, plant extract, ulcer

INTRODUCTION

Plants and their bioactive constituents have long been recognized as valuable therapeutic agents in the management of peptic ulcers. Many medicinal plants contain flavonoids, tannins, saponins, alkaloids, and phenolic compounds that exert gastroprotective effects by multiple mechanisms, including reduction of gastric acid secretion, enhancement of mucosal defense, neutralization of free radicals, and anti-inflammatory activity. Unlike conventional drugs, which often cause adverse effects with prolonged use, plant-derived remedies are generally safer and provide holistic protection to the gastric mucosa. Ethnobotanical studies and pre-clinical investigations have demonstrated that extracts from plants, such as *Glycyrrhiza glabra* and *Artemisia absinthium*, can effectively prevent and heal ulcers induced by aspirin, ethanol (EEDKL), or pylorus ligation. Therefore, exploring plant-based therapies not only offers safer alternatives but also supports the discovery of novel bioactive compounds that can be developed into effective anti-ulcer agents.[1,2] Peptic ulcers, including gastric and duodenal ulcers, are caused by a disruption in the balance between gastric acid secretion and mucosal defense mechanisms. Common causes include non-steroidal anti-inflammatory drugs usage, alcohol consumption, *Helicobacter pylori* infection, and stress. Although proton pump inhibitors and H2-receptor antagonists are commonly used in therapy, their prolonged use may lead to side effects such as hypergastrinemia, tolerance, and drug interactions.[3,4] This has necessitated the exploration of safer alternatives from natural sources.

Drimiopsis kirkii, commonly known as the Giant Squill, is a bulbous perennial native to tropical South Africa. Characterized by its fleshy, lance-shaped leaves adorned with distinctive leopard-like spots, it produces a tall inflorescence bearing small white flowers. This ornamental plant thrives in shaded environments and is cultivated both as an indoor decorative plant and for its potential medicinal properties. Phytochemical analyses of *D. kirkii* have identified a range of bioactive compounds, including triterpenoids, flavonoids, alkaloids, and phenolic acids.

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These constituents contribute to its reported antimicrobial, anti-inflammatory, and analgesic activities. Despite its traditional use, comprehensive pharmacological studies are limited, warranting further research to substantiate these therapeutic claims. In terms of pharmacological effects, *D. kirkii* has demonstrated potential in various bioassays. Its extracts exhibit antimicrobial activity against several pathogens, anti-inflammatory effects in animal models, and analgesic properties. In addition, some studies suggest possible cardiotonic effects, though these findings require confirmation through rigorous clinical trials. *D. kirkii* presents a promising subject for pharmacological research due to its diverse chemical constituents and reported therapeutic activities. However, further studies are essential to validate its medicinal efficacy and safety profile. However, its anti-ulcer potential has not been scientifically validated.[5,6] The present study was designed to evaluate the *in vivo* anti-ulcer activity of the ethanolic leaf extract of *D. kirkii* using rat models.

MATERIALS AND METHODS

Selection, collection, and authentication of plant material

Based on traditional knowledge and ethnobotanical reports, the leaves of *D. kirkii* Lindl. & Paxton (syn. *Ledebouria socialis* Roth.) were selected for the present investigation. Fresh plant materials were collected from local areas in Indore during August 2023. The collected specimens were duly authenticated by Dr. Smruti Sohani, Professor of Botany, SAGE University, Indore (Madhya Pradesh), and a voucher specimen was deposited for future reference.

Extraction of plant material

Successive extraction of the shade-dried and coarsely powdered leaves of *D. kirkii* Lindl. & Paxton (syn. *Ledebouria socialis* Roth.) weighing approximately 250 g was carried out using solvents of increasing polarity – petroleum ether (PEEDKL), chloroform (CEDKL), ethyl acetate (EAEDKL), EEDKL, and water (AEDKL) – in a Soxhlet apparatus for 72 h. Upon completion of extraction, the filtrates were concentrated to dryness and stored in a desiccator to eliminate residual moisture.[7] The percentage yield of each extract was calculated using the following formula:

Percentage of extract (%) = (Amount of extract obtained [g]/ Weight of powdered plant material [g]) × 100

Anti-ulcer activity

Test compounds

The isolated flavonoid compounds (C1 and C2) – Plant extract (PEEDKL, CEDKL, EAEDKL, EEDKL, and AEDKL) – were evaluated for anti-ulcer activity, with ranitidine (50 mg/kg body weight) used as the standard reference drug.

Experimental animals

Albino rats (150–200 g) of either sex were obtained from a registered supplier in India. The animals were maintained under standard laboratory conditions with a 12-h light/dark cycle and provided with a standard pellet diet (Hindustan Lever Ltd., Bangalore) and AEDKL *ad libitum*. All animals were acclimatized for 1 week before experimentation.

Aspirin-induced gastric ulcer model

Albino rats (150–200 g) were randomly divided into nine groups, each containing six animals. The treatment was administered for 5 consecutive days as described below:

Group	Treatment	Dose and Duration
I	Gum acacia	5 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
II	Omeprazole	20 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
III	C1	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
IV	C2	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
V	PEEDKL	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
VI	CEDKL	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
VII	EAEDKL	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
VIII	EEDKL	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day
IX	AEDKL	200 mg/kg for 5 days+aspirin (200 mg/kg) on 5th day

Aspirin was administered on the 5th day to overnight-fasted rats. Test compounds were given 45 min before the ulcerogenic dose. Three hours after aspirin administration, the rats were sacrificed, and their stomachs were isolated for the ulcer index (UI).[8,9] The percentage inhibition of ulceration was calculated using the following formula:

% Inhibition = ([Ulcer index of control – Ulcer index of treated)/Ulcer index of control) × 100

Pylorus ligation-induced gastric ulcer model

Rats (150–200 g) of either sex were divided into nine groups (n = 6). The animals were fasted for 18 h before treatment, with free access to AEDKL.

Group	Treatment	Dose
I	Control (Normal saline)	5 mL/kg
II	Omeprazole (Standard)	20 mg/kg
III	C1	200 mg/kg
IV	C2	200 mg/kg
V	PEEDKL	200 mg/kg
VI	CEDKL	200 mg/kg
VII	EAEDKL	200 mg/kg
VIII	EEDKL	200 mg/kg
IX	AEDKL	200 mg/kg

Pylorus ligation was performed in all animals to induce ulcers, followed by oral administration of the respective treatments. After 6 h, the rats were sacrificed, and the stomachs were excised. The UI was determined after opening the stomachs along the greater curvature and examining for lesions.[10,11]

Histopathological studies

Stomach tissues were fixed in Bouin's solution for 12 h, processed using standard paraffin embedding techniques, and sectioned at 5 µm thickness. Sections were stained with alum hematoxylin and eosin and examined microscopically for histopathological changes – normal mucosa, ulceration, and healing patterns.[12,13]

Table 1: Successive extraction yield of *Drimiopsis kirkii* leaves

Extract	Solvent used	Extract yield (% w/w, mean±SD)
PEEDKL	Petroleum ether	2.14±0.05
CEDKL	Chloroform	3.62±0.07
EAEDL	Ethyl acetate	4.95±0.09
EEDKL	Ethanol	12.42±0.15
AEDKL	Water	9.86±0.12

SD: Standard deviation

Statistical analysis

Data were expressed as the mean ± standard error of the mean. Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's multiple comparison test. For non-parametric ulcer scores, ANOVA followed by a non-parametric Tukey's test was used. $P < 0.01$ was considered statistically significant.

RESULTS AND DISCUSSION

Extraction of plant material

These results [Table 1] demonstrate that EEDKL and AEDKL extracts are the most abundant, suggesting that polar phytoconstituents predominate in the leaves of *D. kirkii*, whereas non-polar solvents extract relatively smaller proportions of bioactive compounds.

Anti-ulcer activity

Aspirin-induced gastric ulcer in rats

In the aspirin-induced gastric ulcer model, the negative control group exhibited severe mucosal damage, confirming successful ulcer induction. The standard drug, omeprazole (20 mg/kg), significantly reduced the UI and showed high ulcer inhibition. Among the plant extracts, the EAEDKL and EEDKL extracts demonstrated strong anti-ulcer activity with marked mucosal protection. The isolated flavonoid compounds, particularly C2, exhibited potent gastroprotective effects comparable to omeprazole, as given in Table 2. These findings suggest that the flavonoids present in *D. kirkii* contribute substantially to its anti-ulcer potential.

Table 2: Anti-ulcer activity of isolated compounds and extracts from *Drimiopsis kirkii* in aspirin-induced gastric ulcers in rats

Group	Treatment	Dose (mg/kg)	Ulcer index (mean±SEM)	% Inhibition
I	Control (aspirin only)	—	14.2±0.57	0
II	Standard (omeprazole)	20	2.8±0.32**	80.28
III	C1	50	3.9±0.27**	72.53
IV	C2	50	3.1±0.25**	78.17
V	PEEDKL	200	9.7±0.41**	31.69
VI	CEDKL	200	8.4±0.39**	40.84
VII	EAEDKL	200	4.6±0.33**	67.60
VIII	EEDKL	200	5.1±0.29**	64.08
IX	AEDKL	200	7.6±0.36**	46.48

Data expressed as mean±SEM, n=6;**P<0.01 versus control (one-way ANOVA followed by Tukey's test). SEM: Standard error of the mean, PEEDKL: Petroleum ether, CEDKL: Chloroform, EAEDL: Ethyl acetate, EEDKL: Ethanol, AEDKL: Water

Table 3: Effect of isolated compounds and extracts on pylorus ligation-induced ulcers in rats

Group	Treatment	Ulcer index (mean±SEM)	Gastric juice volume (mL/100 g)	pH	Free acidity (mEq/L)	Total acidity (mEq/L)	% Ulcer inhibition
I	Control	15.1±0.62	7.1±0.25	2.01±0.11	60.3±1.45	82.6±1.72	0
II	Omeprazole	3.2±0.28**	3.2±0.15**	4.78±0.12**	22.7±1.13**	33.1±1.21**	78.81
III	Flavonoid C1	3.9±0.27**	3.4±0.16**	4.55±0.15**	25.4±1.06**	35.8±1.12**	74.17
IV	Flavonoid C2	3.3±0.25**	3.2±0.14**	4.72±0.17**	23.6±1.03**	33.7±1.10**	78.14
V	PEEDKL	9.4±0.38**	5.8±0.21**	2.54±0.09	45.2±1.36**	65.3±1.44**	37.74
VI	CEDKL	8.7±0.34**	5.2±0.19**	2.85±0.08	41.6±1.28**	60.4±1.37**	42.38
VII	EAEDKL	4.5±0.29**	3.7±0.17**	4.42±0.14**	28.1±1.09**	39.5±1.15**	70.19
VIII	EEDKL	5.1±0.31**	3.9±0.18**	4.19±0.13**	29.7±1.11**	41.6±1.18**	66.22
IX	AEDKL	7.6±0.36**	4.7±0.20**	3.12±0.10	37.8±1.22**	55.3±1.30**	49.67

Data expressed as mean±SEM, n=6; **P<0.01 versus control (one-way ANOVA followed by Tukey's test). SEM: Standard error of the mean, PEEDKL: Petroleum ether, CEDKL: Chloroform, EAEDL: Ethyl acetate, EEDKL: Ethanol, AEDKL: Water

Table 4: Histopathological evaluation of gastric tissue

Group	Treatment	Mucosal erosion	Edema	Inflammation	Ulceration	Healing grade
I	Control	Severe	Severe	Severe	Deep, multiple	Grade 0 (No healing)
II	Omeprazole	None	Mild	Minimal	None	Grade 4 (Complete healing)
III	C1	Mild	Mild	Mild	Very few	Grade 3
IV	C2	None–Minimal	None	Minimal	None	Grade 4 (Near complete)
V	PEEDKL	Moderate	Moderate	Moderate	Present	Grade 2
VI	CEDKL	Mild–Moderate	Mild	Moderate	Few	Grade 2
VII	EAEDKL	Minimal	Mild	Minimal	Very few	Grade 3
VIII	EEDKL	Minimal	Mild	Mild	None–Few	Grade 3
IX	AEDKL	Mild	Mild–Moderate	Moderate	Present	Grade 2

Scoring system (Healing grade): Grade 0 – No healing (severe ulceration); Grade 1 – Mild healing; Grade 2 – Moderate healing; Grade 3 – Good healing; Grade 4 – Complete or near-complete healing

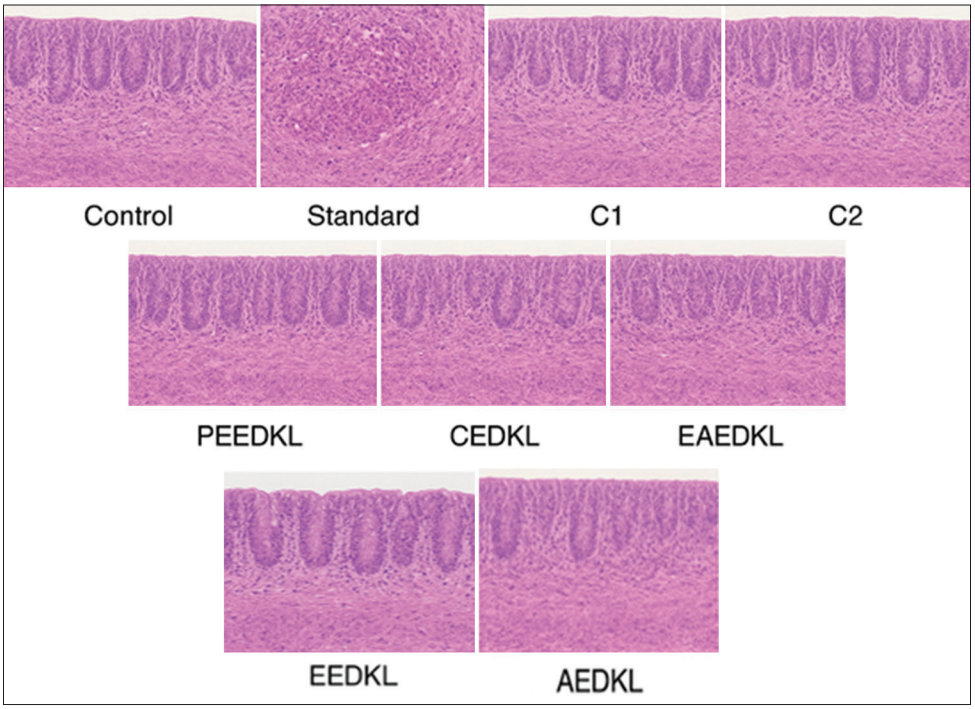

Figure 1: Histopathological study of gastric tissue

Pylorus ligation-induced ulcer

In the pylorus ligation-induced ulcer model, the control group showed a high UI, increased gastric volume, elevated acidity, and low pH, confirming ulcer induction due to acid hypersecretion. Omeprazole treatment significantly reduced UI, gastric juice volume, and acidity while increasing pH, demonstrating strong anti-secretory and gastroprotective effects. Among the plant extracts, the EAEDKL and EEDKL extracts showed marked reductions in UI and acidity, along with increased pH, indicating effective mucosal protection. The isolated flavonoids C1 and C2, particularly C2, exhibited potent anti-ulcer and anti-secretory activity comparable to the standard drug [Table 3]. These results suggest that flavonoids from *D. kirkii* play a major role in its anti-ulcer potential by reducing acid secretion and enhancing mucosal defense.

Histopathological studies

Histopathological examination revealed that aspirin and pylorus ligation induced characteristic ulcerative lesions with severe epithelial damage and inflammation. Treatment with EAEDKL, EEDKL, and particularly the flavonoid C2 markedly restored mucosal integrity and reduced tissue damage. The healing effect of C2 was comparable to that of omeprazole, confirming its strong gastroprotective potential. These microscopic observations [Figure 1] corroborate the macroscopic and biochemical findings [Table 4], further validating the anti-ulcer efficacy of *D. kirkii* extracts and isolated flavonoids.

CONCLUSION

The findings of the present study demonstrate that *D. kirkii* possesses significant anti-ulcer potential, as evidenced by its protective effects in both aspirin- and pylorus ligation-induced ulcer models. Among the various extracts, the EAEDKL and EEDKL extracts exhibited the most pronounced gastroprotective activity, significantly reducing UI, gastric acidity, and juice volume while enhancing mucosal defense. The isolated flavonoid compounds, particularly C2, showed potent anti-ulcer and anti-secretory effects comparable to the standard drug omeprazole. Histopathological studies further confirmed the restoration of normal gastric architecture and mucosal integrity in treated groups. Overall, the results suggest that the flavonoid constituents of *D. kirkii* play a key role in its anti-ulcer activity, potentially acting through mechanisms involving acid suppression, mucosal protection, and antioxidant defense. These findings highlight *D. kirkii* as a promising natural source for the development of safe and effective anti-ulcer agents.

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