Pharmacogn. Res., 2026; 18(2):519-525.
https://www.phcogres.com

Original Article

Evaluation of Anti-Ulcer Activity of Ethanolic Extract of *Prosopis juliflora* seeds in Wistar Albino Rats

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ABSTRACT

Introduction: A peptic ulcer is defined as a breach in the integrity of the mucosa lining the stomach and/or duodenum that results in a localized defect or excavation due to active inflammation. Peptic ulcer disease is a major public health problem affecting 10% of the population worldwide. *Prosopis juliflora* is a widespread phreatophytic plant that has been used since ancient times for medicinal purposes. *Prosopis juliflora* contains bioactive components such as tannins, flavonoids, terpenes and phenolic compounds which possess analgesic, anti-inflammatory, antiulcerogenic, anticancer, antifungal, antihelminthic and immunostimulant properties. **Aim:** This study evaluates the efficacy of ethanolic extract of *Prosopis juliflora* seeds using pylorus ligation-induced gastric ulcerations in Wistar albino rats. **Materials and Methods:** Wistar albino rats were allocated into five groups of five rats each in the pylorus ligation-induced ulcer model. Group I rats were administered normal saline at a dosage of 2 mL/kg p.o., in Group II rats pyloric ligation alone was done, Group III received standard drug Omeprazole 20 mg/kg p.o, Group IV received *Prosopis juliflora* seed extract 250 mg/kg p.o and Group V received *Prosopis juliflora* seed extract 500 mg/kg p.o. In all three groups (Group III-V), drugs were given orally 1 hr prior to pylorus ligation which was carried out under aseptic precautions. 48 hr post pylorus ligation, the rats were anesthetised and the stomach was cut open along the greater curvature to evaluate the ulcer index, gastric pH, free acidity, total acidity and for conducting gross examination of the stomach. **Results:** Phytochemical evaluation of ethanolic extract of *P. juliflora seeds* indicated the presence of tannins, alkaloids, flavonoids, anthraquinones and phenolic compounds which possess pharmaceutical properties such as antibacterial, anti-inflammatory, antipyretic and anti-ulcer activities. Pretreatment of Wistar rats with *P. juliflora* seed extract at doses of 250 mg/kg and 500 mg/kg showed a significant reduction in mean ulcer index and mean ulcer severity score with p value <0.001 compared to the positive control. The gastric secretory parameters were also significantly reduced by *P. juliflora* 500mg/kg with p value <0.001. Group III (Omeprazole) and Group V (*P. juliflora* 500 mg/kg) treated rats showed 63.49% and 53.72% gastroprotection, respectively. **Conclusion:** These findings suggest that the ethanolic extract of *P. juliflora* seeds has potent antiulcer activity and could be considered as a potential alternative source to develop new antiulcer agents.

Keywords: *Prosopis juliflora*, Peptic Ulcer Disease, Pylorus Ligation, *H. pylori*, NSAIDs.

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Received: 15-09-2025;
Revised: 24-11-2025;
Accepted: 09-01-2026.

INTRODUCTION

Peptic ulcer disease is characterized by a discontinuity or erosion in the mucosal lining of the stomach or duodenum, resulting in a localized defect or lesion that arises from active inflammatory processes. PUD encompasses both gastric and duodenal ulcers. Peptic Ulcer Disease (PUD) arises from a disruption in the equilibrium between the aggressive factors-such as gastric acid, pepsin, *Helicobacter pylori* infection, and bile and the defensive mechanisms of the gastric mucosa, including mucus secretion, prostaglandin synthesis, nitric oxide production, mucosal blood flow, cellular resistance, and bicarbonate secretion (Kuna, 2019). Among the various etiological agents, *Helicobacter pylori* infection and NSAID administration remain the predominant contributors to the development of peptic ulcer disease; other factors like alcohol consumption, smoking, stress and Zollinger-Ellison syndrome can also contribute to their development (Richardson, 2009).

Most duodenal ulcers are found in the proximal duodenum, approximately within 3 cm of the pylorus, a region highly susceptible to acid-induced mucosal injury (Asali, 2018). The vast majority of duodenal ulcers are benign, and rarely undergo malignant transformation. Duodenal ulcers are well-defined and generally less than 1 cm in diameter, yet their depth can

DOI: 10.5530/pres.20262223

occasionally extend to 3-6 cm, reaching the muscularis propria. The prevalence of duodenal ulcers has declined, largely due to reduction in *H. pylori* infection rates. Gastric ulcers, in contrast, are more commonly observed in males during the sixth decade of life and are typically located distal to the antrum-corpus mucosal junction. Gastric ulcers carry a risk of malignant transformation that necessitates biopsy at the time of detection.

The gastric mucosa is susceptible to damage primarily driven by the corrosive actions of hydrochloric acid and the proteolytic activity of pepsinogen. Acid-secreting parietal cells contain receptors for various acid stimulants on their surface. Activation of H2 receptors by Histamine leads to activation of Adenyl cyclase-cAMP pathway. Activation of muscarinic and gastrin receptors activates the PKC/phosphoinositol pathway. Each of these signaling pathways activates downstream kinases of H-K-ATPase, leading to acid production.

Chronic infection with *H. pylori,* a gram negative bacteria that colonizes the gastric mucosa has been strongly associated with the development of gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue lymphoma. *H. pylori* produces different virulence factors like γ-Glutamyl Transpeptidase (GGT) that contribute to cellular damage and apoptosis, while the vacuolating cytotoxin (VacA) induces vacuolation and disrupts the integrity of gastric mucosa, the cytotoxin-associated gene A (CagA) protein, that is strongly linked to oncogenic transformation and the Pathogen-Associated Molecular Patterns (PAMPs), such as Lipopolysaccharide (LPS) and flagella, play a critical role in host immune modulation and persistence of infection. The bacterial genome of *H. pylori* harbors the cag pathogenicity island (cag-PAI), which encodes important virulence factors such as the cytotoxin-associated gene A (CagA) protein and the phosphatidylinositol-specific phospholipase C (PicB). *H. pylori* produces urease, which enables its gastric residence and generates ammonia, which produces mucosal injury. Through a Cag A-dependent mechanism, *H. pylori* enhances H^+K^+-ATPase activity, leading to increased acid production. *H. pylori* also produces proteases and phospholipase, causing the breakdown of glycoproteins in the mucus-bicarbonate barrier which forms the primary mucosal defense (Olbe, 2000). NSAIDs induce PUD by inhibiting COX1 and COX2, interrupting prostaglandin synthesis, leading to mucosal injury and impairing mucosal defense. It interrupts mucosal blood flow, decreases mucin and bicarbonate production. NSAIDs cross the lipid cell membrane and get trapped inside the cell in an ionized form, leading to cell injury. NSAIDs also activate the lipoxygenase pathway, leading to the production of proinflammatory cytokines, such as TNF and leukotrienes.

Peptic Ulcer Disease (PUD) is still a common gastrointestinal condition worldwide. Approximately 10% of the world's population is affected by Peptic Ulcer Disease (PUD). In Western countries, the lifelong prevalence of PUD is approximately 5-10%, with an annual incidence of 0.1-0.3%. In India, peptic ulcer disease deaths have reached 68,108 or 0.80% of total deaths based on the most recent WHO data published in 2020 (Sun *et al*., 2013).

Peptic Ulcer Disease (PUD) typically presents with epigastric pain, often described as burning, gnawing, or aching, and may be accompanied by bloating, early satiety, nausea, vomiting, and changes in body weight. Gastrointestinal bleeding can manifest as hematemesis or melena. Duodenal ulcers are frequently associated with nocturnal pain, with symptoms occurring 90 min to 3 hr after meals and often relieved by food or antacids. In contrast, gastric ulcer pain is usually aggravated by food and may be associated with nausea, anorexia, and weight loss. Other less common symptoms include belching, regurgitation, heartburn, and a sensation of abdominal fullness. In gastric ulcers, both basal and stimulated gastric acid secretion is low, while the deficient mucosal defense plays a significant role. In duodenal ulcers, basal and nocturnal acid secretion is high, and bicarbonate secretion is significantly decreased (Jaiswal, 2021).

Complications of PUD include bleeding, perforations, which are most common in NSAID-induced gastric ulcers. Gastrocolic fistula and gastric outlet obstructions, are least common.

Although synthetic antiulcer agents such as proton pump inhibitors, H2 receptor antagonists, and prostaglandin analogs are effective, their chronic use may lead to serious adverse effects, including cardiac arrhythmias, osteoporosis, gynecomastia, and impotence. Furthermore, ulcer recurrence within one year remains a significant limitation of conventional therapy. Consequently, herbal medicines are increasingly considered as alternative treatment modalities owing to their affordability, safety profile, and perceived effectiveness (Singh, 2015).

Prosopis juliflora is a widespread phreatophytic tree, widely distributed in arid and semi-arid areas of the tropical and subtropical regions worldwide and belongs to the Fabaceae family (Dave and Bhandari, 2013). There are 44 species of the genus Prosopis distributed in North, South America, Asia, and Africa. The phytochemical profile of *P. juliflora* is rich in bioactive compounds such as flavonoids (Diarra *et al*., 2015), alkaloids, tannins, anthraquinones, and phenolic compounds (Prabha *et al*., 2014). *Prosopis juliflora* is known for its wide-ranging pharmacological properties, including antibacterial, antifungal, anti-inflammatory, antipyretic, anti-ulcer, and antiprotozoal activities (Preeti *et al*., 2015; Tajbakhsh, 2015; Reddy, 2015). Traditionally, various parts of the plant have been utilized in the management of diabetes, (Rao *et al*., 2024) malaria, conjunctivitis, asthma, liver disorders, and Alzheimer's disease. Beyond its therapeutic applications, *P. juliflora* is valued for its nutritional content and commercial significance.

The present study was designed to investigate the anti-ulcer potential of the ethanolic extract of *Prosopis juliflora* seeds in

Wistar albino rats using a pylorus ligation-induced peptic ulcer model.

MATERIALS AND METHODS

Plant Extract

Prosopis juliflora seeds were washed to remove the contaminants and dried under shade for 2 weeks. Ethanol was used as a solvent to prepare the extract. 50 g of seeds were macerated in 250 mL of ethanol at room temperature with occasional shaking for 24 hr. After decanting, the supernatant was filtered through Whatman filter paper, and the solvent was evaporated using a rotary evaporator maintained at 55 °C. The final yield was stored at -20°C for further analysis in tightly sealed glass vials (Mibrahim *et al*., 2013).

Phytochemical Analysis

Preliminary phytochemical analysis was conducted on the ethanolic extract of *Prosopis juliflora* seeds to detect its bioactive constituents. such as steroids, glycosides, flavonoids, and alkaloids, using standard methods as described by Harborne (Lakshmibai, 2015; Muthuraj, 2019).

Test for tannins: Mixing the extract with sodium chloride and 1% gelatin produced a precipitate, indicating tannins.

Test for saponins: Boiling 300 mg of extract in water for 2 min and shaking vigorously produced froth, confirming the presence of saponins.

Test for Phenolics: Adding 1% ferric chloride to the extract produced a bluish-black color, indicating phenolic compounds.

Test for alkaloids: Treating 1 mL of the ethanolic extract with Dragendorff's reagent produced an orange precipitate, indicating the presence of alkaloids.

Test for steroids: Treating the ethanolic extract with acetic anhydride and concentrated sulfuric acid produced a blue-green ring, confirming the presence of steroids.

Test for flavonoids: Adding dilute ammonia followed by concentrated sulfuric acid to the ethanolic extract resulted in a yellow color, indicating the presence of flavonoids.

Test for cardiac glycoside: Treating the extract with glacial acetic acid, ferric chloride, and concentrated sulfuric acid produced a brown ring at the interface, confirming glycosides.

Animals

Forty-five male Wistar albino rats (7-8 weeks; 200-250 g) were procured from TANUVAS, Madhavaram (Reg. No. 190/GO/ReBiBt-S/Re-L/2000/CPCSEA) and maintained under controlled environmental conditions (24 ± 2 °C, 50-60% humidity, 12 hr light/dark cycle) with free access to standard feed and water. Animals were acclimatized for one week before experimentation.

Evaluation of Anti-ulcer activity of *P. juliflora* seed extract using pylorus ligation method

In the pylorus ligation-induced ulcer model, Wistar albino rats were divided into five groups containing five rats each.

- **Group I** - Normal saline 2 mL/kg p.o (Negative control),
- **Group ll** - Pylorus ligated (Positive control),
- **Group III** -Omeprazole 20 mg/kg p.o (Standard drug),
- **Group IV** - *Prosopis juliflora* seed extract 250 mg/kg p.o,
- **Group V** - *Prosopis juliflora* seed extract 500 mg/kg p.o.

T. Omeprazole 20 mg/kg and seed extracts of *Prosopis juliflora* were suspended in 0.5% carboxy methyl cellulose and given using oral gavage.

Prior to drug administration, the rats were fasted for 48 hr with unrestricted access to water. In Groups III–V, the respective test and standard drugs were administered orally 1 hr before pylorus ligation. The surgical procedure was performed under strict aseptic conditions following the standard established method (Adinortey, 2015).

Rats were anesthetized through intraperitoneal injection of ketamine (80 mg/kg) and xylazine (13 mg/kg). All surgical manipulations were performed carefully to preserve the integrity of the gastric vasculature. The hair present over the incision site in the abdomen was removed with a razor for 2 cm. The skin was disinfected using 70% isopropyl alcohol. A sterile surgical site was maintained by placing a sterile drape over non-sterile parts of the animal and the surrounding area. A short surgical incision was made in the midline of the abdomen below the xiphoid process longitudinally, skin and subcutaneous tissue were retracted and the pylorus was carefully lifted and ligated. The stomach was carefully replaced in position in the abdominal cavity, the subcutaneous tissue and skin were sutured back using absorbable 4-o vicryl.

Following pylorus ligation, the rats were allowed to recover and housed individually. During the postoperative period, access to water was restricted. Meloxicam 2 mg/kg body weight was given subcutaneously to relieve pain postoperatively. Rats were closely observed postoperatively every 1 hr. 4 hr after ligation, the rats were euthanized with an overdose of halothane. The stomachs were carefully excised and opened along the greater curvature to assess gastric lesions. Gastric contents were collected, and the following parameters were evaluated: ulcer index, percentage of ulcer protection, gastric pH, free acidity, total acidity, and gross morphological changes of the gastric mucosa.

Ulcer Scoring

Gastric lesions were assessed according to the method described by (Adinortery, 2015). Ulcers were graded using the following scale: 0 – normal colored stomach; 0.5 – red coloration; 1 – spot

ulcer; 0.5 – hemorrhagic streak; 2 – deep ulcer; and 3 – perforation. The mean ulcer score for each rat was calculated and expressed as the ulcer index (UI). The mean ulcer score for each rat was expressed as an ulcer index (Pandey, 2023). The percentage of ulcer protection was determined using the formula given below:

% Protection= [(Control mean ulcer index-Test mean ulcer index)/Control mean ulcer index] x 100

Gastric pH

The pH of gastric juice was determined by dipping the electrodes of pH meter in a beaker containing gastric contents (Pandey, 2023).

Total and free acidity

Gastric juice (1 mL) was transferred to a 100 mL conical flask, and 2-3 drops of Topfer's reagent were added. The solution was titrated with 0.01 N NaOH until the red color disappeared and a yellow-orange hue appeared, representing free acidity. Phenolphthalein (2–3 drops) was then added, and titration was continued until a faint red color reappeared, indicating total acidity. The acidity was calculated using the formula (Pandey, 2023):

Acidity (mEq/Litre)=Volume of NaOH x Normality of NaOHx100/0.1.

Gross Examination of Gastric Mucosa

Gastric tissue sections were examined for macroscopic changes to evaluate the anti-ulcer effects of *Prosopis juliflora* seed extract. Lesions were assessed for hemorrhage, congestion, edema, and erosion (Pandey, 2023).

Post-experimentation procedures

The carcasses were disposed of by incineration using G.J. Multiclave.

Statistical Analysis

The required sample size was calculated using the formula,

n=DF/k+1.

where DF represents the degrees of freedom and k denotes the number of sub-groups. For a DF value of 20 and five sub-groups (k = 5), five animals were allocated per sub-group.

All experimental data were expressed as Mean ± Standard deviation (SD). Statistical comparisons among groups were performed using one-way analysis of variance (ANOVA) followed by Tukey's *post hoc* test. A p-value less than 0.05 ($p < 0.05$) was considered statistically significant.

RESULTS

Phytochemical screening

Preliminary phytochemical analysis of the ethanolic extract of *Prosopis juliflora* seeds revealed the presence of various secondary metabolites, as summarized in Table 1. 5.05 g of yield was extracted using ethanol as solvent from 50 g of *P. juliflora* seeds.

Pylorus ligation-induced ulcer model

Ulcer scoring

Results showed that pylorus ligation produced ulcers in all treated rats with a mean ulcer score of 21.2±5.0, a mean ulcer severity score of 16.3±1.5, and a mean ulcer index of 3.89±0.66 in the control group, indicating the ulcerogenic effect of pylorus ligation produced by gastric acid accumulation. Oral administration of the ethanolic extract of *Prosopis juliflora* seeds at 250 and 500 mg/kg produced a marked, dose-dependent decrease in ulcer scores compared with the positive control ($p < 0.0001$). Similarly, treatment with omeprazole (20 mg/kg) significantly lowered both the mean ulcer severity score (6.8 ± 2.1) and the mean ulcer index (1.42 ± 0.36) compared to the positive control group, with p-value < 0.01, as presented in Table 2. High dose of *P. juliflora* seed extract 500 mg/kg significantly reduced the mean ulcer index to 1.8±0.39 compared to the standard drug Omeprazole 1.42±0.36, however there was no statistically significant difference observed between the treatment groups ($p = 0.6$). Pretreatment of Wistar rats with *P. juliflora* seed extract (500 mg/kg) showed 53.7% ulcer inhibition compared to 63% ulcer inhibition produced by the standard drug Omeprazole (20mg/kg) with p value<0.01 as shown in Table 2. Thus, *P. juliflora* seed extract was found to have effective ulcer healing properties.

PH and Volume of Gastric juice

Administration of *Prosopis juliflora* seed extract (500 mg/kg) resulted in a significant increase in gastric pH (4.44 ± 0.86), comparable to the effect observed with the standard drug omeprazole (20 mg/kg; 5.24 ± 0.9), with p-value < 0.01 (Table 2). Additionally, the volume of gastric juice was markedly reduced in both the omeprazole-treated group (1.98 ± 0.7) and the *P. juliflora* extract-treated group (4.42 ± 0.27) with p-value < 0.05 (Table 3).

Table 1: Phytochemical Screening.

Sl. No.	Phytoconstituents	*Prosopis juliflora* seed extract
1.	Alkaloids	Positive
2.	Flavonoids	Positive
3.	Tannins	Positive
4.	Phenols	Positive
5.	Saponins	Positive
6.	Steroids	Positive
7.	Cardiac glycosides	Positive

Free acidity and Total acidity

Prosopis juliflora seed extract (500 mg/kg) significantly decreased both free acidity (19.2 ± 1.3) and total acidity (44.0 ± 4.52) compared to the positive control, with *p*-values <0.01 and <0.05, respectively, as presented in Table 3.

Gross Examination of Gastric Mucosa

In the pylorus ligation-induced ulcer model, the control group displayed multiple petechial hemorrhages (black spots), hemorrhagic streaks, and erythema, indicative of ulcer formation (Figure 1). In contrast, the omeprazole-treated group exhibited noticeably fewer petechial spots. Treatment with *Prosopis juliflora* seed extract also resulted in a reduction of petechial lesions compared to the control group, although mild erythema was still observed.

DISCUSSION

Peptic ulcer disease (PUD) is characterized by a breach in the mucosal lining of the stomach or duodenum, which can extend into the muscularis mucosa, predominantly due to the corrosive action of gastric acid and pepsin (Aghareed, 2018). The development of PUD is generally attributed to an imbalance between mucosal defensive mechanisms and aggressive factors that compromise the integrity of the gastric lining (Oble, 2000). Despite the availability of conventional therapies, there is ongoing interest in identifying natural compounds with gastroprotective potential to serve as alternative or adjunct treatments. In this study, phytochemical analysis of the ethanolic extract of *P. juliflora* demonstrated the presence of alkaloids, tannins, phenols, quinines, flavonoids and anthraquinones using standard methods, which were similar to the study done by (Singh *et al.*, 2012) 5.05 g of yield was extracted using ethanol as solvent from 50 g of *P. juliflora* seeds, which was similar to the study done by (Singh *et al.*, 2012).

Ethanol was chosen as the solvent as the yield of plant extract from all parts (leaves, pods, seeds) was high using ethanol, which serves as an efficient solvent for the extraction of both polar and nonpolar compounds (Wamburu, 2013). The seeds of *P. juliflora* are rich sources of alkaloids, flavonoids, quinins, and anthraquinone, which significantly suppresses H^+K^+ATPase activity as reported in the study done by (Gobinath *et al.*, 2014). In addition, *P. juliflora* also exhibits significant anti-inflammatory (Sivakumar *et al.*,) antioxidant (Lakshmibai *et al.*,) and antimicrobial properties (Ahmad *et al.*, 2015).

The accumulation of gastric juice induced by pylorus ligation in Wistar rats compromises mucosal defenses, resulting in gastric mucosal erosion and the development of ulcers, perforation, and bleeding. The gastric acid accumulation in pylorus ligation is caused by stimulation of the vagus nerve due to activation of pressure receptors in the gastric antral mucosa, and the interference with the mucosal blood flow contributes to significant ulcerations (Rakesh, 2011). Any agent that inhibits gastric acid

Table 2: Effect of *Prosopis juliflora* seed extract on ulcer severity score and ulcer index.

Sl. No.	Groups	Total Ulcer score	Total Ulcer Severity Score	Ulcer index	% Ulcer Inhibition
1.	Normal Control	0.0±0	0.0±0	0.0±0	100
2.	Disease Control	21.2±5.01	16.3±1.52	3.89±0.66	-
3.	Omeprazole 20 mg/kg	6.8±2.28**	6.8±2.10**	1.42±0.36**	63.49
4.	*P. juliflora* 250 mg	9.4±2.88**	9±1.69**	1.92±0.28**	50.64
5.	*P. juliflora* 500 mg	8±1.58**	9.2±2.70**	1.8±0.39**	53.72

Values are expressed as Mean±S.D, n=5, *p<0.05, **p<0.01, as compared to disease control using one-way ANOVA followed by *Post hoc* Tukey's test

Table 3: Effect of *Prosopis juliflora* seed extract on acid secretary parameters.

Sl. No.	Groups	Volume of Gastric Juice	PH	Free acidity	Total acidity
1.	Normal Control	2.04±0.46	3.26±0.55	15.8±4.14	29.8±6.14
2.	Disease Control	5.82±0.38	2.8±0.25	30.4±4.15	57.0±7.68
3.	Omeprazole 20 mg/kg	1.98±0.17**	5.24±0.9**	17.6±4.61**	38.2±9.28**
4.	*P. juliflora* 250 mg	3.98±0.24**	4.1±0.4*	21.6±3.1**	43.8±4.86*
5.	*P. juliflora* 500 mg	4.42±0.27**	4.44±0.86**	19.2±1.3**	44.0±4.52*

Values are expressed as Mean±S.D, n=5, *p<0.05, **p<0.01, as compared to disease control using one-way ANOVA followed by *Post hoc* Tukey's test.

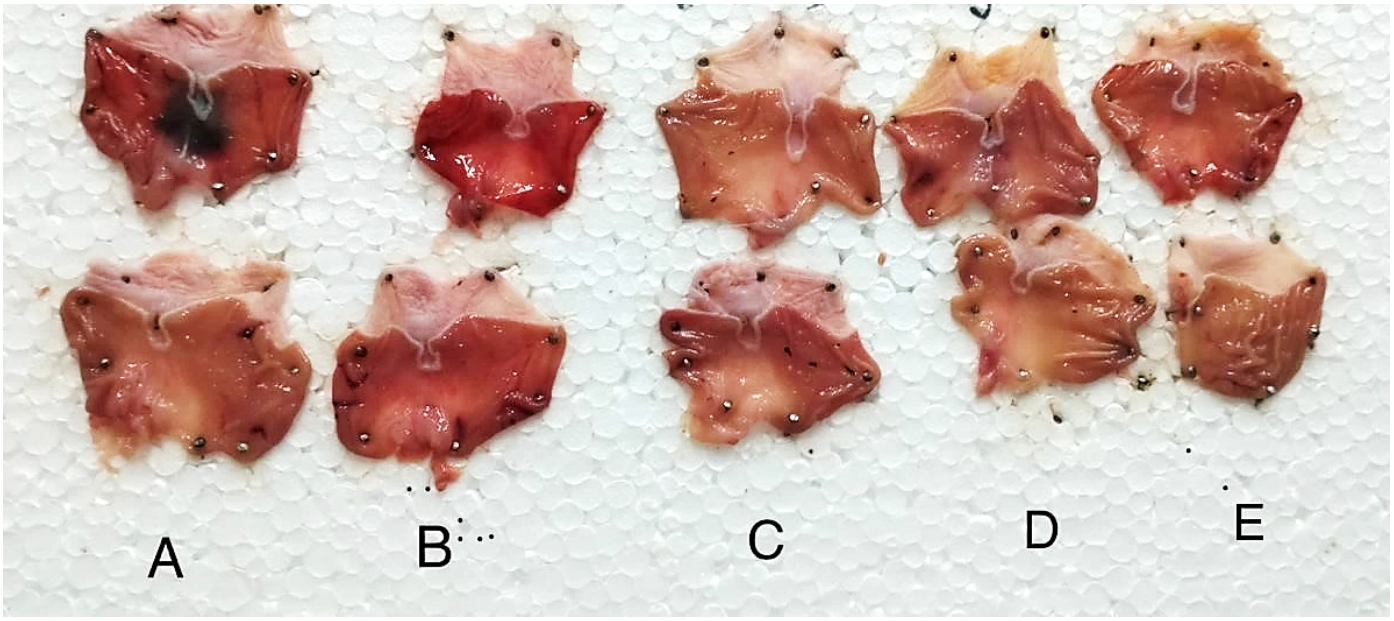

Figure 1: A and B - Pylorus ligated control: marked ulcers along with hemorrhagic streaks, petechial spots, and erythema were observed. C-20 mg/kg omeprazole-mild injuries were observed in the gastric mucosa as compared to the pylorus ligated control group D-250 mg/kg ethanolic extract of *P. juliflora*: less conspicuous petechial spots. E-500 mg/kg *P. juliflora*: significantly reduced gastric mucosal damage with less conspicuous petechial spots compared to control.

secretion and increases mucus production is effective in pylorus ligation-induced ulcers. This study shows that *P. juliflora* seed extract prevents ulcers by its antisecretory and cytoprotective effects.

In this study, administration of the ethanolic extract of *P. juliflora* seeds at doses of 250 and 500 mg/kg resulted in a significant reduction in both the mean ulcer score and ulcer severity index in pylorus ligation-induced gastric ulcers in Wistar rats, compared to the positive control group. There was also a significant reduction in gastric acid volume, free and total acidity in all the treatment groups. The extract markedly raised the pH of gastric secretions, exhibiting efficacy comparable to the reference drug, omeprazole (20 mg/kg). In this study treatment with *P. juliflora* seed extract at a dose of 500 mg/kg conferred 53.7% gastroprotective effect, while the standard reference drug, omeprazole (20 mg/kg), exhibited 63% protection, indicating a potent antiulcer activity. Treatment with *P. juliflora* seed extract resulted in a significant reduction in the ulcer index compared to the ulcerated control group. Both *P. juliflora* and omeprazole (20 mg/kg) produced a marked decrease in ulcer index, indicating comparable anti-ulcer efficacy.

CONCLUSION

The present study demonstrates that the crude ethanolic extract of *Prosopis juliflora* seeds is rich in bioactive phytoconstituents, including flavonoids and alkaloids, which exhibit significant anti-ulcer activity, as evidenced by the marked reduction in pylorus ligation-induced gastric ulcers. These findings suggest that *Prosopis juliflora* possesses significant gastroprotective potential and may serve as a valuable natural source for the development of novel anti-ulcer agents. Further pharmacological and mechanistic studies are warranted to substantiate its therapeutic efficacy and safety profile.

ACKNOWLEDGEMENT

All authors are thankful to Chettinad Hospital and Research Institute and Chettinad Academy of Research and Education for facilitating the conduct of this research.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICS APPROVAL

The study was conducted following approval of the Institutional Animal Ethics Committee (IAEC), following CCSEA Guidelines. (IAEC approval No. IAEC3/ Proposal:87/ A.Lr.63/ Dt:22/8/2022).

ABBREVIATIONS

PUD: Peptic Ulcer Disease; **HCL:** Hydrochloric Acid; **NSAIDs:** Nonsteroidal Anti-Inflammatory Drugs; **COX:** Cycloxygenase PKC-Protein Kinase C; **cAMP:** Cyclic Adenosine Monophosphate; **CAG-PAI:** Cytotoxin-Associated Gene Pathogenicity Island.

SUMMARY

This study exhibits the anti-ulcer activity of *P. juliflora* seed extract at doses of 250 mg/kg and 500 mg/kg in terms of significant reduction in mean ulcer severity score, total acidity and free acidity. *P. juliflora* seed extract shows 53.7% ulcer protection

in pylorus ligation-induced gastric ulcer model in wistar rats. Phytochemical screening indicates the presence of flavonoids and alkaloids with potent anti-ulcer activity.

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Cite this article: Nesam VD, Radhakrishnan A, Raj K, Devi S. Evaluation of anti-ulcer activity of ethanolic extract of *Prosopis juliflora* seeds in Wistar albino rats. Pharmacogn Res. 2026;18(2):519-25.