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Anti-ulcer Activity of Berberine Isolated from *Berberis aristata* in Wistar Albino Rats

Ashwini S. Gawade[1*], Tanuja N. Bhalerao[1], Prachi D. Bhor[1], Nupur A. Barve [1], Anshuvar A. Dhainje[1], Sanika P. Kokate[2], Snehal A. Mahadar [2], Dr. Kiran C. Mahajan[2], Dr. Ganesh Y. Dama[3]

[1]Department of Pharmacology, SGMSPM's Sharadchandra Pawar College of Pharmacy, Dumbarwadi (Otur), Tal-Junnar, Dist-Pune, Maharashtra, India, 401504.

[2]Department of Pharmaceutics, SGMSPM's Sharadchandra Pawar College of Pharmacy, Dumbarwadi (Otur), Tal-Junnar, Dist-Pune, Maharashtra, India, 401504.

[3]Department of Pharmacognosy, SGMSPM's Sharadchandra Pawar College of Pharmacy, Dumbarwadi (Otur), Tal-Junnar, Dist-Pune, Maharashtra, India, 401504.

Corresponding author:

Ashwini S. Gawade,

Department of Pharmacology,

SGMSPM's Sharadchandra Pawar College of Pharmacy, Dumbarwadi (Otur), Tal-Junnar, Dist-Pune, Maharashtra, India, 401504.

E-mail id: gawadeashwini15@gmail.com

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KEYWORDS

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Abstract

Peptic ulcer disease (PUD) is a multifactorial gastrointestinal disorder associated with excessive gastric acid secretion, oxidative stress, Helicobacter pylori infection, alcohol consumption, and prolonged use of non-steroidal anti-inflammatory drugs. Herbal compounds possessing antioxidant and cytoprotective properties have gained attention as safer alternatives for ulcer management. The present study investigated the anti-ulcer activity of berberine isolated from Berberis aristata using an ethanol-induced gastric ulcer model in Wistar albino rats. Acute oral toxicity studies were conducted according to OECD guideline 425, and berberine was found to be safe at the selected dose range. Animals were divided into five groups consisting of normal control, ethanol control, standard treatment with omeprazole (20 mg/kg), berberine low dose (50 mg/kg), and berberine high dose (100 mg/kg). Gastric parameters including ulcer index, percentage ulcer inhibition, gastric volume, gastric pH, and total acidity were evaluated. Histopathological examination of gastric tissues was also performed. Berberine treatment significantly reduced ulcer index and gastric acidity while improving gastric pH and mucosal integrity compared with the ethanol control group. The high-dose berberine group showed gastroprotective activity comparable to omeprazole. Histopathological findings further confirmed reduced mucosal erosion and inflammation in berberine-treated animals. The study demonstrates that berberine possesses significant anti-ulcer activity, possibly mediated through antioxidant and cytoprotective mechanisms.

Introduction:

Peptic ulcer disease is a chronic gastrointestinal disorder characterized by disruption of the gastric or duodenal mucosal lining due to an imbalance between aggressive and defensive factors of the gastrointestinal tract[1].

Aggressive factors include gastric acid, pepsin, ethanol, Helicobacter pylori infection, and non-steroidal anti-inflammatory drugs[2] whereas defensive factors include mucus secretion, bicarbonate production, prostaglandins, and mucosal blood flow [3].

Globally, peptic ulcer disease affects millions of individuals and remains a significant healthcare burden[2]. Although proton pump inhibitors and H2 receptor antagonists are widely used for treatment, long-term administration may produce adverse effects, recurrence, and drug interactions[3]. Therefore, there is growing interest in plant-derived bioactive compounds with improved safety profiles and therapeutic efficacy[4].

Berberis aristata, commonly known as Daruharidra, is an important medicinal plant extensively used in Ayurvedic medicine for the treatment of inflammatory disorders, infections, liver diseases, and gastrointestinal conditions[5]. Berberine, the principal isoquinoline alkaloid present in Berberis aristata, exhibits antioxidant, antimicrobial, anti-inflammatory, antidiabetic, and gastroprotective properties[6].

Previous studies have demonstrated the anti-ulcer potential of several medicinal plants and alkaloids in experimental ulcer models[2]. However, limited experimental evidence is available regarding the gastroprotective activity of isolated berberine from Berberis aristata in ethanol-induced gastric ulceration[7].

Therefore, the present study was designed to evaluate the anti-ulcer activity of berberine isolated from Berberis aristata in ethanol-induced gastric ulcer models in Wistar albino rats[6].

Materials and Methods:

Plant Material and Chemicals:

Berberis aristata root extract containing berberine was procured from a certified herbal supplier and used for the experimental study[5]. Omeprazole was used as the standard anti-ulcer drug[8]. Analytical grade chemicals and reagents including ethanol, hydrochloric acid, sodium hydroxide, chloroform, diethyl ether, ferric chloride, lead acetate solution, and sulfuric acid were used throughout the study[9].

Experimental Animals:

Healthy Wistar albino rats weighing between 150–220 g were selected for the study[10]. Animals were maintained under standard laboratory conditions with controlled temperature and humidity and were provided standard pellet diet and water ad libitum[8]. The animals were acclimatized to laboratory conditions before initiation of the experiment[9].

Acute Oral Toxicity Study:

The acute oral toxicity study was performed according to OECD guideline 425[11]. Female Wistar rats were fasted for 24 hours before administration of the test compound while water was provided freely[10]. Berberine extract was administered orally at a limit dose of 2000 mg/kg body weight[11]. Animals were observed continuously during the first 2 hours and periodically for 24 hours for behavioral, neurological, and autonomic responses[10]. Mortality and general physical condition were monitored daily for 14 days[11].

Experimental Design:

Thirty Wistar albino rats were randomly divided into five groups containing six animals in each group[7].

Group I: Normal control receiving distilled water [8]

Group II: Ethanol control receiving absolute ethanol [12]

Group III: Standard group receiving omeprazole (20 mg/kg) [8]

Group IV: Test group receiving berberine extract (50 mg/kg)[6]

Group V: Test group receiving berberine extract (100 mg/kg)[7]

All treatments were administered orally for seven consecutive days[10].

Induction of Gastric Ulcer:

Gastric ulceration was induced using the ethanol-induced ulcer model[12]. Animals in ulcer control and treatment groups received absolute ethanol orally to produce gastric mucosal injury[13]. After completion of treatment, animals were sacrificed by cervical dislocation and stomachs were isolated for biochemical and histopathological evaluation[10].

Evaluation of Gastric Parameters:

Ulcer Index:

The stomachs were opened along the greater curvature and examined for gastric lesions[8]. Ulcer severity was scored according to standard grading criteria, and ulcer index was calculated.[7].

Percentage Ulcer Inhibition:

Percentage ulcer inhibition was calculated using the following formula:

[(gastric mucosal injury index of model control group - gastric mucosal injury index of treatment group)/ gastric mucosal injury index of model group]× 100 [7.]

Determination of Gastric Volume and pH:

Gastric contents were collected into a graduated measuring cylinder and gastric volume was recorded[8]. The pH of gastric juice was determined using a calibrated digital pH meter[10].

Determination of Total Acidity:

An aliquot of gastric juice was diluted with distilled water and titrated against 0.01 N sodium hydroxide using phenolphthalein as an indicator[8]. Total acidity was expressed in mEq/L[7].

Histopathological Examination:

Stomach tissues were washed with normal saline and fixed in 10% formalin solution[10]. The tissues were processed, embedded in paraffin wax, sectioned, and stained with hematoxylin and eosin[12]. Histopathological changes were observed under a light microscope to evaluate mucosal damage and protective effects of treatment[13].

Results & Discussion:

Acute Toxicity Study:

No mortality or significant behavioral abnormalities were observed in animals treated with berberine at 2000 mg/kg. The findings indicated that berberine was safe within the tested dose range.

Table No 1: Acute Toxicity Study Observation

No .of animals	Dose/Treatment	Parameter		Observation
3 Female wistar rat	Plant extract BA(2000mg/kg)	Behavioral profile	Alertness	Normal
			Restlessness	Normal
			Irritability	Normal
			Fearfulness	Normal
		Physical state	Lacrimation	Normal
			Loss of appetite	Normal
			Salivation	Normal
		Mortality		All three animals were survive

Effect of Berberine on Ulcer Index:

Ethanol administration produced severe gastric ulceration in the control group with a high ulcer index value. Treatment with berberine significantly reduced ulcer formation in a dose-dependent manner. The 100 mg/kg dose exhibited marked gastroprotective activity comparable to omeprazole.

Table No 2: Determination of % ulcer index, % ulcer protection, % ulcer inhibition

Group	Treatment	Ulcer Index(Mean±SEM)	% Ulcer Protection	% Ulcer Inhibition
Group I	Normal control	0.92 ± 0.12	--	--
Group II	Ethanol control	17.24 ± 1.31	--	--
Group III	Omeprazole (20mg/kg)	3.18 ± 0.36	81.85 %	81.55 %
Group IV	Berberine (50mg/kg)	7.42 ± 0.58	56.96 %	56.96 %
Group V	Berberine (100 mg/kg)	4.06 ± 0.41	76.45 %	76.45 %

Figure 1: Comparative evaluation of ulcer index (Mean ± SEM), % ulcer inhibition, and % ulcer protection in normal control, ethanol control, omeprazole-treated, and berberine-treated groups in ethanol-induced gastric ulcer rats.

Effect on Gastric Parameters:

Berberine significantly decreased gastric volume and total acidity while increasing gastric pH compared with the ethanol control group. The high-dose group showed improved gastric protection.

Table No 3: Effect of Berberine on Gastric Parameters

Group	Gastric Volume (mL)	Gastric pH	Total Acidity(mEq/L)
Normal control	1.74 ± 0.18	4.68 ± 0.21	32.84 ± 2.16
Ethanol control	6.12 ± 0.42	1.62 ± 0.12	92.48 ± 4.22
Omeprazole	2.08 ± 0.24	5.02 ± 0.18	36. 14 ± 2.74
Berberine (50 mg/kg)	3.84 ± 0.28	3.56 ± 0.16	58.22 ± 3.42
Berberine (100 mg/kg)	2.52 ± 0.20	4.22 ± 0.19	42.68 ± 2.95

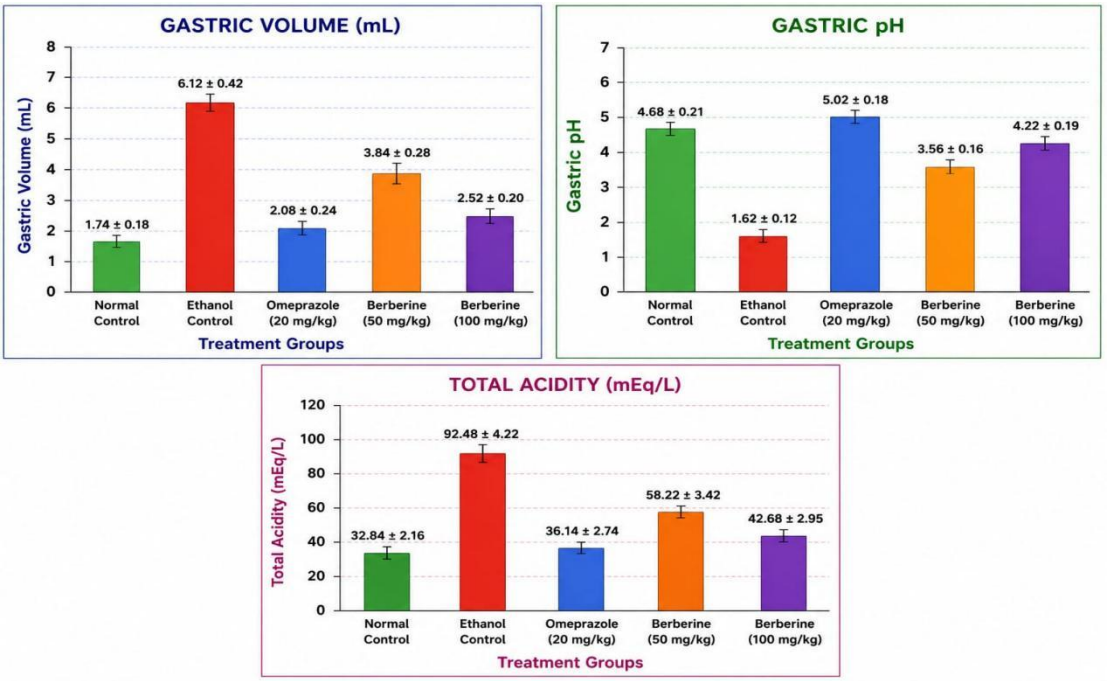

Figure 2: Comparative evaluation of gastric volume (mL), gastric pH, and total acidity (mEq/L) in normal control, ethanol control, omeprazole-treated, and berberine-treated groups in ethanol-induced gastric ulcer rats.

Histopathological Findings:

Histopathological examination of ethanol-treated gastric tissues revealed severe mucosal erosion, edema, and inflammatory cell infiltration. Berberine-treated groups showed reduced gastric damage and preservation of mucosal architecture. The protective effect was more prominent in the high-dose berberine group.

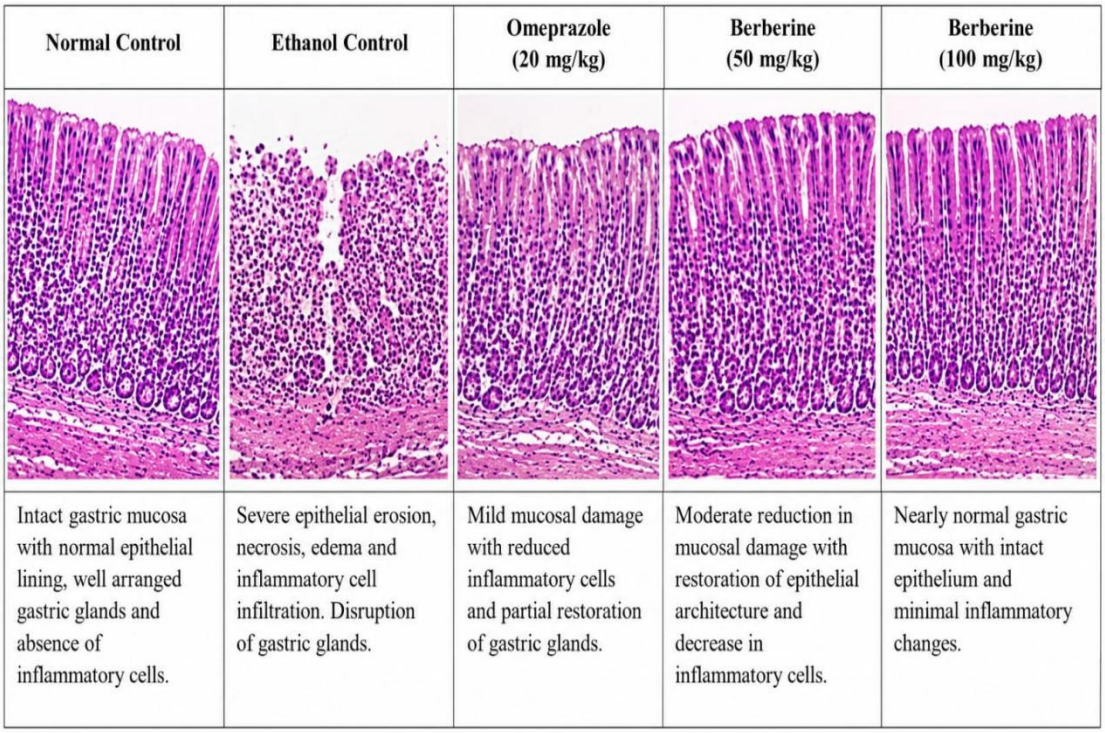

Figure 3: Histopathological examination of stomach tissue sections showing the protective effect of omeprazole and berberine against ethanol-induced gastric mucosal damage (Hematoxylin and Eosin stain, 400× magnification).

The present investigation demonstrated significant anti-ulcer activity of berberine isolated from Berberis aristata against ethanol-induced gastric ulcers in Wistar albino rats. Ethanol-induced ulceration is a commonly employed experimental model that produces gastric mucosal injury through oxidative stress, inflammation, lipid peroxidation, and excessive acid secretion.

The reduction in ulcer index observed in berberine-treated groups indicates substantial gastroprotective activity. The high-dose berberine group demonstrated better efficacy than the low-dose group, suggesting a dose-dependent protective effect. Improvement in gastric pH and reduction in gastric acidity further support the antisecretory action of berberine.

Berberine possesses antioxidant and anti-inflammatory activities that may contribute to gastric mucosal protection. Previous reports have demonstrated that berberine modulates oxidative stress pathways, suppresses inflammatory mediators, and improves tissue healing.

Conclusion:

In conclusion, the present study revealed that berberine isolated from Berberis aristata exhibited significant gastroprotective and anti-ulcer activity in ethanol-induced gastric ulcer models in Wistar albino rats. Treatment with berberine notably decreased ulcer formation, gastric acidity, and gastric volume while improving gastric pH and preserving the integrity of the gastric mucosa in a dose-dependent manner. The higher dose of berberine showed protective effects comparable to the standard drug omeprazole, which was further supported by histopathological findings showing reduced mucosal damage and inflammation. The observed activity may be associated with the antioxidant, anti-inflammatory, and cytoprotective properties of berberine. These findings indicate that berberine has potential as a natural and safe therapeutic agent for the management of peptic ulcer disease, although further pharmacological and clinical studies are required to confirm its effectiveness and mechanism of action.

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