RESEARCH ARTICLE

Evaluation of antiulcer activity of the poly-herbal combination extract

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Objective: Antiulcer activity of a Poly-Herbal Combination (PHCE) containing extracts of *Gymnema sylvestre*, *Momordiaca charantia*, *Syzygium cumini*, *Trigonella foenum*, *Psidium guajava*, *Tinospora cardifolia*, *Boerhavia diffusa*, *Coriandrum sativum*, *Andrographis paniculata*, mixture of Haritaki, Bibhitaki and Amalaki, mixture of black pepper and dry ginger and *Withania somnifera* was assessed in rats' model.

Methods: The antiulcer activity of the poly-herbal combination extract (PHCE 300 and 500 mg/kg) was tested, as well as the therapeutic efficacy of PHCE.

Findings: When the results of the evaluations were compared to the control group and standard medicine, PHCE (300 and 500 mg/Kg[-1], p.o.) dose was found to be particularly protective against ethanol-induced, indomethacin-induced and aspirin induced ulcer models.

Conclusion: *Gymnema sylvestre*, *Momordiaca charantia*, *Syzygium cumini*, *Trigonella foenum*, *Psidium guajava*, *Tinospora cardifolia*, *Boerhavia diffusa*, *Coriandrum sativum*, *Andrographis paniculata*, mixture of Haritaki, Bibhitaki and Amalaki, mixture of black pepper and dry ginger and *Withania somnifera* were discovered to have antiulcer action in PHCE. According to the findings, the PHCE can be employed in the treatment and management of stomach ulcer illness.

Key Wwords: *Gastric ulcer; Ulcer index; Poly-herbal formulation; Indomethacin; Ethanol*

INTRODUCTION

People are currently taking interest towards herbal medicine due having low toxicity, low cost and ease of access to herbs. Allopathic medications, on the other hand, have a long list of drawbacks, including stomach ulcers, liver toxicity, skin rashes and other side effects [1]. The gastrointestinal condition of peptic ulcer disease is fairly frequent. It is caused by an imbalance between greater stomach acid secretion and defensive gastric mucosal protective function, as well as an irregular lifestyle. The goal of peptic ulcer treatment is to either reduce excess acid output or strengthen the protective effect of the stomach's gastric mucosal layer [2]. Commonly proton pump inhibitors and H_2 receptor antagonists' medicines are highest selling drugs in market which is using for the treatment and management of gastric ulcer disorders; however, both drugs have a high rate of recurrence, adverse effects and drug interactions [3]. Ayurvedic practitioners have employed bioactive ingredients for the treatment and management of gastric ulcer disease. Secondary metabolites of natural plants, such as flavonoids, saponins, tannins, gums and mucilage, are all bioactive substances [4]. To treat ulcers, natural medications are very effective, safer and cost-efficient [5]. In rats, the antiulcer activity of a Poly-Herbal Combination Extract (PHCE) containing leaf extracts of *Gymnema sylvestre, Momordiaca charantia, Syzygium cumini, Trigonella foenum, Psidium guajava, Tinospora cardifolia, Boerhavia diffusa, Coriandrum sativum, Andrographis paniculata*, mixture of Haritaki, Bibhitaki and Amalaki, mixture of black pepper and dry ginger and *Withania somnifera* was assessed [6]. Selected poly-herbal combinations have never been employed before by any researcher and there are currently no research publications available from a literature survey. As a result, these PHCE were chosen for the treatment and management of ulcer disease. These PHCE contain a variety of therapeutically active compounds. All of these putative bioactive compounds may have good stomach ulcer protective effect and can be utilized for the management or prevention of gastric ulcer disease, according to the hypothesis behind the selection of this combination (List of potential antidiabetic bioactive molecules having plants were given in Table 1 [7-15].

TABLE 1
List of potential antidiabetic bioactive molecules having plants

Name of plants	Phytochemicals	Pharmacological properties
Gymnema sylvestre	Flavones, anthraquinones, d-quercitol, gymnemic acid, gymnemosides, gymnemasaponins, lupeol, β-amyrin related glycosides and stigmasterol	Antidiabetic, hepatoprotective and anti-inflammatory activities, etc.
Momordiaca charantia	Triterpenoids, saponins, polypeptides, flavonoids, alkaloids and sterols	Antifertility, antiulcer, antihyperglycemic, hepatoprotective immunomodulation, antioxidant, antimutagenic, antilipolytic, etc.
Syzygium cumini	Anthocyanins, glucoside, ellagic acid, isoquercetin, kaemferol and myrecetin	Antidiabetic, throat infection, asthma, hepatoprotective, dysentery and ulcers, etc.
Trigonella foenum	Ederagin glycosides. Alkaloids such as trigocoumarin, nicotinic acid, trimethyl cou-marin and trigonelline	Antidiabetic, antioxidant, anticarcinogenic, hypocholesterolemic, hepatoprotective and immunological activities, etc.
Psidium guajava	Iso-caryophyllene, veridiflorene, farnesene, dl-limonene, δ-cadinene, α-copaene, α-humulene, τ-cadinol	Used in problems of dental, sleeping, liver, convulsion, respiratory, wound healing, pain and treatment of diabetes mellitus, etc.

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Tinospora cardifolia	Alkaloids, terpenoids, lignans, steroids and others	Reported antioxidant, hepatoprotective, anticancer, wound treatment, anticancer and immunomodulating activity, etc.
Boerhavia diffusa	Alkaloids (punarnavine), rotenoids (boeravinones A to J) and flavones	Reported used in treatment of inflammatory, stone, microbial, asthma, urine, liver and diabetic treatment, etc.
Coriandrum sativum	Linalool, α-Pinene, β-Pinene, γ-Terpinene, α-Cedrene, α-Farnasene, p-Cymene, limonene, citronellal, camphor geraniol, anethole	Reported activity against of microbial, liver toxicity, diabetic, fatty condition, convulsion, cancer and inflammation
Andrographis paniculata	Andrographolide and 14-deoxy-11,12-didehydroandrographide, etc.	It is having anti-inflammatory, liver protective and management of blood pressure property, etc.
Mixture of Haritaki, Bibhitaki and Amalaki	Flavonoids, alkaloids, phenols	Cardiovascular disease, blood pressure disease, poor liver function, large intestine inflammation, hepato protective and ulcerative colitis
Trikatu (Mixture of *Piper nigrum* (kali mirch), *Zingiber officinale* (adhrakh) and long piper longum (pippali)	Piperine, gingerols, shogaols and paradols, oleoresins and alkaloids	Bioavailability enhancer, fevers, gastric and abdominal disorders, urinary difficulties, hepato protective, neuralgia and boils etc.

MATERIALS AND METHODS

All the chemicals were analytical grade.

Collection of plant material

The plant materials like *Gymnema sylvestre, Momordiaca charantia, Syzygium cumini, Trigonella foenum, Psidium guajava, Tinospora cardifolia, Boerhavia diffusa, Coriandrum sativum, Andrographis paniculata*, mixture of Haritaki, Bibhitaki and Amalaki, mixture of black pepper and dry ginger and *Withania somnifera* were purchased from the local market of Mandsaur, Madhya Pradesh. The plant material was authenticated by pharmacognosist, Dr. Sandeep Kumar Singh. Plant materials were used for poly-herbal formulation (entral ayurvedic research Institute, Jhansi, Uttar Pradesh). Plant material was collected and washed under tap water and shade dried.

Extraction of poly-herbal formulation

Dried plant materials were coarsely grinded into a coarse powder. The coarse powder of plant material was macerated in an airtight container in 70:30 ratio of ethanol to distilled water for 15 days with regular shaking. The resulting solvent was filtered and evaporated at 40 degrees celsius using a rotary vacuum evaporator, resulting in a poly herbal hydro alcoholic crude extract of 150 grams after filtering and concentration of the residue. The extracted substance was chosen for the study of hepato protective activity and stored in a cool place for further analysis. The composition of the poly herbal materials used in the extraction process is provided below (Table 2) [16-18].

TABLE 2
Composition of herbal plants for poly-herbal extraction

Name of plants	Biological name	Quantity
Gudmar leaves	*Gymnema sylvestre*	100 gm
Karela seed	*Momordiaca charantia*	100 gm
Jamun seed	*Syzygium cumini*	100 gm
Methi seed	*Trigonella foenum*	100 gm
Amruda leaves	*Psidium guajava*	100 gm
Giloya	*Tinospora cardifolia*	100 gm
Punarnava	*Boerhavia diffusa*	100 gm
Coriander leaves	*Coriandrum sativum*	100 gm
Kalmegha leaves	*Andrographis paniculata*	100 gm
Triphala	Mixture of Haritaki, Bibhitaki and Amalaki	100 gm
Trikatu	Mixture of black pepper and dry ginger	100 gm
Aswagandha	*Withania somnifera*	100 gm

Preliminary phytochemical screening

Herbal plants comprise a diverse array of bioactive compounds, such as alkaloids, glycosides, volatile oils, tannins, saponins, flavonoids and other secondary metabolites, which are responsible for their therapeutic effects. In order to identify the presence of both primary and secondary metabolites, the extract was subjected to a series of chemical tests [19-20].

Experimental animals

The experimental protocol involving the use of male albino wistar rats weighing between 200 and 230 g was approved by the institutional animal ethics committee. The animals were housed in an animal facility that was sanctioned by the committee for control and supervision of animal experiments.

Screening models for anti-ulcer activity evaluation

Acute toxicity studies: In compliance with animal ethical guidelines, an acute toxicity study of PHCE was conducted on adult Wistar rats. Three rats were administered a single oral dose of PHCE at a concentration of 2000 mg/kg, p.o body weight. The rats were then monitored for 1, 2, 4, 8 and 24 hours for any toxicological manifestations such as changes in behavior, locomotion, convulsions or mortality. Subsequently, the rats were observed for 14 days, during which their behavior and activity patterns were closely scrutinized. No signs of toxicity were detected during the course of the study.

Antiulcer activity: To evaluate the antiulcer activity, the animals were divided into eight groups, each group consisting of six animals (n=6).

Ethanol-induced gastric ulcers model: The experimental animals were allocated into four groups, each comprising six rats. Gastric ulceration was induced by administering absolute ethanol (1 ml/200 gm) and to prevent coprophagy, the animals were placed in specially designed cages.

- Group I (Control) received 1% SCMC and ethanol.
- Group II (Standard) received 10 mg/kg of Omeprazole.
- Group III received 300 mg/kg of ethanolic polyherbal extract orally.
- Group IV received 500 mg/kg of ethanolic polyherbal extract orally.

TABLE 3
Effect of PHCE on ethanol, indomethacin and aspirin induced ulcer models

Groups	Parameter	Control	Standard control	PHCE 300 mg/kg	PHCE 500 mg/kg
Ethanol	Ulcer index	23.24 ± 0.05	8.508 ± 0.164*	14.70 ± 0.036*	7.018 ± 0.248*
	% Ulcer inhibition	-	63.42	36.74	69.8
Indomethacin	Ulcer index	23 ± 0.576	7.98 ± 0.249*	14.82 ± 0.308*	8.226 ± 0.231*
	% Ulcer inhibition	-	65.3	35.56	64.23
Aspirin	Ulcer index	21.26 ± 0.371	7.685 ± 0.248*	15.78 ± 0.208*	10.536 ± 0.223*
	% Ulcer inhibition	-	63.85	25.77	50.47

Note: Data are expressed as mean± S.E.M. (n = 6), **p < 0.01; ns: not significant *vs.* control.

Calculation of Ulcer Index (UI) and percent inhibition (% I)

- Ulcer Index (UI)=Number of ulcer in control-Number of ulcer in test/Number of animals
- Percent Inhibition (% I)=(UI of control-UI of test/UI of control) × 100

Indomethacin-induced gastric ulcers model: The experimental animals were allocated into four groups, each comprising six rats. Gastric ulceration was induced by administering absolute ethanol (1 ml/200 gm) and to prevent coprophagy, the animals were placed in specially designed cages.

- Group I (Control) received 1% SCMC and ethanol.
- Group II (Standard) received 10 mg/kg of omeprazole.
- Group III received 300 mg/kg of ethanolic polyherbal extract orally.
- Group IV received 500 mg/kg of ethanolic polyherbal extract orally.

In the pre-treatment phase, omeprazole (20 mg/kg) or an extract at a dose of 300 mg and 500 mg/kg was administered orally (gavage) to rats daily for 21 days. On the final day, these rats were given indomethacin, a single gavage of 3 mg/kg body weight for ulcer induction, 2 hours after the omeprazole or extract dose. Rats in groups 1 and 2 were respectively dosed with vehicle or indomethacin in parallel (on the final day). Prior to indomethacin oral treatment, all rats were fasted for 24 hours and kept in wide wire mesh-bottom cages to avoid coprophagia. In addition, water access was prevented for 2 hours prior to indomethacin dosing. Four hours after the indomethacin/vehicle gavage, all rats were euthanized by chloroform and their stomachs were excised (Table 3).

Aspirin induced model: The experimental animals were allocated into four groups, each comprising six rats. Gastric ulceration was induced by administering absolute ethanol (1 ml/200 gm) and to prevent coprophagy, the animals were placed in specially designed cages.

- Group I (Control) received 1% SCMC and ethanol.
- Group II (Standard) received 10 mg/kg of Omeprazole.
- Group III received 300 mg/kg of ethanolic polyherbal extract orally.
- Group IV received 500 mg/kg of ethanolic polyherbal extract orally.

The animals were administered with aspirin (150 mg/kg) orally to induce ulcers, followed by 45 minutes of PHCE and the administration of omeprazole to selected animal groups as a standard medication for ulcer protection. After 5 hours, the animals were euthanized and lesions in the gastrointestinal mucosa were observed and recorded using a microscope (10X). Gastric mucosa samples were subjected to histopathological tests and the ulcer index was calculated to determine the ulcer score (Figure 1).

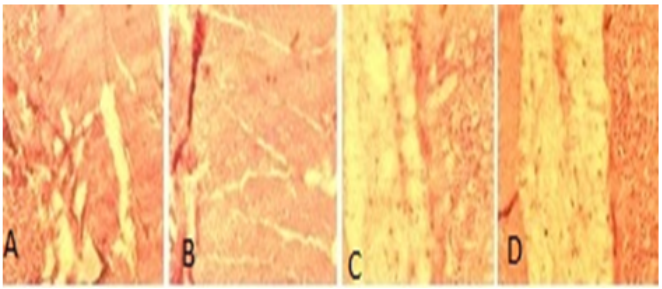

Figure 1: Ethanol-induced scenario, histopathology of rat stomach mucosa samples. Note: A) Negative control, which indicates that the structure is normal. B) Sucralfate 100 mg/kg as a standard control. C) PHCE (300 mg/kg) shows the usual structure. D) PHCE (500 mg/kg) indicates that the structure is normal.

Histopathology: After collecting the gastric mucosa contents from each group of animals and cutting them into small pieces, the stomachs were cut into little pieces. Small stomach fragments were encased in paraffin wax

Albino wistar rats weighing between 180 gm-250 gm were individually housed in separate cages, with each cage containing six rats. The animals were fasted for 24-36 hours with unrestricted access to water before ethanol administration. Peptic ulcers were induced by administering total ethanol (1 ml/200 gm). Group 1 was administered only 1% SCMC, while group 2 received regular omeprazole at a dose of 10 mg/kg. The third and fourth groups were given the poly-herbal hydro alcoholic extract of the polyherbal formulation at doses of 300 mg and 500 mg/kg, respectively. The treatment was repeated for seven days to evaluate the antiulcer activity of the compounds. On the 8th day, the drugs were orally administered 30 minutes before the administration of ethanol. After 6 hours, the rats were anesthetized with ether and the stomach and greater curvature were incised. The stomach was then gradually cleaned under fresh water and the inner surface was checked for any signs of ulceration. The frequency and rating of ulcers were determined microscopically using a 10X magnification lens. The mean ulcer score for each animal was expressed as the ulcer index (Table 3).

and implanted. Microtome was used to cut 5 m thick stomach sections, which were then mounted on glass slides using normal procedures. The tissues on the mounted slide were stained with Hematoxylin Eosin stain (H and E) and the resulting slides were examined under a light microscope.

Statistical analysis: The data was presented as mean SEM. One-way ANOVA multiple comparison tests were used to analyze the results.

RESULTS AND DISCUSSION

Phytochemical screening: Carbohydrates, proteins, saponins, flavonoids, tannins and glycosides were discovered in preliminary phytochemical screening of the PHCE. The outcomes are listed in the table below (Table 4).

TABLE 4
Results of preliminary phytochemical screening

Sr. no	Phyto-constituent category	Inference
1	Carbohydrate	Positive
2	Cardiac glycoside	Negative
3	Flavonoids	Positive
4	Alkaloids	Positive
5	Tannin and phenolic component	Positive
6	Protein	Positive
8	Saponin	Positive

Pharmacological activities

Acute toxicity studies: Acute toxicity studies under observation study no signs and symptoms of toxicity were found during the acute toxicity study of PHCE after oral administration of dose up to 3000 mg/kg.

Effect of Poly-Herbal Formulation (PHCE) on ethanol-induced gastric ulcers: Acute toxicity studies under observation during the acute toxicity investigation of PHCE following oral administration of doses up to 3000 mg/kg, no signs or symptoms of toxicity were seen.

Gastric lesions induced by ethanol: PHCE concentrations of 300 and 500 mg/kg were used in the experiments. The ulcer index and % inhibition were used to calculate the anti-ulcer (ulcer preventive) activity. In comparison to control, an acute dose of PHCE 300 and 500 mg/kg of body weight inhibited ulcers by 63.42 and 69.80 percent, respectively and the ulcer index was found to be 14.700.036 and 7.0180.248. The anti-ulcer activity of the PHCE was comparable to that of a conventional medication. The conventional sucralfate (100 mg/kg p.o.) significantly (P0.05) reduced ulcer formation, with an ulcer inhibition rate of 76.29 percent and a UI of 5.5080.164, which was similar to the PHCE 500 mg/kg treated group. The PHCE-treated group had a UI of 7.0182.48 and 69.80 percent inhibition. These results indicated that PHCE was most potent as compared to other treated groups; results are shown in Table 3 and Figure 1.

Effect of Poly-Herbal Formulation (PHCE) on indomethacin-induced gastric ulcers: *Gymnema sylvestre, Momordiaca charantia, Syzygium cumini, Trigonella foenum, Psidium guajava, Tinospora cardifolia, Boerhavia diffusa, Coriandrum sativum, Andrographis paniculata*, mixture of Haritaki, Bibhitaki and Amalaki, mixture of black pepper and dry ginger and *Withania somnifera* were studied for their pharmacological potential. When PHCE and standard misoprostol (100 mcg/kg, p.o) were compared to control, the ulcer index PHCE and standard misoprostol (100 mcg/kg, p.o) revealed a substantial reduction in ulcer index. The conventional misoprostol considerably (P0.05) reduced ulcer formation, with a 65.30 percent ulcer inhibition rate and a UI of 7.980.249, which was very close to the PHCE 500 mg/kg treated group. The PHCE group had a 64.23 percent inhibition rate and a UI of 8.2260.231.

The anti-ulcer activity of PHCE was investigated and it was found to be suitable for the treatment and management of GIT problems. The outcomes of the investigation backed up PHCE's pharmacological potential. The purpose of the trial was to see if PHCE could protect against gastric/peptic ulcers, the results are shown in Table 3.

Aspirin induced model: The emergence of stomach lesions was suppressed in a dosage-dependent manner when PHCE was given orally at various dose levels. When compared to the negative control group, a higher dose of PHCE at 500 mg/kg b.w. showed 52.87 percent ulcer prevention with a higher dose, there was an increase in ulcer prevention. The conventional omeprazole considerably (P0.05) reduced ulcer formation, with an ulcer inhibition rate of 63.85% and a UI of 7.6850.248, which was similar to the PHCE 500 mg/kg treated group. The PHCE-treated group had a UI of 8.2260.231 and a 59.96 percent inhibition.

Histological results: The antiulcer efficacy of PHCE was further validated by histopathological study of rat stomach samples, which showed that it reduced congestion, oedema and bleeding in the gastric mucosa (Figure 1). The protective effect of PHCE was investigated using histopathology of an isolated stomach. All animal groups of lesion and epithelial erosion had their histological results examined. The histological tissue observation investigation revealed that PHCE was shown to be significantly ulcer protective when compared to conventional treated animals. The number and severity of stomach ulcers in treated mice treated with PHCE extract (500 mg/kg) were compared to control and PHCE (300 mg/kg) in untreated animals and after standard medication, histopathology results showed that PHCE 500mg provided the highest protection. The outcomes are listed in the Table below (Figure 1).

PHCE 500 mg has been shown to have strong ulcer protective properties. The phytochemical analysis of PHCE revealed that it includes a wide range of beneficial chemicals. The inclusion of phenolics, flavonoids, saponins and tannins in this PHCE may explain its anti-ulcer preventive properties. In general, we've discovered a slew of anti-ulcer bioactive chemicals that have been previously identified in studies. Ethanol is often used to produce gastric ulcers in albino rats. When ethanol reaches the location of gastric mucosa, it causes vascular injury and triggers cell necrosis *via* increasing mucosal permeability and the release of bioactive chemicals. The effects of PHCE and standard on ethanol- and stress induced gastrointestinal damage were studied. PHCE reduced gastrointestinal damage caused by ethanol and stress. In Hcl-ethanol; indomethacin; and aspirin produced ulcer models, PHCE showed the best gastro-protection of ulcers at different doses of 300 mg/kg and 500 mg/kg, respectively. In a Hcl-ethanol generated ulcer model at 500 mg/kg, the best meaningful protective ulceration inhibition findings were determined to be 69 percent. The outcomes of the stomach mucosa tissue protection were validated by histological investigation. The outcomes are listed in the Table below (Figure 1).

A stomach ulcer is caused by anti-Helicobacter pylori bacteria. It has been observed that several bioactive substances such as genistein, hesperidin, pancreatin, irisolidone and cabreuvin have anti-ulcer action. The presence of flavonoids in PHCE could explain its gastroprotective properties. The flow of sodium and potassium ions in the lumen is increased by flavonoids, which improves ulcer healing. The tannin concentration of PHCE-500 mg had an astringent effect and helped ulcers heal faster. Both of these phytoconstituents work together to improve anti-ulcer healing and protection and tannins may compete with H_2 receptor antagonists. PHCE-500 mg lowers gastric secretion and prevents acid secretion triggered by histamine production and gastric hormone in the same way that H_2 receptor antagonists like Ranitidine do. The anti-ulcer activity of PHCE could be mediated in part by its anti-acid secretion and protective effect, according to the results of the experiments. Thus, the anti-secretary and cytoprotective effect of PHCE extract can be explained in part by the presence of flavonoids and tannins. Finally, the findings of the anti-ulcer activity investigation revealed that PHCE-500 mg can help with the treatment and management of gastric and intestinal ulcers.

CONCLUSION

It has been established that peptic ulcers are primarily caused by an imbalance between protective and destructive biological components. Gastric ulcers, in particular, are attributed to the misalignment of protective biological components. Based on the results of the ulcer model investigation, PHCE has demonstrated potential as a therapeutic agent in various ulcer-induced models. The cyto-protective properties of PHCE are believed to stem from the presence of bioactive chemicals with potentially therapeutic effects. However, a comprehensive investigation is necessary to elucidate the actual mechanism responsible for PHCE's antiulcer efficacy.

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CONFLICT OF INTEREST

All of the authors disclose that they have no competing interests.

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