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GASTROPROTECTIVE EFFECTS OF POLYHERBAL AYURVEDIC FORMULATION: AN AVIPATTIKAR CHURNA

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ABSTRACT

The current study was undertaken to investigate the effect of the avipattikar churna against experimental gastric ulcers. Pretreatment of aqueous extract of churna at the dose of 540 mg/kg, for seven days was studied against ethanol-induced gastric mucosal damage. The aqueous extract showed significant reduction in gastric ulceration against ethanol-induced gastric mucosal damage. The results were comparable with omeprazole (reference standard). In the ethanol-induced gastric ulcer model, treatment with both the aqueous extract and omeprazole showed significant antioxidant activity as evident from the reduction in the extent of lipid peroxidation that was measured in terms of malondialdehyde (MDA), when compared with the control group. Pretreatment of aqueous extract of churna for 7 days in 6-h pylorus-ligated animals, showed significant reduction in the ulcer index. Furthermore, in the pylorus ligation model, significant reduction ($p<0.05$) was observed in total acidity, total acid output, pepsin activity, and pepsin output, along with a significant rise in the total carbohydrate to protein ratio (reflecting mucin activity) when compared with the control group. The mechanism of its antiulcer activity could be attributed to a decrease in gastric acid secretory and antioxidant activities leading to gastric cytoprotection. The ulceroprotective activity of aqueous extract of Avipattikar churna could be mediated through its antioxidant activity, vasodilatation, and gastric cytoprotection.

Keywords: Cytoprotective, Antioxidant activity, stress ulcer, gastric ulcer, avipattikar churna

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INTRODUCTION

Modern scientific evaluations of Ayurvedic formulations are carried out with a view to validating the traditional uses of herbal medicines to explore the unexplored wonders which lay in the depth of traditional systems of medicine. "Avipattikar churna" is a polyherbal Ayurvedic medicine used as remedy for hyperacidity, indigestion, anorexia, urinary retention, constipation and piles[1]. According to Ayurvedic physicians ulcer formation is due to improper digestion of food. Though Avipattikar churna is claimed to decrease hyperacidity thereby being useful in curing peptic ulcers, there is no scientific proof to support this claim[1, 2]. It Gives relief from sour and bitter eructation, inflammation in throat, chest and abdomen, sour salivation, bilious vomiting distaste, nausea and constipation[2]. Pure and high quality range of Avipattikar churna that is a dynamic combination of herbs designed to strengthen agni (digestive fire) without aggravating pitta. This unique formula removes heat from the intestines, providing an antidote for pungent foods. It is generally known to maintain normal level of acidity. Avipattikar churna was prepared as per Ayurvedic Formulary of India. Avipattikar churna has been selected for the current study. However, Avipattikar churna has not been thoroughly evaluated for pharmacological activity. Therefore, the current work aimed to study the effect of aqueous extract of Avipattikar churna on experimentally induced gastric ulcer models.

MATERIAL AND METHODS

Procurement of plant material: Avipattikar churna consists of fourteen ingredients viz., *Zingiber officinale, Piper nigrum, Piper longum, Terminalia chebula, Terminalia bellerica, Embelica officinalis, Cyperus rotundus, salt (vida lavana), Embelia ribes, Elettaria cardamomum,Cinnamomum tamala, Syzgium aromaticum, Operculina terpethum, and Saccharum officinarum*. All ingredients which are used in Avipattikar churna, whose botanical names, local name, and parts used, have been given in formulation. All these ingredients were procured from the herbal supplier of Ahmedabad city of Gujarat. The authentification were done by the taxonomist, School of science, Botany department, Ahmadabad, India, and a voucher specimen (PHCOG/05/09) was deposited in the Department of Pharmacognosy and Phytochemistry, KBIPER, Gandhinagar, India. These ingredients were to be used for the present study.

Preparation of Avipattikar churna: The Avipattikar churna were prepared in the department of Pharmacognosy and Phytochemistry by using authenticated raw materials as per the procedure given in Ayurvedic Formulary of India (Anonymous 2003). All the ingredients viz., *Zingiber*

officinale, Piper nigrum, Piper longum, Terminalia chebula, Terminalia bellerica, Embelica officinalis, Cyperus rotundus, salt *(vida lavana), Embelia ribes, Elettaria cardamomum, Cinnamomum tamala, Syzgium aromaticum, Operculina terpethum,* and *Saccharum officinarum* were powdered separately, passed through 60 # sieve and then mixed together in proportions as specified in Ayurvedic Formulary of India.

Preparation of the extract

In-house preparation of Avipattikar churna (100 g) was extracted with 2: l of chloroform water (1: 1000) by maceration. The filtered extract evaporated under vacuum gave a dry yield of 18.4% (w/w) and was stored in air tight container until further use. The dose for experimental study was calculated by extrapolating the human dose to animal dose to animal dose based on the surface area ratio.

Drugs and chemicals:

Omeprazole was obtained from Zydus Research Centre, Ahmedabad, India. All different organic solvents and reagents used for the current study were of analytical grade (AR) and obtained from S.D. Chem. Pvt. Ltd. (Mumbai, India). Fresh drug solutions were prepared in 1% carboxy methyl cellulose (CMC) and were administered orally.

In-house preparations of Avipattikar churna have been standardized on the basis of organoleptic characters, physical characteristics and Physico-chemical properties. The set parameters were found to be sufficient to evaluate the churna and can be used as reference standards for the quality control/quality assurance laboratory of a Pharmaceutical house.

Physicochemical parameters of Avipattikar churna:

The Avipattikar churna were subjected to various physiochemical tests such as loss on drying, total ash, acid insoluble ash and extractive values using standard procedures.

Phytochemical analysis of Avipattikar churna:

Preliminary phytochemical analyses were performed to detect the presence of various phytoconstituents like tannins, flavonoids, saponins, sterols, alkaloids, and glycosides, anthraquinone, carbohydrates, etc.

Animals:

Wistar albino rats (Zydus Cadila Limited, Ahmedabad, India) of either sex weighing 175–225 g were selected for the current study. Animals were fed a standard chow diet and water that was freely available and maintained under standard conditions of a 12-h dark-light cycle, 60 ± 10% humidity, and a temperature of 21.5 ± 1^0C. Coprophagy (and thus re-ingestion of any drug) was

prevented by keeping the animals in cages with gratings on the floors. The distribution of animals in the groups, the sequence of trials, and treatment allotted to each group was randomized. Freshly prepared solutions of drugs or chemicals were used throughout the study. All experiments complied with university guidelines for animal experimentation. Throughout the entire study period, the rats were monitored for growth, health status, and food intake capacity to be certain that they were healthy. Utmost care was taken to ensure that animals were treated in the most humane and ethically acceptable manner. The animals were sacrificed with an overdose of ether anesthesia after the completion of the experiments. The stomachs were removed, opened along the greater curvature, washed with saline, and examined using a 6.4 binocular magnifier. Lesions were assessed by two unbiased observers.

Methodology:

The animals were divided into following groups of six.

Group I (control): Rats received only aqueous suspension of 1% CMC vehicle with respect to the individual ulcerogenic procedure.

Group II (drug treatment): Rats received aqueous extract (540 mg/kg,p.o.) of churna for 7day.

Group III: Rats received standard omeprazole (20mg/kg, p.o.) for 7 day.

Ethanol-induced gastric mucosal damage

The drug extracts in 1% CMC solution were administered orally once daily for seven consecutive days to respective groups. On seventh day gastric lesions were induced by absolute ethanol (0.2ml, p.o.) was administered 1 hr after respective treatment and control groups in 24 h fasted rats [3]. Animals were sacrificed 2 h after the ethanol administration, and gastric lesions were measured in terms of ulcer index (UI) determined[4]. Each lesion of the stomach was measured along the greatest length and breath. For circular lesions, the diameter was measured and area calculated. In case of petechies, five of them were considered to be equivalent to 1mm2 of ulcerated area. The total area of the stomach mucosa and that of ulcerated mucosa were calculated: Ulcer index =10/X, where X= (Total mucosal area)/ (Total ulcerated area). Further, the effect of drug administration on lipid peroxide levels was evaluated. The stomach of each rat in each case was dissected out quickly, blood blotted off, washed with ice-cold saline and a 10% homogenate was prepared in phosphate buffer (10mM, pH 7.4). The homogenates were centrifuged at 3000 rpm at 0^0C for 15 min using Remi C-24 high speed cooling centrifuge (Japan). The clear supernatant was used for the estimation of lipid peroxidation (MDA content).

The assay for microsomal lipid peroxidation was carried out[5]. The protein concentration in all samples was also determined[6].

Pylorus-ligation (PL) model:

On seventh day, one hour after drug administration pylorus was ligated. Rats fasted for 24 h were anesthetized with ether, and a portion of abdomen was opened by a small midline incision below the xiphoid. The pylorus portion of the stomach was lifted and ligated (with care being taken not to occlude blood vessels) [7]. The stomach was closed with interrupted sutures. Six hours after the pylorus ligation, animals were sacrificed. The stomach was dissected and the contents collected, measured, centrifuged, and subjected to biochemical analysis described below. Parameters investigated include: a) ulcer index (UI) as described earlier, b) acid secretory parameters, and c) mucoprotective parameters. Acid secretory parameters include measurement of volume of gastric secretion, total acidity determined by titrating against 0.01N sodium hydroxide to pH 8.0 using phenolphthalein as an indicator, and total acid output (product of total acidity and volume of gastric secretion). Further, pepsin activity was determined using hemoglobin as a substrate[8]. Total carbohydrates (TC) [5], total protein content (PR) [10], mucin activity (TC=PR), and gastric mucus content (g)[11] were considered as a measure of the mucoprotective parameters.

Statistical analysis: The results were expressed in terms of mean ± SEM. The significance of difference between mean values for the various treatments was tested using one-way analysis of variance test (ANOVA test) followed by Tukey's multiple range tests[12] wherever applicable to assess statistical significance of difference between the groups.

RESULTS AND DISCUSSION:

Physicochemical parameters of Avipattikar Churna:

Avipattikar churna has been evaluated for various physicochemical parameters Results were shown in Table 1.

Table -1: Physicochemical parameter of Avipattikar churna

Sr no	Parameter (% w/w)	Avipattikar churna
1	Loss on drying	5.2%
2	Total ash value	6.2%
3	Acid insoluble ash	0.19%
4	Water soluble extractive	68%
5	Alcohol soluble extractive	20%

Preliminary Phytochemical Screening

On preliminary phytochemical screening, Avipattikar churna showed presence of phyto-constituents as per Table 2.

In the current study, it is suggested that Avipattikar churna possess significant antiulcer activity as compared with omeprazole. Since centuries a number of medicinal plants and formulations have been used in the treatment of gastric ulcer. In spite of all these, treatment of gastric ulcer has continued to be big therapeutic challenges to be pharmacologists. In an effort to further search curative and safe agent for the treatment of gastric ulcer in medicinal plant, present studies were under taken for this purpose. Avipattikar churna is therefore undertaken to substantiate the claim in Ayurveda as a remedy for ulcer. Avipattikar churna is prescribed by the traditional Ayurvedic practioner and clinically it is well accepted. Still, there is no pharmacological studies have not been reported for Avipattikar churna. Hence, our efforts were devoted in this direction. Therefore, the present work is an attempt to study pharmacognostical, phytochemical and gastro-cytoprotective activity of Avipattikar formulation.

Table-2: Preliminary phytochemical qualitative study of Avipattikar churna

Sr No.	Test	Avipattikar churna
1	Tannins	+ve
2	Flavanoids	+ve
3	Saponins	+ve
4	Sterols	+ve
5	Alkaloids	-ve
6	Glycosides	+ve
7	Carbohydrate	+ve

Ethanol-induced gastric mucosal damage:

a) Effect on ulcer index: Pretreatment of Avipattikar churna (540 mg/kg/p.o.) showed significant protection (80.54%) against ethanol induced gastric mucosal damage, when compared with control group and result were comparable with that of omeprazole (64.67%) treated animals. Pretreatment of Avipattikar churna showed gastric ulcer protection effect that is evident from decrease gastric mucosal ulceration (0.516 ± 0.028) against ulcer induced by ethanol induce model (1.658±0.035) in rats and the reduction in ulcer index by omeprazole was found significant (0.923 ± 0.014). Hence, Avipattikar churna (540 mg/kg/p.o.) showed significant protection as shown in Table 3.

Table-3: Effect of Avipattikar churna (540 mg/kg/p.o.) on ulcer index in ethanol-induced gastric mucosal damage in rats

Group/ Parameters	Control (mean±sem)	Omeprazole(20 mg/kg) (mean±sem)	Avipattikar Churna (540mg/kg) (mean±sem)
Ulcer index (UI)	1.095 ± 0.053	0.393 ± 0.015 *	0.213 ± 0.011 *
Protection (%)	_	64.67	80.54

Effect of Avipattikar churna (540mg/kg, oral/7 days) and omeprazole (20 mg/kg, oral/7 days) on ulcer index in ethanol-induced gastric mucosal damage in rate. Results represent mean ± S.E.M., n=6.

Statistical analysis was done by one-way ANOVA followed by tukey's multiple range test F_{tab} = (3.68232) and F_{cal} (2, 15) = (200.4902) UI (Ulcer index). * P<0.05, when compared with control group

b) Lipid peroxides (LPO) activity:

Pretreatment of Avipattikar churna (540 mg/kg/p.o.) showed significant reduction in MDA content when compared with control group and result were comparable with that of omeprazole treated animals as show in Table 4.

Table-4: Effect of Avipattikar churna (540 mg/kg/p.o.) on lipid per oxidation against ethanol –induce gastric mucosal damage in rats

Group/Parameters	Control (mean±sem)	omeprazole(20 mg/kg) (mean±sem)	Avipattikar Churna (540mg/kg)(mean±sem)
MDA content (µmol/mg protein)	0.201 ± 0.004	0.172 ± 0.003 *	0.146 ± 0.003 *

Effect of Avipattikar churna (540 mg/kg/p.o.) and omeprazole (20 mg/ kg/ p.o.) on the level of TBARS in ethanol-induced gastric mucosal damage in rate. Result are mean ±S.E.M, n= 6. Statistical analysis was done by one-way ANOVA followed by tukey's multiple range test F_{tab} = (3.68232) and F_{cal} (2, 15) = (440.8937) UI (Ulcer index). * P<0.05, when compared with control group.

Peptic ulcer is a sore in the lining of the stomach or the first part of the small intestine (called duodenum). Ulcers in the stomach are often called gastric ulcers. Ulcers in the duodenum are called duodenal ulcers. The etiology of gastro-duodenal ulcers is influenced by various aggressive and defensive factors such as gastric acid secretion, pepsin secretion, parietal cell, mucosal barrier, mucus secretion, gastric blood flow, cellular regeneration, and endogenous protective agents (prostaglandins and epidermal growth factors). The defense mechanism of the gastrointestinal mucosa against aggressive factors mainly consists of functional, humoral and neuronal factors. Alkaline mucus secretion, mucosal microcirculation, and motility act as functional factors, while prostaglandin and nitric oxide act as humoral factors. All of the above factors are known to contribute to mucosal protection. Gastric mucosal glycoprotein plays one of the most important roles as a defensive factor of the gastric mucosa.

Gastric mucosal damage induced by ethanol is reported to be due to mucosal leukotriene release[13], sub mucosal venular constriction[14] and eventual injury[15]. Gastric mucus is an important protective factor for the gastric mucosa and consists of a viscous, elastic, adherent and transparent gel formed by 95% water and 5% glycoprotein that cover the entire gastrointestinal mucosa. Moreover, mucus is capable of acting as an antioxidant, and thus can reduce mucosal damage mediated by oxygen free radicals. The protective properties of the mucus barrier depend not only on the gel structure but also on the amount or thickness of the layer covering the

mucosal surface. Reactive oxygen species are known to be involved in the pathogenesis of ethanol-induced gastric mucosal injury in vivo[16]. This causes damage to the cell and cell membranes[17]. Oral administration of necrotizing agent ethanol, ethanol- hydrochloric acid etc., stimulate release of prostaglandins from the stomach to prevent gastric lesions through adaptive cytoprotective[18].

In the present study, we observed that oral administration of Avipattikar churna showed significant anti ulcer activity in ethanol induced gastric mucosal model along with alteration in antioxidant enzyme activity. It was observed in our study that, Avipattikar churna pretreatment resulted in significant reduction in MDA content, suggesting their efficacy in preventing free radical-induced damage. The mechanism of anti-ulcer activity in this model therefore, can be attributed to the free radical scavenging activity of Avipattikar churna that in turn might lead to gastric cyto-protection[19].

In the healthy stomach, there is a balance between aggressive factors and the protection afforded by pre-epithelial, epithelial, and sub epithelial mechanisms of gastric mucosa. Secretions of mucus and bicarbonate by surface epithelial cells constitute a mucus-bicarbonate barrier, which is regarded as the first line of defense against potential ulcerogens. Although the pathogenesis of ulcer is multifactorial, ulcer is considered to be due to derangement of balance between aggressive and defensive factors. Hence, an attempt was made to study the status of aggressive factors namely acid and pepsin and important defensive factors such as mucin secretion.

Pylorus-Ligated Gastric Ulcer Model:

a) Effect on ulcer index:

Pretreatment of Avipattikar churna (540 mg/kg/p.o.) showed significant protection (68.87%) against pylorus-ligated gastric ulcer model. When compared with control group and result were comparable with that of omeprazole (44.33%) treated animals. Pretreatment of Avipattikar churna showed gastric ulcer protection effect that is evident from decrease gastric mucosal ulceration (0.516 ± 0.028) against ulcer induced by pylorus-ligated gastric ulcer model (1.658 ± 0.035) in rats and the reduction in ulcer index by omeprazole was found significant (0.923 ± 0.014). Hence, Avipattikar churna (540 mg/kg/p.o.) showed significant protection as per Table 5.

Table-5: Effect of Avipattikar churna (540 mg/kg/p.o.) on ulcer index in pylorus- ligated gastric ulcer model

Group/Parameters	control (mean±sem)	Omeprazole (20 mg/kg) (mean±sem)	Avipattikar Churna (540mg/kg) (mean±sem)
Ulcer index (UI)	1.658 ± 0.035	0.923±0.014*	0.516±0.028 *

Protection %	_	44.33	68.87

Effect of Avipattikar churna (540mg/kg, oral/7 days) and omeprazole (20 mg/kg, oral/7 days) on ulcer index in pylorus-ligated gastric ulcer mode in rate. Results represent mean ± S.E.M., n=6. Statistical analysis was done by one-way ANOVA followed by tukey's multiple range test F_{tab} = (3.68232) and F_{cal} (2, 15) = (440.8937) UI (Ulcer index). * P<0.05, when compared with control group.

b) Effect on acid secretors parameters.

Avipattikar churna showed reduction in volume of gastric acid secretion (0.816 ± 0.021) when compared to control group (1.441 ± 0.071) as per table 6. Reduction in volume gastric acid secretion was comparable with that of reference standard ranitidine (1.066 ± 0.040). Avipattikar churna (540mg/kg, oral/7 days) showed significant decrease in total acidity (4.5±0.093) when compared to control group (7.133 ± 0.164) as per table 6. Along with pepsin content was significantly reduced by Avipattikar churna (540mg/kg, oral/7 days) (17 ± 0.966) when compared to control group (47.166 ±1.046) as per Table 6.Reduction in pepsin content was comparable with that of reference standard omeprazole (26.666 ± 1.211).

Table-6: Effect of Avipattikar churna (540 mg/kg/p.o.) on acid secretary parameters in pylorus-ligated gastric ulcer model:

Group/ Parameters Mean ±SEM	control (mean±sem)	omeprazole (20 mg/kg) (mean±sem)	Avipattikar Churna (540mg/kg) (mean±sem)
Volume of gastric acid ml/100gmb.w.	1.441 ± 0.071	1.066 ± 0.040	0.816 ± 0.021*
Total Acidity m Eq/lit.	7.133 ± 0.164	6.016 ± 0.094 *	4.5 ± 0.093 *
Acid output µ Eq./100 gm b.w.	10.288 ± 0.579	6.423 ± 0.277 *	3.673 ± 0.127 *
Pepsin activity (mg/ ml)	47.166 ±1.046	26.666 ± 1.211 *	17 ± 0.966 *
Pepsin output (µg /100 gm b.w.)	67.966±1.510	28.419 ±1.010 *	3.162±0.504

Effect of Avipattikar churna (540mg/kg, oral/7 days) and omeprazole (20 mg/kg, oral/7 days) on Volume of gastric acid in pylorus- Ligated gastric ulcer mode in rate. Results represent mean ± S.E.M., n=6. Statistical analysis was done by one-way ANOVA followed by tukey's multiple range test F_{tab}=3.682; F_{cal} (2, 15) = 41.5113 (Volume of gastric acid), 117.186 (Total Acidity), 77.225 (Acid output), 205.016 (Pepsin activity), 501.08 (Pepsin output). * P<0.05, when compared with control group.

c) Effect on mucoprotective parameters:

Avipattikar churna at the dose of (540mg/kg, oral/7 days) showed significant increase in total carbohydrate content (544.166 ±1.166) when compared to control group (283 ± 5.452) as per Table 7 and increase in total carbohydrate content by omeprazole was found significant (496.333

± 0.918). Avipattikar churna at the dose of (540mg/kg, oral/7 days) showed significant decrease in total protein content (254.166 ±1.720) when compared to control group (363 ± 5.537) and decrease in total protein content by omeprazole was found significant. Based on total carbohydrate (TC) and protein content (PR) results, mucin activity was determined in terms of TC: PR ratio. Avipattikar churna at the dose of (540mg/kg, oral/7 days) and omeprazole showed significant rise in TC: PR ratio, when compared to control group as per Table 7.

Table-7: Effect of Avipattikar churna (540 mg/kg/p.o.) on mucoprotective parameters in pylorus-ligated gastric ulcer model

Group/ Parameters Mean ±SEM	control (mean±sem)	omeprazole (20 mg/kg) (mean±sem)	Avipattikar Churna (540mg/kg) (mean±sem)
Total carbohydrate (TC) mg/ml	283 ± 5.452	496.333 ± 0.918*	544.166 ± 1.166 *
Protein content (PR) mg/ml	363 ± 5.537	360.333 ± 2.905	254.166 ±1.720*
Mucin activity (TC:PR Ratio)	1.113 ± 0.021	1.368 ± 0.019	1.510 ± 0.014*
Mucus content	0.210 ± 0.008	0.420 ± 0.039*	0.5 ± 0.036*

Effect of Avipattikar churna (540mg/kg, oral/7 days) and ranitidine (27 mg/kg, oral/7 days) on total carbohydrate pylorus- ligated gastric ulcer mode in rate. Results represent mean ± S.E.M., n=6. Statistical analysis was done by one-way ANOVA followed by tukey's multiple range test F_{tab} = (3.68232) and F_{cal} (2, 15) = (1816.08) Total carbohydrate (TC), F_{cal} (2, 15) = (983.4403) Protein content (PR), F_{cal} (2, 15) = (440.8937) Mucin activity (TC: PR Ratio).* P<0.05, when compared with control group.

Gastric acid and pepsin are important factors for the formation of ulcers in pylorus-ligated rats[10]. Increased synthesis of nucleic acids and metabolism of carbohydrates and other compensatory mechanism could be responsible for the ulceration due to pylorus-ligation[7]. Peptic ulcers occur only in those regions of the gastrointestinal tract that are bathed by digestive juices secreted by the stomach. These juices contain hydrochloric acid and pepsin. It is generally accepted that acid secretion is activated by three separate pathways: the neural, hormonal and paracrinal (local) pathways[20]. The major chemical transmitter substances are acetylcholine, gastrin and histamine. Pepsin, in contrast to acid, has received relatively little attention as the endogenous aggressor in gastric juice, although both gastric acid and pepsin are important factors for the formation of ulcers in pylorus-ligated rats. It is hyper secretion of gastric acid that is mainly responsible for production of ulcers in pylorous-ligation model. Pylorous-ligation has been reported to reduce blood flow and enhance acid secretion. In this model it has been proposed that the digestive effect of accumulated gastric juice and interference with gastric blood circulation are responsible

for the induction of ulcers. This procedure also induces a substantial release of histamine, an event that seems to be associated with an increase in free histamine levels in the gastric juice. Therefore, along with the gastric acid secretion, reflux or neurogenic effect has been proposed to play some role in the formation of gastric ulcers in pylorus ligation model. Further, increased synthesis of nucleic acids and metabolism of carbohydrates and other compensatory mechanism could also be responsible for the ulceration due to pylorus ligation.

As such it is essential to recognize the pathological phenomenon related to ulcer formation at the level of the cell membrane, glycoprotein, mitochondria, nucleic acids, enzymes and proteins and to study selectively each of these factors with regard to time and lesion development. Moreover, one of the essential criteria to determine the status of mucosal resistance/ barrier is the state of mucus secretion.

In the present study, we observed that pretreatment with Avipattikar churna showed significant reduction in total acidity and pepsin activity in treated animals

Beside this in the present work, we observed that pretreatment with Avipattikar churna showed significant increase in mucus activity & mucus content. Therefore, it is suggested that Avipattikar churna suppressed the gastric damage caused by aggressive factor and caused increase in defensive factor in term of gastric cytoprotective.

Hence, it is evident from over observation that Avipattikar churna showed inhibitory effect on acid secretion mechanism, free radical scavenging activity and significant rise in gastric mucin activity. So, it cause increase in defensive mechanism.

Based on present study, it is suggested that Avipattikar churna suppressed the gastric damage caused by aggressive factors and caused increase in defensive factors like increase in glycoprotein content of gastric juice and mucus wettening property of stomach mucosa. Hence, inhibition of gastric acid secretion and mucoprotective effect altogether led to strengthening of gastric mucosal barrier. Therefore, the mechanism of anti ulcer activity of Avipattikar churna could be attributed to gastric cytoprotection.

CONCLUSION

Based on our results, it can be concluded that an avipattikar churna as a polyherbal ayurvedic formulation possess significant gastroprotective effects.

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