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Research Article

EVALUATION OF ANTI-ULCER ACTIVITY OF *GODHUMA YUSHA* AGAINST EXPERIMENTALLY INDUCED ULCERS IN RATS

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ABSTRACT

Present study was an animal experimental study involving albino rats on whom peptic ulcer was induced and treated the same with the *Godhuma Yusha* or the wheat decoction to evaluate its role on the anti-ulcer activity. The plant material used for the study such as wheat grains were collected from the local farmers, shade dried and made into granules or powdered coarsely. Aqueous extract or the *Godhuma Yusa* was prepared by decoction method by adding the coarse powder of 100ml of drinking water and was boiled for 15 minutes. After cooling the supernatant was used to study the anti-ulcer activity. Healthy albino rats of wistar strain of either sex about 180–200mg used for the study were purchased and were caged individually and kept in air-conditioned room. Throughout the study, animals were maintained at normal laboratory conditions, at standard rat pellet diet, drinking water and libitum. Animals were being divided into four groups, consisting of six animals each. Control group (group - I) received 1% CMC (5ml/kg), positive control group (group- II) received 1% CMC (5ml/kg), test drug- 1 (group- III) received herbal suspension (5ml/kg), Test drug - 2 (group - IV) received herbal suspension in 1% CMC (5ml/kg). Total ulcer score, mean ulcer score, ulcer index, ulcer incidence were assessed before and after the intervention. The results were analysed statistically using student's 't' test and Mann whitney test. The present study showed that the test drug (*Godhuma Yusha* or the wheat decoction) has significant anti-ulcer activity as evidenced by bio-chemical parameters.

INTRODUCTION

Peptic ulcer disease is defined as group of disorders affecting the gastrointestinal system and is characterized by the caseation of inner lining of the gastrointestinal (GI) tract that occurs due to the secretion of pepsin or gastric acid and extends into the muscularis propria layer of the gastric epithelium. This is particularly observed in the stomach and proximal duodenum parts of the abdomen and may include even the lower oesophagus and distal duodenum or jejunum. In case of gastric ulcer, it presents with epigastric pain that usually occur within 15-30 minutes following a meal and in duodenal ulcer pain tends to occur 2-3 hours after the meal. [1] With a prevalence ranging from 5% to 10% with a lifetime risk, peptic ulcer has become a global problem. [2] Major risk factors causing peptic ulcer includes *Helicobacter pylori*-associated PUD and NSAID-associated PUD.[3] Its pathology begins with an imbalance between the protective and destructive layers of the gastric mucosa leading to a defect that involves muscularis mucosa. As a result, inner layers are affected by the acidic concentration with the protective superficial mucosal layer getting damaged

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and further, mucosal cells ability to secrete bicarbonate is compromised. It is established that the most common causative factor known as H. pylori colonizes the gastric mucosa and thereby causes inflammation. It also diminishes the bicarbonate secretion with acidic and gastric metaplasia advancement. [1] Gastric and duodenal ulcers are discerned based on the relationship between the timings of meals. Symptoms of peptic ulcer varies based on the age and the location of the disease and it includes bloating, abdominal fullness, epigastric abdominal pain, nausea and vomiting, weight loss, melena, weight gain and hematemesis etc.[4]

A careful requires history taking, physical examination, laboratory tests, invasive and non-invasive tests are essential for the proper diagnosis and treatment. Esophagogastroduodenoscopy is the most accurate and the Gold standard diagnostic test diagnosing gastric and duodenal ulcers. In addition, currently testing for Helicobacter pylori is suggested in all patients with peptic ulcer disease. In some patients, Endoscopy may be required to acknowledge the diagnosis.[1] In conventional medicine, management of the peptic ulcer includes include H2-receptor antagonists and the proton pump inhibitor (PPIs) due to its H2 receptor blockers and superior healing and efficacy. PPIs are known to block the acid production in the stomach resulting in relief of symptoms and promote healing simultaneously. First-line treatment comprises a triple regimen with two antibiotics and a proton pump inhibitor in which clarithromycin and metronidazole, or amoxicillin and pantoprazole are suggested for 7 to 14 days.[5] PPIs and Antibiotics work synergistically to eradicate *H. pylori* and in turn cure peptic ulcer effectively. [6] In case of a refractory peptic ulcer is which is above 5mm in diameter and that does not heal despite 8-12 weeks of PPI therapy and if conservative management fails, the disease will be managed with surgical intervention. [7]

Ayurvedic science gives prime importance to *Agni* or the *Jatharagni* or the digestive fire. According to this science, all the diseases have the impairment of *Agni* and specifically impairment of this leads to the imbalance of digestion and metabolism, leading to *Amlapitta*. *Amlapitta* is one such disease caused by the impairment of the *Jatharagni*. It is quoted in the *Shastras* that when *Pitta dosha* attains *Amla guna* excessively, it leads to the manifestation of the disease called *Amlapitta*. Detailed description of *Amlapitta* is available in Ayurveda samhitha's since the period of *Samhita,* where *Charaka Samhita* has described both *Nidana* and the *Samprapti* of *Amlapitta. Nidana* includes *Kulattha, Lavana rasa* and *Viruddha ahara* etc as the *Nidana* or the aetiological factors for *Amlapitta*. While explaining *Grahani Chikitsa*, Acharya Charaka has described the pathogenesis of *Amlapitta*.[8] In *Sushruta Samhita* also, we find the description of *Amlapitta* under the name *Amlika* similar to *Amlapitta* and is said to cause due to excessive intake of *Lavana rasa*. [9] *Kashyapa Samhita* is the first available text which has explained about *Amlapitta* as separate disease.[10] *Harita Samhita* explains about *Amla hikka and Amlapitta* with their treatment. [11] Two types of *Amlapitta* are described in Madhava Nidana. [12] Bhavaprakasha, [13]Chakra dutta, [14] and in Yogaratnakara,[15] *Amlapitta* is described in detail. As per Ayurveda, *Amlapitta* is defined as a psychosomatic ailment and can be correlated with diseases like hyperacidity, gastritis, peptic ulcer etc.

There are many traditional systems of medicine in the world, each of different associated philosophies and cultural origins. Some of these, such as Tibetan traditional medicine, remain relatively localized in their country of origin, while others such as Ayurvedic and Chinese traditional medicines are increasingly used in many different areas of the world. Plants have played a significant role in maintaining health and improving the quality of human life for thousands of years. In recent times, focus on plant research has increased all over the world and a large body of evidence has collected to show immense potential of medicinal plants used in various traditional systems. Now days, various approaches have been made to study herbal drugs for treatment of various GIT disorders. Today we a bunch of herbal drugs that have very good potential to treat Peptic ulcer and other GIT disorders both traditional knowledge and scientific data.

However, there is also a recent trend to evaluate plant or plant products in experimental animals as a means of providing experimental or pharmacological basis to the drugs used in traditional systems of medicine based on their clinical application. Present study is to find out the effect of anti–ulcer activity of *Godhūma Yūṣa* in *Āmlapitta*. The present study aimed to evaluate anti-ulcer activity of wheat decoction or against experimentally induced ulcers in rats.

MATERIAL AND METHODS

Plant Material

The plant material wheat grains collected from the local Farmers of Kamalapur, Hampi, and were authentified by Department of Dravya guna, S. V. Ayurvedic Medical College. The plant material was shade dried and made into granules (OR) powdered coarsely. Aqueous extract (or) *Godhuma Yusha* was prepared by decoction method. Coarse powder added

to 100ml, of drinking water and it was boiled for 15 minutes. After cooling the supernatant was used to study the anti-ulcer activity.

Chemicals: Sodium chloride, hydrochloric acid, folin-ciocalten reagent, ether.

Instruments: Centrifuge, U.V. Spectrometer, pH Digital.

Animals

Healthy albino rats of wistar strain of either sex about 180–200mg used for the study were purchased from S.V. Enterprises, Bangalore. The animals were caged individually and kept in air-conditioned room at a temperature of 22 +- 2 degrees Centigrade with 50%+-10% relative humidity with 12 hours light and dark cycle. Throughout the study animals were maintained at normal laboratory conditions. Animals were maintained at standard rat pellet diet (Pranav Agro's Ltd., India) and drinking water and libitum.

Instruments

UV- visible Spectro-photometer (Analytical Systems model no: AUV 2060), electronic balance (Shimadzu, model no. DS-852J), Homogenizer (Ever Shine, Model No. 607) and Cooling Centrifuge (Remi Model No. C-24 BL).

Experimental Design

Animals were divided into 5 groups each of 6 animals (n=6).

Group-I - Served as normal
Group-II - Served as control
Group-III - Standard control
Group-IV - Test
Group - Shyam control

Table 1: Treatment Schedule

S.No	Group	Treatment	Purpose
1	I	Vehicle	Serves as normal
2	II	Vehicle + Pylons ligation on 28th day.	Serves as control.
3	III	Vehicle + Ramtidi + PLon 28th day.	Serves as standard control
4	IV	Test drug + Pylons ligation on 28th day.	To study the effect of test drug.
5	V	Vehicle + Surgical Incigim	Serves as shyam control.

Method Of Induction of Ulcer

Pylorus Ligation Method

This model is the oldest model of gastric ulcers developed by shay et al. It is very simple and maximally reproducible method for the production of gastric ulcers in rats which is caused by accumulation of acid gastric juice in stomach. Hence, it has been used for the preliminary screening of anti-secretory activity of novel drugs. In this model, after the treatment, the animals were fasted for 48 hours. During fasting, the animals were grouped individually and bedding was removed and care was taken to prevent caprophagy and cannibalism. After 48 hrs., of fasting, the animals were anesthetized using ether. Under ether anesthesia a middle incision was made on the abdomen below the xiphoid process. Abdomen was opened and duodenum was exposed. A ligature was made near the duodenum and it was replaced into peritoneal cavity. Abdomen was closed. The animals were observed for 19 hrs., and animals were sacrificed using excess ether. The abdomen was cut open. Ligation was made at the oesophageal and stomach was excised. Stomach was opened in greater curvature. The contents were collected and pH of the contents was noted. The stomach was washed under running tap water. It was fixed on the board and number of ulcers were noted. Ulcer index was calculated.

Experimental Pharmacology

Fig.1. Wistar strain albino rats in (6 rats in each Group)

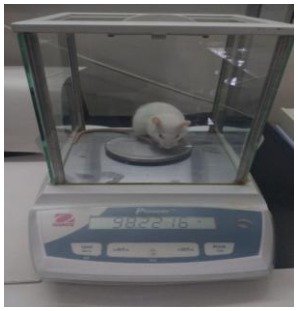

Fig.2. Weighing of Wistar rat on first day of experiment (98gm)

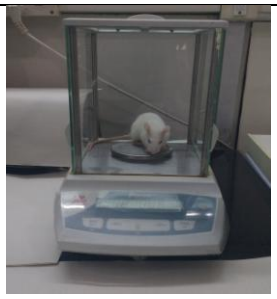

Fig.3. Weighing of Wistar rat on 15th day of experiment (116gm)

Fig.4. Metabolic cage

Parameters Monitored

- Free acidity
- Total acidity
- Total protein
- Total carbohydrate
- Pepsin activity
- Ulcer index

Estimation of Free and Total Acidity

Centrifuge the gastric content at 1000 x 100 g for 20 minutes. Note the volume of the gastric juice pipette out 1ml., of the supernatant liquid and dilute it to 10ml., with distilled water. Then total activity of gastric juice was estimated by titration with 0.01N Sodium Hydroxide using Tofer's reagent and phenapthalein as an indicator to determine free and total acidity. The result was expressed as the free and total acid output, which was expressed as mEr/liter of body weight. The acidity is calculated by using the formula.

Acidity = Volume of NaOH x Normality of NaOH x 100

Estimation of Total Proteins (P)

One milliliter of gastric juice and 9ml., of 95% alcohol was mixed, shaken, and then mixture was centrifuged at 3000 x g for 15 minutes to obtain the precipitation. This precipitate was dissolved in 1ml., of 0.1N NaOH. Next 0.9ml of distilled water was added to 0.1ml., of the above mentioned solution. Out of this solution, 0.4ml., was taken in another test tube. Four milliliters of alkaline reagent was added to this test tube and kept for 10 minutes. The 0.4ml., Phenol reagent was added to this test tube and kept for 10 minutes for colour development. The readings were taken against the blank prepared with distilled water. The protein content was obtained by calculating with the use of standard curve prepared with bovine albumin. The concentrations of proteins were expressed in terms of micrograms per milliliter of gastric juice.

Estimation Of Total Carbohydrate (Tc)

One ml. of 5% Phenol was pipette out into test tubes containing 0.15ml., gastric juice and a blank containing 0.15 ml of distilled water and mixed thoroughly. Five milliliters of 96% H_2SO_4 was added and mixed slowly. After 10 minutes, the test tubes were shaken and placed in water kept at 20°Centigrade for 20 minutes. The optical density of the developed yellow orange chromospheres was read in a UV Spectrometer at 482 nm., Several concentrations of glucose standard solution were run to prepare a standard curve. Total Carbohydrates were expressed in terms of micrograms per milliliter liberated in gastric juice Muco adhesive activity was expressed as the ratio of total carbohydrates and protein content.

Estimation of Pepsin Activity

Centrifuged gastric juice of 0.1ml., was added to 1ml., of borine albumin (0.5% W/V in 0.01N HCL, pH2) and incubated for 20 minutes at 37° Centrigrade. A duplicate background control tube (gastric juice blank) in which 1ml., of 0.01N HCl was run simultaneously. The hydrolysis was stopped by adding 2ml., of 10% tri chloro acetic acid. All tubes were heated in boiling water both for 5 minutes, the precipitate was removed by centrifugation (9000 x g for 10 minutes). A total of 1ml., of the supernatant was mixed with 0.4 ml., of 2.5 N NaOH and 0.1ml., of the Folin-ciocalteu reagent and the volume was adjusted to 10 ml., with distilled water. The absorbance was measured at 700nm., The peptic activity was calculated in terms of micrograms of tyrogine liberated per milliliter of gastric juice.

Indomethacin Administration

Animal dose = 25mg/kg body wt

50mg of indomethacin is suspended in 10ml of 1% carboxymethyl cellulose in water, each ml of the suspension contains 100mg of indomethacin

x = body wt X 5ml/1000g

x = body wt X 0.005ml

Doses of the drugs were calculated for each animal based on the body weight and respective volumes were administered orally with help of gavage needle.

Drug administration:

24 female Wistar albino rats weighing 150-200g were selected and marked with picric acid, (head, body, tail, head and body, head and tail and colorless). They were divided into 4 groups of 6 animals each they are fed on standard diet (extruded rodent diet from Wet Care) and water ad libitum. With the help of tuberculin syringe (1ml) and gavage needle the respective volume of drugs were administered orally to the animals. First the Anti-ulcerogenic property of Himcocid and Ulsaton were evaluated in indomethacin induced gastric ulcer model in wistar albino rats, then the efficacy of both poly herbal preparations were compared.

Method

Respective drugs were administered for 6 days. From day 5 the animals were kept fasting with water ad libitum, on day 6, the animals received 1% CMC, test drug-1 or 2, 2 h prior to the administration of indomethacin (20mg/kg, orally). Overnight fasted animals were sacrificed by ether anesthesia 3 hrs after the last dose of ulcerogen. The stomach was incised along the greater curvature and examined for

ulcers. The stomach was opened along the greater curvature and mounted on a moist cork board, the ulcers were examined and scored according to the method described by Ganguly and Bhatnagar, 1973.

- 0: Normal colored stomach
- 0.5: Red coloration
- 1: Spot ulcers
- 1.5: Haemorrhagic streak
- 2: Ulcers
- 3: Perforation

Mean ulcer score for each animal was expressed as ulcer index. The percentage of ulcer inhibition was determined as follows:

In each group, the total score, mean score, standard deviation, standard error of mean, p value and ulcer index were calculated.

Study design

Animals will be divided into four groups, consisting of six animals each.

Group – I	Normal control	1% CMC	30 mins later 1% CMC
Group – II	Positive control (ulcer induced group)	1% CMC	30 mins later indomethacin in 1% CMC
Group – III	Ulcer induction treated with herbal preparation-1	Herb in 1% CMC	30 mins later indomethacin in 1% CMC
Group – IV	Ulcer induction treated with herbal preparation-2	Herb in 1% CMC	30 mins later indomethacin in 1% CMC

Control group (group – I) received 1% CMC (5ml/kg).

Positive control group (group – II) received 1% CMC (5ml/kg).

Test drug – 1 (group – III) received herbal suspension (5ml/kg).

Test drug – 2 (group – IV) received herbal suspension in 1% CMC (5ml/kg).

Body Weight

Animals	**Control Cage – I**	**Positive control Cage – II**	**Himcocid Cage – III**	**Ulsaton Cage – IV**
Head	155	166	158	167
Body	163	182	180	170
Tail	167	175	171	178
Head & body	178	163	178	168
Head & tail	160	163	156	163
Colourless	160	165	159	160
Average body weight	163.8	169	167	167.9

Statistical Analysis

Ulcer scoring data were represented as mean, median and in interquartile ranges. Comparisons of the ulcer scores were analysed with Mann-Whitney U test. Significance was established when the probability value was less than 0.05. Probability values were denoted as $*p<0.05$ and $**p<0.01$ and $***p<0.001$.

Indomethacin Induced Gastric Model

Table 2: Group - 1 (Normal Control)

Group - I	**Type Of Gastric Mucosa**	**Control**
Head	Normal colored stomach	**0**
Body	Red coloration	**0.5**
Tail	Normal colored stomach	**0**
Head & body	Red coloration	**0.5**
Head & tail	Normal colored stomach	**0**
Colourless	Normal colored stomach	**0**

Number of values	6
Minimum	0
25% Percentile	0
Median	0
75% Percentile	0.5
Maximum	0.5
Mean	0.1667
Std. Deviation	0.2582
Std. Error	0.1054
Lower 95% CI of mean	-0.1043
Upper 95% CI of mean	0.4376

Indomethacin Induced Gastric Model

Table 3: Group 2 (Positive Control)

Group – I	Type of Gastric Mucosa	Positive Control
Head	Perforation	3.0
Body	Ulcers	2.0
Tail	Perforation	3.0
Head & body	Ulcers	2.0
Head & tail	Ulcers	2.0
Colourless	Perforation	3.0

Number of values	6
Minimum	2
25% Percentile	2
Median	2.5
75% Percentile	3
Maximum	3
Mean	2.5
Std. Deviation	0.5477
Std. Error	0.2236
Lower 95% CI of mean	1.925
Upper 95% CI of mean	3.075

The following parameters were recorded:

1. Total ulcer score
2. Mean ulcer score
3. Ulcer index
4. Ulcer incidence
5. The results were analysed statistically using student's 't' test and Mann whitney test.

Statistical Analysis

All the data was expressed as mean +- SEM. Statistical significance between more than two groups was tested using one way ANOVA followed by the Dunnet's test. Statistical significance was determined at $P < 0.05$.

RESULTS

Effect of Test Drug and Volume of Gastric Secretion

Increase in volume of gastric contents after 19 hours observed in Pyloric ligated (P.L) group animals when compared to normal group animals.

Test and standard group animals shows significantly reduce in gastric contents when compared to PL group.

Effect of Test Drug on pH of the Gastric Content

There was a significant decrease in the pH of the gastric content noticed in PL group when compared to normal and significant increase in the pH was observed in both Standard and Test group animals.

Effect of Test Drug on Total and Free Acidity

Animals of PL group showed increase in the free acidity and total acidity of the gastric contents after 19 hrs., when compared to normal group where as Standard and Test group animals showed significant decrease in both free and total acidity when compared to PL group.

Effect of Test Drug on Protein, Total Carbohydrate and Pepsin

Table 2: Effect of Wheat on Various Parameters of P.L. Induced Gastric Ulcers in Rats

S.No.	Group	Volume of Gastric Content	Ph Of The Gastric Content	Free Acidity	Total Acidity	Ulcer Index	Pepsin Activity	Total Carbohydrates	Proteins
		(Ml.,)		(Meq/L)	(Meq/L)		Mg Tyrosine Liberated/Ml.	(Mg/Ml)	(Mg/Ml)
1	Normal	0.0+-0.0	3.31+-0.231	0.0+-0.00	0.0+-0.00	0.0+-0.0	0.0+-0.0	0.0+-0.0	0.0+-0.0
2	Pl Control	9.967+-1.53	2.03+-0.66	8.613+-8.19	170.1+-0.76	12.954+-2.300	3.991+-0.15	5.154+-0.102	6.761+-0.3
3	Ranitidine	6.267+-2.154	2.89+-1.75	25.157+-4.531	39.123+-11.34	2.13+-1.210	1.211+-0.614	11.651+-2.971	0.834+-0.421
4	Test-I	8.123+-1.321	2.57+-0.54	34.651+-5.112	96.32+-9.218	3.94+-0.417	2.817+-0.812	7.85+-0.149	2.015+-1.651

Effect of Test Drug and Ulcer Index

Rise in the ulcer index was observed in the PL group when compared to normal animals and there was decrease in Ulcer Index was observed in standard and test groups when compared to PL group.

Animal feed and maintenance for 28 days

Animal air-conditioning for 28 days

Surgical Instruments - Scissors

- Suture needle
- Surgical Suture

Chemicals: Folin - Ciocalten reagent

- 0.01N NaOH (Sodium Hydroxide solution)
  - 95% alcohol
  - Phenol reagent
  - 96% H_2SO_4
  - Borine Albumin
  - T.C.A (Try Chloro Acetic Acid)
  - Surgical spirit
  - Ether etc.,

DISCUSSION

The term peptic ulcers include both the ulcers of the stomach or the duodenum. The formation of these ulcers depends on the peptic activity in the gastric juice and presence of acid with a breakdown of the mucosal defence inside the stomach. [16] Ulcers are defined as the open sore of either the skin or mucus membrane that presents with the features such as sloughing of inflamed tissue and superficial tissue loss.[17] In peptic ulcers, lining of stomach or the duodenum is eroded. [18] These peptic ulcers are understood as gastric ulcer or duodenal ulcer based on the site of ulceration. Sometimes patients might present with both simultaneously. Gastric ulcers are characterized by severe pain and seen more in older age group. Consumption of food increases the pain and is also associated with vomiting, nausea and weight loss. In contrast in case of duodenal ulcer, pain aggravates after the digestion of consumed food. [19]

Gastric ulcer is a common disorder where discontinuity in the gastric mucosa is observed. It occurs due to imbalance between aggressive factors (gastric acid, pepsin and gastrin) and defensive factors (mucus secretion, proste glands and bicarbonates). The modern approach to control gastro duodenal ulceration is inhibiting H+-K+-ATP are pumped to control increased acid secretion neutralize the gastric acid, eradication of H.Pylori and scavenge reactive oxygen species. A range of drugs like histamine blockers and Proton pump inhibitors although has been used for efficient management of gastric hyper secretion. Many of these drugs pose adverse effects. Phytel sources have been popular, partly because of their low cost and minimal side effects.

Ayurvedic Pharmacopoeia of India and the Indian Medica comprises vast information regarding ethno-medicinal herbs, which are effective as

antiulcer agents and they have been studied and evaluated for its effects by many researchers for its antiulcer activity. A study that conducted a preliminary photochemical screening of the medicinal plants evaluated and showed the presence of important secondary metabolites like flavonoids and tannins which are the active principles of antiulcer activity. [20] Present study is one such study that was conducted to evaluate the role of *Godhuma Yusha* and its anti-ulcer activity on experimentally ulcer induced albino rats.

Pylorus ligation induced ulcer model is the important model that shows the possible changes of the parameters for gastric content e.g., volume of gastric juice, total acidity, pH, pepsin activity. PL induced ulcers are due to auto digestion of the gastric mucosa and breakdown of the gastric mucosal service. Gastric acid and pepsin are important factors for the formation of ulcers in PL rats. In stomach, digestive effects of accumulated gastric juice in the induction of gastric ulcers is well documented in the PL model.

Reduction in the volume of the gastric acid output, free acidity and total acidity and an increase in the pH of the gastric contents in the test and standard groups revealed the antacid activity an anti-secretory activity. These might be an involvement of inhibiting H+-K+-ATP are by Test drug, thus decreasing the secretion of gastric juice. The Ulcer Index was also reduced in the groups treated with Test and Standard group when compared to control group animals. This shows the ulcer protective effect of test drug.

Pepsin, a proteolytic enzyme, which is active at pH 1- 3 is important in the digestion of various protein in food. It is also responsible for the auto digestion of proteins in the mucosal layer and causes ulcer formation. The estimation of activity of Pepsin is also an important marker parameter in the anti-ulcerogenic effect of Test drug. The test drug reduced the pepsin activity in the gastric secretion in pylorus ligated rats. The decrease in Pepsin activity contributes the anti-ulcerogenic activity of test drug and it may be due to decrease in secretion and increase in pH of the stomach contents.

Increased mucus secretion by the gastric mucosal cells can prevent gastric ulceration by several mechanisms, including lessening of stomach wall friction during peristalsis and gastric contractions, improving the buffering of acid gastric juice and by acting as an effective barrier to back diffusion of hydrates and decrease in protein levels in the gastric mucosa may contribute towards its anti-ulcerogenic and cytoprotective effects by increasing the viscosity of the gastric mucus. Decrease in protein content also signifies decrease in leakage from the mucosal cells - indicating increased mucosal resistance. This suggests the increase in glyco-protein content of the gastric mucosa. In the present study from all these results, it may be understood that the test drug (*Godhuma Yusha* or the wheat decoction) has significant anti-ulcer activity as evidenced by bio-chemical parameters.

Recommendations for Further studies

Further studies are needed to work on the possible involvement of inhibition of H+-K+-ATP uses in anti-secretory activity.

CONCLUSION

Present study showed that the test drug (*Godhuma Yusha* or the wheat decoction) has significant anti-ulcer activity as evidenced by bio-chemical parameters.

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