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Protective effect of *Tecoma stans* leaf extract on Experimentally induced gastric ulcers in rats

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Abstract: *Tecoma stans* leaves have been documented in Ayurvedic literature for treatment of various ailments especially in gastralgia. To lay scientific evidence for this ethno botanical usage, the study was designed to investigate the gastroprotective effects against aspirin induced, and pylorus ligation gastric ulcer models. Preliminary phytochemical investigation was carried out to identify various constituents present in extract and found to contain alkaloids, carbohydrates, glycosides, tannins, saponins, phytosterols, flavonoids, proteins and amino acids. The various relevant biochemical markers like mean ulcer index, gastric volume, gastric pH, free acidity and total acidity were estimated to assess the gastro protective potential of the extract. The test extract was also screened for its influence on tissue GSH levels and lipid peroxidation. The treatment with test extract has reversed all the biochemical markers of ulcer to the near normal levels in a dose dependant manner. From the results it may be concluded that the test extract possess gastroprotective activity. The gastro protective properties of the plant may be attributed to the polyphenolic compounds like flavonoids and tannins that are present in the plant. Thus it supports the traditional use of *Tecoma stans* in treatment of gastrointestinal disorders.

Keywords: *Tecoma stans*, antiulcer, aspirin, pylorus ligation.

Introduction:

Man has used plants as medicines for thousands of years[1] . Peptic ulcers have been described as an imbalance between the luminal acid peptic attacks versus the mucosal defence[2]. Its incidence is increasing due to rapid development and civilizational constraints. The estimate of incidence of peptic ulcer vary ranging between 3–10%[3]. The treatment of peptic ulcers with plant products used in folk medicine and the protection of induced gastric ulcer in laboratory animals using medicinal plants was reported[4] . Gastric ulcer is among the most serious diseases in the world. The etiology of gastroduodenal ulcers is influenced by various aggressive and defensive factors such as acid–pepsin secretion, parietal cell, mucosal barrier, mucus secretion, blood flow, cellular regeneration and endogenous protective agents such as prostaglandins and epidermal growth factors. Some other factors, such as inadequate dietary habits, excessive ingestion of non-steroidal anti inflammatory agents, stress, hereditary predisposition and infection by Helicobacter pylori, may be responsible for the development of peptic ulcer[5].

Medicinal plants occupied an important position in the socio-cultural, spiritual and medicinal arena of rural people of India. The Indian system of medicines i.e. Ayurveda, Siddha, Unani and Homeopathic systems predominantly use plant-based raw materials in most of their

preparations and formulations. According to WHO estimated that, 80% of the populations of developing countries rely on traditional medicines, mostly plant drugs, for their primary health care. Demand for medicinal plants is increasing in both developing and developed countries due to growing recognition of natural products, being non-narcotic, having less side effects, easy availability and at affordable price.

In one of our field survey we found an ever green, bushy plant namely *Tecoma stans.* Kunth or Yellow Trumpet bush belonging to the family Bignoniaceae is generally described as a perennial tree or shrub. There are reports that, leaves contain Flavanoids, Luteolin, Hyperoside, Indole oxygenase Alkaloids like Tecomanine, Tecostanine, Boshniakine, 5-dehydroskytanthine and δ-skytanthine[6]. Areial parts are used in the treatment of stomach problem, gastritis, diarrhea[7].Leaves are used in the treatment of diabetes, stomach pains, diuretic[8], decrease in cholesterol and triglycerides[9], flowers posses narcotic and anesthetic activity[10]. Therefore the present study was undertaken to evaluate the antiulcer activity of 70% Ethanolic extract of *Tecoma stans* leaves (EETSL) against aspirin and pylorus ligation induced gastric ulcer in rats.

MATERIALS AND METHODS

Collection of plant material and extraction: Fresh leaves of *Tecoma stans* were collected locally and authenticated by Prof. K. Prabhu, Dept. of Pharmacognosy, S.C.S College of Pharmacy, Harapanahalli, India. A herbarium specimen No. SCSCOP.Ph.Col Herb.No.012/2006-2007 was preserved in our college museum. The leaves were shade dried separately at room temperature and pulverized. The dried powder of the leaves were defatted with pet ether and then extracted with 70% ethanol using soxhlet apparatus. The extract was concentrated under reduced pressure using roto flash evaporator and stored in airtight container in refrigerator below 10^0C. The 70% ethanolic extract which was used for phytochemical and pharmacological investigations after subjecting it to preliminary phytochemical studies.

Animals: Wistar albino rats of both sex and albino mice were obtained from Sri Venkateshwara Enterprises, Bangalore. The animals were housed in polypropylene case at $27^0 \pm 2^0$ with 12 hour dark/light cycle. They were fed with standard rat feed (Gold Mohur Lipton India Ltd.) and water *ad libitum*. The husk in the cages was renewed thrice a week to ensure hygeinity and maximum comfort for animals. All the animal experiments were carried out accordance with the guidelines of CPCSEA and studu was approved by Institutional Animal Ethics Committee (Ref.No.SCSCOP/665/2008-09 dated 24.11.2008).

Acute toxicity study: A safe oral dose of the extract was determined by acute oral toxic class method of Organization of Economic Co-Operation and Development guideline No 420[11] . The extract was found to be devoid of mortality at 2000mg/kg. Hence, 2500 mg/kg was considered as LD50 cutoff value. The doses at 1/ 10th (250 mg/kg, p.o.) and1/ 5th (500mg/kg, p.o.) were selected for the evaluation of gastroprotective activity.

EXPERIMENTAL DESIGN: Healthy albino Wister rats were randomly assigned to 5 different groups having six animals in each group in both the model, the animals were fasted for 24 h.

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i. Aspirin-induced gastric ulcer[12]:

The animals of group I received vehicle, group II (aspirin 100 mg/kg in 2% w/v gum acacia p.o.), group III received Standard (Lansoprazole 8 mg/kg p.o), group IV and group V received test extract in two different doses such as 250 mg/kg and 500 mg/kg per oral. Animals were then sacrificed by an overdose of anesthetic ether. The stomach was dissected out and a small opening was made along the greater curvature. All the gastric content was drained into a graduated centrifuge tube and used for biochemical estimations. The stomach was then cut open along the greater curvature and evenly spread out on a dissection board. The number of ulcer per stomach were noted & severity of the ulcer scored microscopically with the help of hand lens (10x) and scoring was done as per S.K. kulkarni (1987) [13].

ii. Pylorus ligation induced ulcer[14]:

Group I was treated as control received vehicle, group as positive control, group III was treated with lansoprazole standard and groups IV & V received plant extracts (250 mg/kg and 500 mg/kg). Sixty minutes after administration of the drugs/vehicle, the animals were anaesthetized using anaesthetic ether and a midline incision was made just below the xiphoid process. The stomach was lifted out and ligated at the level of the pylorus region. Then the stomach was replaced and the abdomen wall was closed by interrupted sutures. The animals were then housed separately and food and water was withheld for 6 h following which they were sacrificed by an overdose of anesthetic ether. The stomach was then dissected out, gastric contents were collected and the boundary and ulcerated area was traced as mentioned above. Then the percentage protection was calculated.

$$\text{Percentage protection} = 1 - \frac{Ut}{Uc} \times 100$$

Where, Ut = Ulcer index of treated group

Uc = Ulcer index of control group

Determination of free acidity and total acidity: 1 ml of gastric juice was pipetted into 250 ml conical flask, added 2 –3 drops of Topfer's reagent and titrated with 0.01 N sodium hydroxide until all traces of red colour disappears and the colour of the solution turns to yellowish orange. The volume of the alkali added was noted. This volume corresponds to free acidity. Then 2 – 3 drops of phenolphthalein solution was added and titration was continued until a definite red tinge reappears. Again the total volume of alkali added was noted. This volume corresponds to total acidity.

Acidity was calculated by using the formula

$$\text{Acidity} = \frac{\text{Volume of NaOH} \times \text{Normality of NaOH} \times 100}{0.1} \text{ meq/L/100 gm}$$

STATISTICAL ANALYSIS: The data was represented as mean ± SEM(n=6), results were analysed by one-way ANOVA followed by Dunnett's multiple comparison test using Graphpad prism 5.0 software. P value less than 0.05 was considered to be statistically significant.

RESULTS: The effect of 500mg/kg of 70%EETSL on Mean Ulcer Index (MUI), GSH% increase, and LPO % inhibition in aspirin induced gastric ulceration was 59.10%, 41.35%and 49.15%which were comparable to the protection offered by Lansoprazole i.e., 70.70%, 98.15%and 49.15% respectively. Whereas, in Pylorus ligated rats, the 70%EETSL showed 20.00% and 74.00% protection w.r.t. MUI for lower and higher doses, respectively.

The impact of same(500mg/kg) dose on reduction in gastric parameters like mean volume, mean free acidity, mean total acidity and mean gastric pH were 4.86±0.29, 37.0 ± 0.50, 39.50±0.25, 4.93±0.21, 2.93±0.2 compared to 3.45±0.06, 29.05±0.63, 35.85±0.34 and 6.83± 0.10reduction showed by Lansoprazole, as shown in table 1 and 2.

Table 1: Effect of 70% EETSL on Aspirin induced Mean ulcer index, GSH levels and LPO in rats.

Groups	Treatment	Mean ulcer index ± SEM	% protection	GSH		LPO	
				Absorbance Mean ± SEM	% increase	Absorbance Mean ± SEM	% inhibition
I	Control (1ml vehicle)	2.83 ± 0.42	--	0.237 ± 0.02	--	0.238 ± 0.004	--
II	Positive Control (aspirin 100 mg/kg, p.o)	2.99±0.35	--	0.205±0.01	--	0.269±.002	--
III	Standard (lansaprazole 8 mg/kg, p.o)	0.83 ± 0.30***	70.70%	0.709 ± 0.01***	98.15%	0.121 ± 0.00***	49.15%
IV	70% EETSL (250 mg/kg p.o.)	1.40± 0.40*	50.60%	0.304 ± 0.006*	28.27%	0.183 ± 0.00**	23.10%
V	70% EETSL (500 mg/kg p.o.)	1.16 ± 0.10**	59.10%	0.335 ± 0.017**	41.35%	0.121 ± 0.01***	49.15%

Values are the mean ± S.E.M. of six rats / treatment
Significant *** P<0.001 Vs. Control

Table 2: Effect of 70% EETSL on pylorus ligation induced gastric ulceration in rats

Groups	Treatment	Mean ulcer index ± SEM	% protection	Mean vol. of Gastric Juice (ml) ± SEM	Mean Free Acidity (mEq/L/ 100g) ± SEM	Mean Total Acidity (mEq/L/ 100g) ± SEM	Mean Gastric pH ± SEM	GSH		LPO	
								Absorbance Mean ± SEM	% Increase	Absorbance Mean ± SEM	% Inhibition
I	Control	3.16 ±0.38	--	3.80 ±0.6	42.61 ±1.11	52.30 ± 0.43	3.35 ±0.20	0.403 ± 0.02	--	0.298 ±0.03	--
II	Positive Control	5.98 ±0.41	--	8.60 ±0.21	62.41 ±0.14	92.33 ±3.59	3.25 ±4.16	0.386 ±00.4	--	0.326 ±0.01	--
III	Standard (Lansoprazole 8mg/kg)	0.80± 0.20***	80.80%	3.45 ±0.06***	29.05 ±0.63***	35.85 ±0.34***	6.83± 0.10***	0.786 ±0.007***	95.03%	0.124 ± 0.002***	61.96%
IV	70%EETSL (250mg/kg)	3.33 ± 0.64**	20.0%	4.93 ±0.78*	52.80 ±0.75*	42.50 ± 0.50*	3.81 ±0.35^{ns}	0.504 ±0.006**	25.06%	0.190 ± 0.00***	41.71%
V	70% EETS (500 mg/kg)	1.08 ± 0.08***	74.0%	4.86 ±0.29*	37.0 ± 0.50**	39.50 ±0.25**	4.93± 0.21**	0.552 ±0.014**	36.97%	0.130 ± 0.00***	60.12%

Values are the mean ± S.E.M. of six rats / treatment
Significant *** P<0.001 Vs. Control

DISCUSSION: According to Robert et al. (1979) reported that the necrotizing agents-induced gastric ulcers, the lesions were characterized by multiple haemorrhage red bands of different sizes along the longitudinal axis of the glandular stomach. This model is extensively used to screen drugs for cytoprotection. Various factors that have been implicated in the pathogenesis of gastric ulcers are an increase in gastric acid secretion, pepsin activity and oxidative stress in the gastric mucosa, and a decrease in mucous and bicarbonate secretion[15]. NSAID's like aspirin causes gastric mucosal damage by decreasing prostaglandin levels through inhibition of PG synthesis[16]. Ethanolic extract of the study plant

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was significantly effective in protecting gastric mucosa against aspirin induced ulcers at all the dose level studied.

Similarly, the control group of Shay rat preparation model (pyloric ligation) showed increase in gastric secretion, free and total acids and decrease in gastric pH. There was severe gastric ulceration as indicated by higher ulcer index values (5.98). This may be due to pressor receptors (antral region) mediated vagovagal reflex leading to surge of acetylcholine followed by raised acid secretion and also due to generation of ROS. This increased secretion of acid and pepsin in pyloric ligation model leads to digestion of gastric mucosa and breakdown of mucosal barrier as pepsin is active only at lower pH[17] .n addition pyloric ligation also reduced GSH content of the gastric mucosa and increased the lipid peroxidation. Consequently reduction of gastric acid production as well as reinforcement of gastric mucosal protection has been the major therapeutic approaches of peptic ulcer disease[18]. The preliminary phytochemical studies revealed the presence of flavonoids in ethanolic extract; various flavonoids have been reported for its anti-ulcerogenic activity with good level of gastric protection[19]. So, possible antiulcer activity of 70%EETSL may be due to its flavonoid content. In this study we observed that *Tecoma stans* provides significant anti-ulcer activity against gastric ulcers in rats.

Conclusion: From the results it may be concluded that, the 70%EETSL has gastroprotective potential against aspirin and pylorus ligation induced ulcers in rats.

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