

International Journal of Integrative sciences, Innovation and Technology(A Peer Review E-3 Journal of *Science Innovation Technology*)

Section A – Basic Sciences; Section B – Applied and Technological Sciences; Section C – Allied Sciences

Available online at www.ijit.webs.com**Research Article****ANTI GASTRIC ULCER ACTIVITY OF TRIPHALA KARPAM (SIDDDHA FORMULATION) IN RATS****G. PRAMOD REDDY^{1*}, L. KAVITHA², GOWTHAMAN², R. KUMARESAN², R. GANESAN¹, P. SATYA RAJESWARAN¹, S. JEGA JOTHI PANDIAN¹.**¹Siddha Central Research Institute, Anna Hospital Campus, Arumbakkam, Chennai – 600106, T.N., India.²School of Chemical and Biotechnology, Sastra University, Thanjavur – 613 402, T.N., India.Corresponding Author Email: gpramod01@gmail.com**ABSTRACT**

The present study was aimed to evaluate anti-ulcer activity of *Triphala karpam* (TK) a Siddha formulation was investigated in rats to evaluate the anti-ulcer activity by using aspirin and pyloric ligation models experimentally induced gastric ulcer. The parameters taken to assess anti-ulcer activity were volume of gastric secretion, pH, free acidity, total acidity and ulcer index. The results indicate that *Triphala karpam* (TK) a Siddha formulation 200mg/kg showed significantly decreases the volume of gastric acid secretion, pH, free acidity, total acidity and ulcer index with respect to standard.

KEYWORDS: *Triphala karpam, anti-ulcer activity, Aspirin.***INTRODUCTION**

Peptic ulcer is worldwide problem and its prevalence is quite high in India. Several field studies from different parts of our country suggest its occurrence in 3 to 10 per thousand populations. The exact cause of peptic ulcer is not known, the disease results in chronic suffering, loss of working hours and occasional fatality. Smoking, alcoholism and spices add to the severity of the disease that often precipitate serious complication of ulcer [1]. Indigenous drugs possessing fewer side effects should be looked for as a better alternative for the treatment of peptic ulcer. There is evidence concerning the participation of reactive oxygen species in etiology and pathophysiology of human disease, such as neurodegenerative disorders such as gastro inflammation and gastric ulcer [2, 3].

Herbal medicine is fast emerging as an alternative treatment to available synthetic drugs for treatment of ulcer possibly due to lower costs, availability, fewer adverse effects and perceived effectiveness. *Triphala karpam* is a proprietary Siddha medicine with eight ingredients and indicated for peptic ulcer [4] it is one of the fast moving products as per the Market survey conducted by the co-author in Chennai. Every ingredient in this formulation possesses anti-ulcer property which is the rationale to check out the Synergistic activity of the

Drug. All the Ingredients are astringents which will help in combating peptic ulcer [5]. In the present study an attempt has been made to validate the antiulcer activity of *Triphala karpam* (TK) Siddha formulation.

MATERIAL AND METHODS**Animals**

Wistar Albino rats (150-200mg) of either sex were used in this investigation. They were maintained at standard housing condition and fed with commercial diet (Hindustan lever Ltd., Bangalore) and provided with water *ad libitum* during the experiments. The Institutional Animal Ethical Committee permitted the study (No: 92/PHARMA/SCRI, 2011).

Anti-ulcer activity**Pyloric ligation model [6] (8-15)**

The albino rats of either sex weighing between 150-200g were divided into 5 groups of 6 animals. The animals were deprived of food for 24 hours, before the commencement of experiments, but water allowed at *ad libitum*. After the fasting period the rats were anaesthetized with light ether. The abdomen was opened and the pyloric end was ligated with a thread [7]. All the samples were given 60 minutes prior to pyloric ligation [8].

Group-I received distilled water (1ml/kg, p.o) act as a control, Group-II received Ranitidine (30 mg/kg, p.o.) act as a standard and Group-III and IV received *Triphala karpam* (100&200 mg/kg, p.o) After 4 hours of pyloric ligation all the animals were sacrificed to observe gastric lesions. The gastric juice was collected and centrifuged at 1000 rpm for 10 minutes. The volume of gastric juice (ml) as well as pH of gastric juice was noted [9]. Then the gastric juice was subjected to biochemical estimation [10]. The gastric ulcer score was recorded according to the method described by Aguwa and Ukwe (1997) [11]. Gastric content were assayed for total acidity by titration against 0.01N NaOH using phenolphthalein as indicator. The volume of gastric content was measured and the total acidity and free acidity were estimated [12]. The data concerning the pH, acid secretion were analyzed by One-Way analysis of variance (ANOVA) and followed by student 't' test were show in Table 1[13].

Aspirin induced gastric ulcer [14]

In the aspirin induced ulcer experiments, five groups of albino rats with each group consisting of six animals were used. The first group served as a control group, the second group served as standard and the third and fourth groups were treated respectively with *Triphala karpam* (100mg/ 200mg), orally for 8 days. Control animals received normal saline (2ml/kg) for 8 days. After 8 days of treatment .animals were fasted for 24 hrs. ulcer was produced by administration of aqueous suspension of aspirin (a dose of 200 mg/kg orally) on the day of sacrifice .The animals were sacrificed 4h later and stomach was opened to calculate the ulcer index by kunchandy method Table 2 [15].

RESULTS

Acute toxicity studies of the various extracts of the *Triphala karpam* Siddha formulation did not exhibit any signs of toxicity up to 2g/kg body weight. Since there was no mortality of the animals found at high dose. Hence 100 and 200 mg/kg dose of the *Triphala karpam* selected for evaluations of anti-ulcer activity.

Pylorus ligation induced ulcer

The results of oral administration of the *Triphala karpam* at 100 and 200 mg/kg b.w on different chemical parameters in rats were represented in Table 1.

Triphala karpam a Siddha formulation in different doses produced a reduction in the ulcer index, gastric volume, free acidity, total acidity and raised gastric pH significantly in comparison with control group.

Table 1: Effect of various extracts of *Triphala karpam* against pylorus ligation induced gastric ulcer in rats

Results are mean ± S.E.M . (n=6) Statistical comparison was performed by using ANOVA coupled with student 't' test.

Group	Treatment and Dose mg/Kg	Gastric volume (ml)	pH	Free acidity (mEq/l)	Total acidity (mEq/l)	Ulcer score	% Inhibition of ulcer
I	Control distilled water ml/kg	7.5±0.12	2.85±0.14	26.91±0.06	59.60±0.30	4.54±0.56	---
II	Standard ranitidine 30 mg/kg p.o	3.3±0.07	5.56±0.12	10.45±0.02	22.76±0.24	1.74±0.12	82.80**
III	<i>Triphala karpam</i> 100mg/kg	5.1±0.21	4.24±0.14	17.47±0.08	39.65±0.08	3.04±0.14	70.24**
IV	<i>Triphala karpam</i> 200mg/kg	4.2±0.14	4.96±0.18	15.32±0.07	31.24±0.21	2.54±0.16	78.34**

* P<0.05, **P<0.01, ***P<0.001 were consider statistically significant when compared to control group.

Ranitidine standard drug produced significant reduction gastric ulcer and total acid output as compared to control group. But *Triphala karpam* 200mg/kg showed almost similar effects as that of ranitidine (30mg/kg) in reducing the gastric volume.

Compared to control group the entire test showed elevation in pH indicating their capacity to reduced the acidity of the gastric juice. The *Triphala karpam* at 200mg/kg indicated almost equipotent effect as that of ranitidine. Gastric free acidity is increased in control animals due to pylorus ligation. *Triphala karpam* at 200mg/kg decreased the gastric free acidity respectively. When compared to ranitidine effect, *Triphala karpam* showed significant effect in reducing the gastric free acidity.

Total acidity showed decrease in various extract when compared to control. *Triphala karpam* at 200 mg/kg reduced the mean ulcer score respectively and percentage curative ratio of *Triphala karpam* at 200 mg/kg was almost comparable to that of standard ranitidine.

Aspirin induced ulcer

Table 2 summarizes the results obtained in the experimental model of aspirin induced gastric ulceration in rats. *Triphala karpam* was found to possess remarkable ulcer protective properties at 100 & 200 mg/kg. The maximum effect of ulcer protection (70.46%) were produced at 200 mg/kg *Triphala karpam* and the standard drug ranitidine 30 mg/kg gave 82.68% of ulcer protection.

Table 2: Effect of *Triphala karpam* against aspirin induced gastric ulcer in rats

Group	Treatment and Dose mg/Kg	Aspirin	
		Ulcer index	% of Ulcer protection
I	Control distilled water ml/kg	8.2 ± 0.60	----
II	Standard Ranitidine 30 mg/kg p.o	2.30 ± 0.43	82.68***
III	Triphala karpam 100mg/kg	4.46 ± 0.65	46.54**
IV	Triphala karpam 200mg/kg	2.92 ± 0.32	70.46***

Results are mean ± S.E.M.(n=6) Statistical comparison was performed by using ANOVA coupled with student 't' test. **P<0.01, ***P<0.001 were consider statistically significant when compared to control group.

DISCUSSION

The anti ulcer activity of *Triphala karpam* was evaluated by pylorus ligation, aspirin induced ulcer models. These models represent some of the most common causes of gastric ulcers in human. So it has been proposed that in pyloric ligation, the digestive effect of accumulated gastric juice and interference of gastric blood circulation are responsible for induction of ulceration [16]. The anti ulcer activity of *Triphala karpam* in pylorus ligation model is evident from its significant reduction in gastric volume total acidity, free acidity, ulcer index and increase in pH of gastric juice. NSAIDs like aspirin causes gastric mucosal damage by decreasing prostaglandin levels through inhibition of PG synthesis. [17] *Triphala karpam* a Siddha formulation was significantly effective in protecting gastric mucosa

against aspirin induced ulcers. In this study we observed that *Triphala karpam* Siddha formulation provides significant anti ulcer activity against gastric ulcer in rats.

CONCLUSION

On the basis of the present results, it can be concluded that the anti ulcer activity elucidated by *Triphala karpam* Siddha formulation could be mainly due to the modulation of defensive factors through an improvement of gastric cytoprotection and partly due to acid inhibition.

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