IJPSR (2012), Vol. 3, Issue 08 **(Research Article)**

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 02 April, 2012; received in revised form 15 May, 2012; accepted 19 July, 2012

ANTI ULCER EFFECT OF *BASELLA ALBA* LEAF EXTRACT IN ASPIRIN INDUCED ALBINO RATS

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Key words:
Anti ulcer,
Aspirin,
Basella alba,
Non steroidal anti inflammatory drugs

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ABSTRACT

The present study was designed to evaluate the anti ulcer effect of *Basella alba* in aspirin induced ulcerated rats. Aspirin induced ulcer was revealed by increased ulcer index, decreased gastric pH, increase in the levels of pepsin, Thio barbituric acid reactive substance (TBARS). Lipid hydroperoxides and decrease in the levels of enzymatic and non enzymatic antioxidants. Treatment with the plant extract brought back the altered parameters to normal.

INTRODUCTION: A peptic ulcer, also known as ulcus pepticum, PUD or peptic ulcer disease, is an ulcer (defined as mucosal erosions equal to or greater than 0.5 cm) of an area of the gastrointestinal tract that is usually acidic and thus extremely painful. As many as 80% of ulcers are associated with *Helicobacter pylori*, a spiral-shaped bacterium lives in the acidic environment of the stomach. Ulcers can also be caused or worsened by drugs such as aspirin and other NSAIDs. Although the rate of simple gastric ulcer is in decline, the incidence of complicated gastric ulcer and hospitalization has remained stable, partly due to the concomitant use of aspirin in an aging population [1].

Gastric ulcers are also associated with considerable morbidity related to chronic epigastric pain, nausea, vomiting, and anemia [2]. Rarely, an ulcer can lead to a gastric or duodenal perforation. This is extremely painful and requires immediate surgery [3].

NSAID-induced ulcers account for approximately 26% of gastric ulcers, and they are believed to be secondary to a decrease in prostaglandin production resulting from the inhibition of cyclooxygenase. The topical effects of NSAIDs are superficial gastric erosions. However, the risk of gastroduodenal ulcer is not diminished with parental or rectal use of NSAIDs indicating injury occurring from the systemic effect of NSAIDs on the gastrointestinal mucosa. The greatest risk of developing an ulcer occurs during the first 3 months of NSAID use; thereafter, the risk decreases but continues to be present. Whether, concurrent *H. pylori* infection and NSAID use are synergistic in producing gastric ulcers remains unclear, recent accumulating evidence indicates that patients with *H. pylori* infection may be twice as likely to get a bleeding peptic ulcer**.**

Plants with medicinal properties "The gift of mother nature to mankind" are in use for centuries in the traditional system of medicine like Ayurveda, Unani, Siddha etc., in India & other countries for the treatment of diseases including ulcer. They are considered to be effective and non-toxic. Basella is a popular leafy vegetable used by large population. It's medicinal value although known well not emphasized adequately often due to the fact that it is already consumed as vegetable.

The aim of present study is to evaluate the anti ulcer effect of *Basella alba* leaf extract against aspirin induced ulcer in female albino rats.

MATERIALS AND METHODS:

Collection of plant material: Fresh and disease free leaves of *Basella alba* were collected from Sundarakkottai village, Thiruvarur District, Tamil Nadu, India. The leaves were identified with the available literature and authentified by the botanist Dr. K. Kandavel, S.T.E.T. Women's College, Mannargudi.

Experimental animals: Albino Wister female rats 7 – 8 weeks old, weighing 150-200 g, were used for the present study. The animals were divided into 4 groups of six each and housed in polypropylene cages. The animals were maintained at 12 hours light and 12 hours dark cycles. The control and experimental animals were provided with food and water *ad libitum*.

Preparation of the extract: 500g of *Basella alba* leaves *were* dried, powdered and then soaked separately in 1500 ml of 95% ethanol overnight. After filtration, the residue obtained was again resuspended in equal volume of 95% ethanol for 48h and filtered again. The above two filtrates were mixed and the solvent was evaporated in a rotavapour at 40-50°C, under reduced pressure. A dark semi solid material (7.23 %) obtained was stored at 0–4°C until used.

Induction of ulcer: Aspirin suspended in 1% carboxy methyl cellulose was administered as intra peritoneal injection at a dose of 150 mg/kg to rats as ulcer inducer.

Experimental design:

Group I: Served as Control animals and did not receive any other treatment.

Group II: Animals were treated with single intra peritoneal injection of Aspirin at a dose of 150 mg/kg., body wt.

Group III: Animals co treated with ethanolic leaf extract of *Basella alba* (200 mg/kg body weight) and aspirin (150mg/kg) for 21 days.

Group IV: Animals were treated with ethanolic leaf extract of *Basella alba* alone (200 mg/kg) for 21 days. After the period of drug administration, the effectiveness of the herbal extract was evaluated through biochemical estimations using standard procedures.

Statistical Analysis: All quantitative measurements were expressed as mean ± SD for control and experimental animals. The data were analyzed using one way analysis of variance (ANOVA) on SPSS/PC (statistical package for social sciences, personal computer) and the group means were compared by Duncan's Multiple Range Test (DMRT). The results were considered statistically significant if the p value were less than 0.05.

RESULTS AND DISCUSSION: Peptic ulcer disease (PUD) is one of the common diseases. The causes of PUD are increased gastric acid secretion and /or reduced gastric cytoprotection. Peptic ulcer diseases occur mainly due to consumption of NSAIDs, infection by *Helicobacter pylori*, stress or due to pathological conditions such as *Zollinger-ellision syndrome* [4].

Cause of PUD due to NSAIDs include, factors that increase acid secretion, reduction of gastric mucosal blood flow, inhibition of prostaglandin synthesis ,disruption of mucosal barrier, inhibition of mucus and bicarbonate secretion in the gastro intestinal mucosa [5, 6]. That is an imbalance between increased aggressive factors and decreased protection factors.

In the present study, parameters such as ulcer index, percentage of ulcer inhibition, gastric pH, pepsin content, Thiobarbituric acid reactive substances, lipid hydro peroxides, SOD, GPx, CAT, GSH, Vitamin C, Vitamin E have been analyzed. The results suggest that aspirin altered the parameters to considerable extent, which were restored to near normal with *Basella alba* leaf extract.

Aspirin produced mucosal injury was confined to glandular stomach which was revealed by increase in ulcer index and decrease in the percentage of ulcer inhibition in group II (**Table 1**). Treatment with *B. alba* produced remarkable changes to cure ulcer.

TABLE 1: THE LEVEL OF ULCER INDEX AND % ULCER INHIBITION IN CONTROL AND EXPERIMENTAL RATS

Groups	Ulcer Index	Ulcer inhibition %
Group – I	0. 5 ± 0.04	98
Group – II	15.86 ± 0.78*	12*
Group – III	8.59 ± 0.19**	66**
Group – IV	0.4 ± 0.03**	99**

Values are given as mean ± S.D. (n=6 rats), * - significantly different when compared with group I, **- significantly different when compared with group II

Aspirin-induces H^+/K^+ ATPase in gastric parietal cells. Thus increases the gastric acid secretion. In the present study increased gastric acid secretion resulted in decreased pH in aspirin induced rats which were also evident from the increased pepsin content in group II (**Table 2**). *Basella alba* brought back the altered values to normal.

TABLE 2: PH AND PEPSIN CONTENT OF GASTRIC JUICE IN CONTROL AND EXPERIMENTAL RATS

Groups	pH of gastric juice	Pepsin (per ml/hr)
Group – I	4.1 ± 0.2	2.2 ± 0.09
Group – II	2.17 ± 0.12*	4.3 ± 0.1*
Group – III	4.2 ± 0.3**	2.4 ± 0.05**
Group – IV	3.76 ± 0.3**	2.1 ± 0.09**

Values are given as mean ± S.D. (n=6 rats), * - significantly different when compared with group I, **- significantly different when compared with group II

NSAIDs induced PUD mainly due to inhibition of prostaglandin [7]. Prostaglandin has inhibitory effect on hydrochloric acid secretion in the stomach cells. NSAIDs blocks the syntheses of prostaglandin by inhibiting cyclooxygenase (COX I and COX II). Inhibition of both COX-I and COX-II induces gastric ulcer [8].

Recently, reactive oxygen species (ROS) have also shown to play a critical role in gastric ulceration process. The role of ROS in the development of acute experimental gastric lesion induced by NSAIDs is well known [9]. The inflammatory reactions induced by aspirin are a significant source of ROS in the gastric tissues. ROS damage membrane proteins by causing lipid peroxidation in membranes by attacking unsaturated fatty acids. Lipid peroxidation is measured as the amount of TBARS in the gastric mucosa.

There was a significant elevation of TBARS in gastric mucosa in ulcerated group of rats when compared to normal groups. On treatment with herbal extract, there was significant reduction in level of TBARS when compared to ulcer –induced group (**Table 3**). Our present study also correlates with these findings.

TABLE 3: THE LEVEL OF PLASMA AND TISSUE TBARS AND LIVER LIPID PEROXIDES IN ULCERATED ANIMAL MODELS

Groups	TBARS		Liver Lipid Hydroperoxides (mmol/100g)
	Plasma (mmol/dL)	Liver (nmol/100g tissue)	
Group – I	0.203 ± 0.02	0.76 ± 0.06	8.69 ± 0.29
Group – II	0.421 ± 0.03*	3.33 ± 0.31*	25.01 ± 1.3*
Group – III	0.387 ± 0.02**	2.94 ± 0.24**	13.87 ± 0.8**
Group – IV	0.210 ± 0.01**	0.79 ± 0.07**	9.27 ± 0.2**

Values are given as mean ± S.D. (n=6 rats), * - significantly different when compared with group I, **- significantly different when compared with group II

Experimental evidence indicates that an imbalance in the production and removal of ROS play a crucial role in gastric mucosal damages due to aspirin and other NSAIGs. As a consequence of this process, oxidative damage occurs [10, 11]. Organisms do, however, have enzymatic and non enzymatic defense mechanisms against the toxicity and tissue damage of ROS. Superoxide dismutase is one of the antioxidant enzymes and is part of these enzymatic defense mechanisms.

It has been reported that superoxide dismutase activity in rat stomach tissues is decreased by NSAIDs. Superoxide dismutase plays an important role in eliminating gastric damage by partially preventing oxidative damage. Superoxide dismutase destroys the highly reactive O_2 radical converting, it into the less reactive H_2O_2 that can be destroyed by the Catalase reaction.

The activity of SOD, Catalase and Glutathione peroxidase in gastric mucosa was significantly decreased in ulcerated rats, when compared to normal group (**Table 4**).

TABLE 4: LEVELS OF ENZYMATIC ANTIOXIDANTS IN CONTROL AND EXPERIMENTAL ANIMALS

Groups	SOD (U/L)	GPx (U/L)	CAT (U/L)
Group – I	75.63±0.39	3.64±0.27	140.72±3.86
Group – II	42.12±0.19*	0.75±0.06*	97.25±1.14*
Group – III	65.29±0.32**	3.43±0.245**	115.80±0.24**
Group – IV	73.75±0.24**	1.82±0.16**	139.42±1.82**

Values are given as mean ± S.D. (n=6 rats), * - significantly different when compared with group I, **- significantly different when compared with group II

Decrease in SOD and CAT causes oxidative damage since superoxide radicals were not converted to H_2O_2 subsequently H_2O_2 cannot be removed. The levels of SOD and CAT were increased and their activity is restored in herbal drug treated rats when compared to ulcer-induced rats. Thus our results agree with the previous author's findings.

Non enzymatic anti oxidants such as GSH, Vitamin C and Vitamin E play a defensive role against ulcer. Decrease in the levels of these anti oxidants in group II indicates the extent of ulceration. Treatment with the herbal extract brought back these parameters to normal (**Table 5**).

TABLE 5: LEVELS OF NON ENZYMATIC ANTIOXIDANTS IN CONTROL AND EXPERIMENTAL ANIMALS

Groups	GSH	Vitamin C (mg/dL)	Vitamin E (mg/dL)
Group – I	4.65 ± 0.41	1.72 ± 0.11	2.17 ± 0.18
Group – II	1.69 ± 0.08*	0.25± 0.01*	0.56 ± 0.03*
Group – III	3.69 ± 0.31**	1.65 ± 0.11**	1.65± 0.15**
Group – IV	3.18 ± 0.21**	0.86± 0.05**	0.98 ± 0.05**

Values are given as mean ± S.D. (n=6 rats)

Thus, it can be concluded that *Basella alba* has gastro protective potential in healing peptic ulcer.

CONCLUSION: The present study revealed that *Basella alba* has been endowed with gastro protective potential. Further studies are required to identify the mechanism of action of active principles to cure ulcer.

ACKNOWLEDGEMENT Authors are thankful to Dr. V. Dhivaharan, Correspondent, S.T.E.T. Women's College, Mannargudi for his support and guidance.

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How to cite this article:
Venkatalakshmi P and Senthamaraiselvi V. Anti Ulcer effect of *Basella alba* Leaf Extract in Aspirin induced Albino Rats. *Int J Pharm Sci Res* 2012; Vol. 3(8): 2539-2542.