

**GASTROPROTECTIVE EFFECT OF *SESBANIA GRANDIFLORA* LINN. SEEDS EXTRACT.****Pallavi. B\*, Sunil. N, Syed Mansoor Ahamed, Nandheesh R.**

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**Article Info:** Received 18 February 2020; Accepted 02 April 2020**DOI:** <https://doi.org/10.32553/jbpr.v9i2.735>**Address for Correspondence:** Pallavi. B**Conflict of interest statement:** No conflict of interest**ABSTRACT:**

**Background:** A peptic ulcer is a sore in the lining of the stomach or the first part of the small intestine. Ulcer can be developed inside the inner lining of the stomach (gastric ulcer) or the small intestine (duodenal ulcer). Peptic ulcers are usually aggravated by an imbalance between destructive and defensive factors in the stomach, it affects nearly 10% of world population. The objective of the study was to investigate the protective effect of ethanolic extract of *Sesbania grandiflora* Linn. Seeds (SGEE) on peptic ulcer induced by ethanol in experimental rats.

**Objectives:** Gastroprotective effect of *Sesbania grandiflora* Linn. Seeds extract on experimental

**Materials and Methods:** The gastroprotective effect of SGEE was studied using ethanol (70% ) induced acute gastric ulcer models. The animals were divided into five groups, each group contains six animals. SGEE was administered in two doses, (200 mg/kg and 400 mg/kg, p.o). The parameters investigated include acid volume, pH, total acidity, ulcer index, total protein, glutathione, lipid peroxidase, catalase & histopathological studies.

**Results:** 1. In gastroprotective effect study, SGEE significantly inhibited the development of ulcers induced by ethanol.

2. The SGEE significantly reduced the acid volume, total acidity, total acidity, ulcer index, lipid peroxidation & increases in pH, glutathione & catalase level.

3. Histopathological studies also revealed that SGEE is gastro-protective. Ranitidine (100 mg/kg) is used as standard drug.

**Conclusion:** All the observation implies that SGEE possess significant protective activity against ethanol induced gastric ulcer in experimental rats. 400 mg/kg doses has shown more protection compared to 200 mg/kg (dose dependent activity was obtained).

**Keywords:** Antiulcer; *Sesbania grandiflora*; Ethanol; Ranitidine.

**Introduction**

Peptic ulcers are usually aggravated by an imbalance between destructive and defensive factors in the stomach. The endogenous destructive factors in the stomach are HCl, pepsin, biliary reflux, lipid peroxidation, and the formation of reactive oxygen species (ROS) and the exogenous factors are excessive use of ethanol, indiscriminate use of nonsteroidal anti-inflammatory drugs (NSAID), stress, smoking, and infection by *Helicobacter pylori* bacteria. The defensive factors are mucus-bicarbonate barrier, mucin secretion, surface phospholipids, prostaglandins (PGs), nitric oxide (NO), mucosal blood flow, cell renewal, growth factors, and antioxidant enzymes. Oxidative stress, present in the process of gastric ulceration, increases the formation of ROS that can disrupt epithelial cell integrity. An excess production of ROS metabolites may overwhelm the endogenous antioxidants<sup>1</sup>. The treatment of acute stress ulceration and erosion is principally preventive. Reduction of gastric acid production as well as re-enforcement of gastric mucosal

production has been the major approaches for therapy of peptic ulcer disease. As a result, more and more drugs, both herbal and synthetic are coming up offering newer and better options for treatment of peptic ulcer. The type of drugs varies from being proton pump inhibitors to H<sub>2</sub>-antagonists or cytoprotective agent. Though these drugs to a certain extent have been successful in treating and controlling peptic ulcer still the treatment is unsatisfactory due to lack of complete information about etiology and pathophysiology of the disease<sup>2</sup>.

At the same time, each of these drugs confers simpler to several side effects like arrhythmias, impotence, gynaecomastia, enterochromaffin like cell (ECL), hyperplasia and haemopoietic changes<sup>3</sup>. Plants have been a major source of therapeutic agents for alleviation or cure of human disease since time immemorial. Treatment of symptomatology related to gastric ulcers or gastritis with medicinal plants are quite common in traditional medicine worldwide<sup>4</sup>.

Flavonoids and tannins from the herbs are reported to inhibit prostaglandin (PG) synthesis<sup>5</sup>. In one of our field surveys, we found a plant and identified it as *Sesbania grandiflora* Linn. belonging to family Fabaceae has been extensively used in various gastrointestinal disorders traditionally. Such as, in the treatment of diarrhoea, dysentery and juice of the leaves for arresting bleeding in gastric troubles and plant contains active constituents like flavonoids, tannins & other constituents<sup>6</sup>. Therefore, the present study is designed to investigate the anti-ulcer activity of *Sesbania grandiflora* Linn. in rats.

## Materials and Methods

**Collection and authentication of the plant:** The fresh seeds of *Sesbania grandiflora* Linn. belonging to the family Fabaceae were collected from the outskirts of Tumkur, Karnataka, in July 2018. The seeds were identified and authenticated by Dr. Chidananda, Head of the Department of Botany, Sree Siddaganga College of Arts, Science and Commerce for Boys, B.H. Road, Tumkur-572102.

**Extraction and preparation of test Sample:** The Fresh seeds of *Sesbania grandiflora* Linn. were dried under the sunlight condition, powdered with the help of grinder and stored in an airtight container. The powder was weighed (400 g). Extraction was carried out with 1600 ml distilled ethanol using Soxhlet apparatus by maintaining 20 °C temperature for 12 h. the extract was evaporated under vacuum. The extract was found 25% in the form of semi solid, greenish brown colour after vacuum evaporation. The extract was stored in refrigerator for future use. The required quantity of extract was suspended in 0.1% Tween-80 and was used for this antiulcer activity study.

## Antiulcer studies

### Grouping of animals

Sl.no	Groups	No. of animals in each group	Parameters studied
1.	Control Saline	Six	1. Ulcer index
2.	Ethanol 70%		2. Acid volume
3.	SG (200 mg/ Kg) + 3 ml Ethanol		3. pH
4.	SG (400 mg/ Kg) + 3 ml Ethanol		4. Total acidity
5.	Ranitidine (100mg/Kg) + 3 ml Ethanol		5. Total protein
			6. Glutathione
			7. Lipoperoxidation
			8. Catalase
			9. Histopathology

### a) Ethanol induced gastric ulcer<sup>7</sup>

**Purpose and rationale:** Intra gastric application of absolute ethanol is a reproducible method to produce gastric lesions in experimental animals. These lesions can be at least partially inhibited by various drugs, such as some prostaglandins. The protective effect against various irritants has been called cytoprotective activity.

The method has been modified by several authors. Witt et al. (1985) described a method to objectively quantify the extent of ethanol-induced gastric lesions utilizing a transmission densitometer to measure the optical density of the photographic negative of the stomach mucosa.

**Procedure:** Albino Wistar rats either sex weighed 250–300g were deprived of food 24 h prior to the experiment but were allowed free access to water *ad libitum*. During this time, they were kept in restraining cages to prevent coprophagy. The rats were administered either the vehicle, standard drug orally 1h prior to administration of 1 ml 70% ethanol. Untreated animals are included as control. After 1 h administration of ethanol, animals were sacrificed by over dose of diethyl ether; the stomachs are excised, cut along the greater curvature, and gently rinsed under tap water. The gastric lesions will be scored, according to the method of Valcavi et al. (1982)<sup>8</sup>.

## Results:

**Table 1:** Effect of SGEE on acid volume, pH, ulcer index and total acidity against ethanol induced gastric ulcer in rats.

Sl.no	Group and Dose (mg/kg p.o)	Acid volume (ml)	pH	Ulcer index	Total acidity (mEq/LI)
1.	Normal (Saline)	3.85±0.435	4.45 ± 0.20	0.927±0.16	95.8±1.03
2.	EtOH 1ml (70%)	5.30±0.15 <sup>a</sup>	3.38 ± 0.20 <sup>a</sup>	3.75±0.26 <sup>a</sup>	102.1±1.11
3.	SGEE 200mg + 3 ml Ethanol	2.73±0.21 <sup>c</sup>	6.77 ± 0.17 <sup>c</sup>	1.30±0.26 <sup>c</sup>	71.22±0.71 <sup>c</sup>
4.	SGEE 400 mg + 3 ml Ethanol	2.33±0.18 <sup>c</sup>	6.67 ± 0.197 <sup>c</sup>	1.03±0.30 <sup>b</sup>	67.97±0.21 <sup>c</sup>
5.	Ranitidine 100 mg + 1ml Ethanol	2.43±0.233	6.89 ± 0.29	1.284±0.26 <sup>b</sup>	70.59±0.22 <sup>c</sup>

Values are expressed as mean ± SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to <sup>a</sup>P < 0.001, <sup>b</sup>P < 0.01, <sup>c</sup>P < 0.05 when compared with inducer group.

**Table 2:** Effect of SGEE on total protein, glutathione, lipid peroxidation and catalase against ethanol induced gastric in rats.

Sl.no	Group and dose (mg/kg p.o)	Total protein (µg/gm)	Glutathione (µg/gm)	LPO (nM/mg of protein)	Catalase units/mg of protein
1.	Normal (Saline)	12.79±0.537	35.82 ± 3.67	22.6± 2.4	28.2±6.2
2.	EtOH 1ml (70%)	38.54±1.03	23.65 ± 2.91 <sup>b</sup>	59.6±5.6 <sup>a</sup>	23.6±2.2 <sup>c</sup>
3.	SGEE 200mg + 3ml Ethanol	25.07±3.98	29.85± 3.51	31.4±3.5 <sup>c</sup>	35.1±3.8 <sup>b</sup>
4.	SGEE 400 mg + 3ml Ethanol	18.53±1.237 <sup>c</sup>	38.7 ± 2.81 <sup>a</sup>	27.9 ± 2.8 <sup>c</sup>	38.2±4.1 <sup>a</sup>
5.	Ranitidine 100 mg + 3ml Ethanol	13.48±0.86 <sup>a</sup>	40.7 ± 2.69 <sup>a</sup>	26.04 ± 2.3	39.2±1.4 <sup>a</sup>

Values are expressed as mean ± SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to <sup>a</sup>P < 0.001, <sup>b</sup>P < 0.01, <sup>c</sup>P < 0.05 when compared with inducer group.

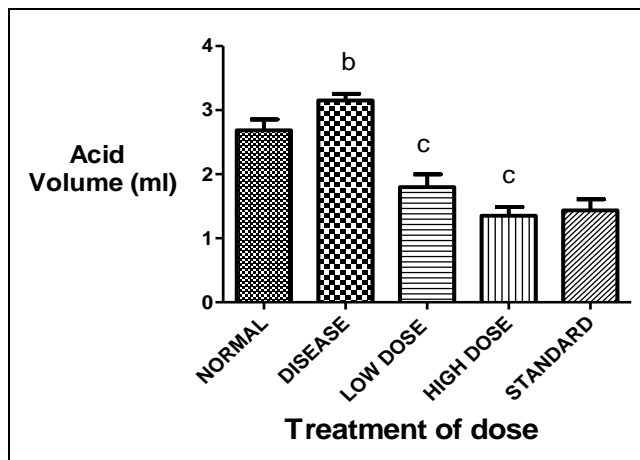


Figure 1:

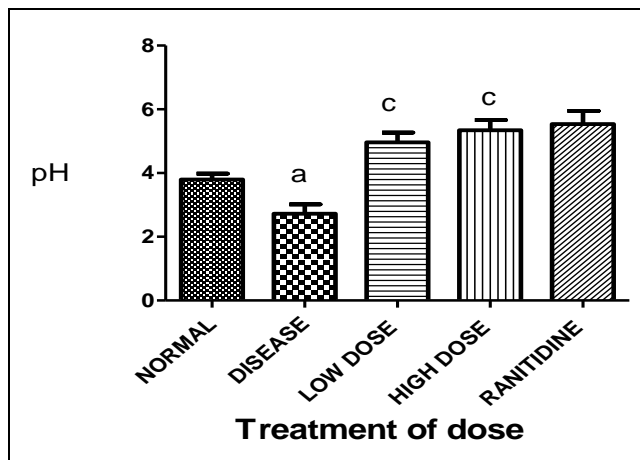


Figure 2:

Values are expressed as mean ± SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to <sup>b</sup>P < 0.01, <sup>c</sup>P < 0.05 in Fig 1, a= P < 0.001, c= P < 0.05 in Fig 2 when compared with control group.

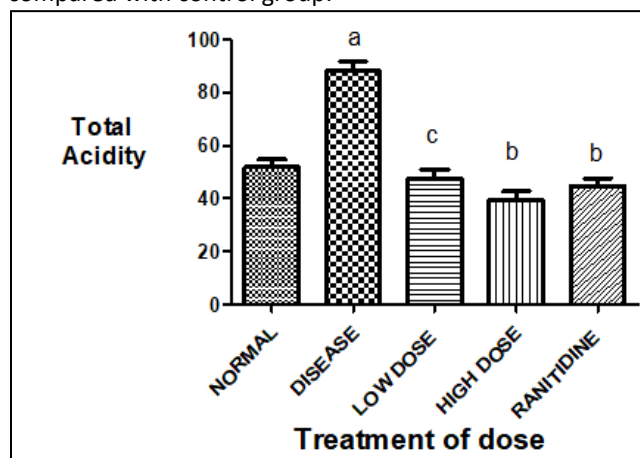


Figure 3:

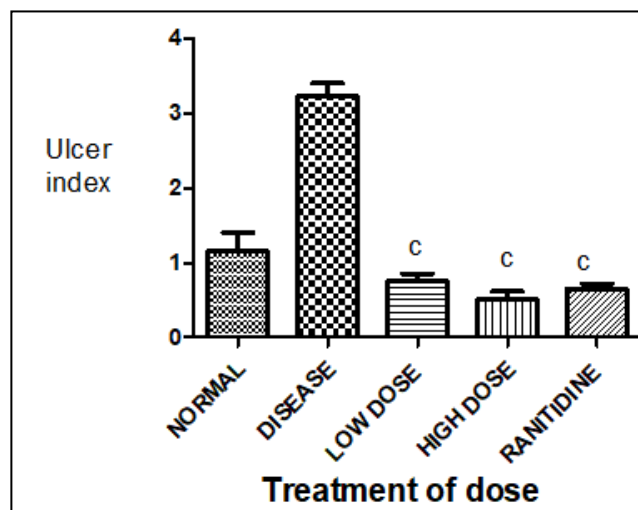


Figure 4:

Values are expressed as mean ± SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to c= P < 0.05 in Fig 3, a= P < 0.001, b=P < 0.01, c= P < 0.05 in Fig:- 4 when compared with control group.

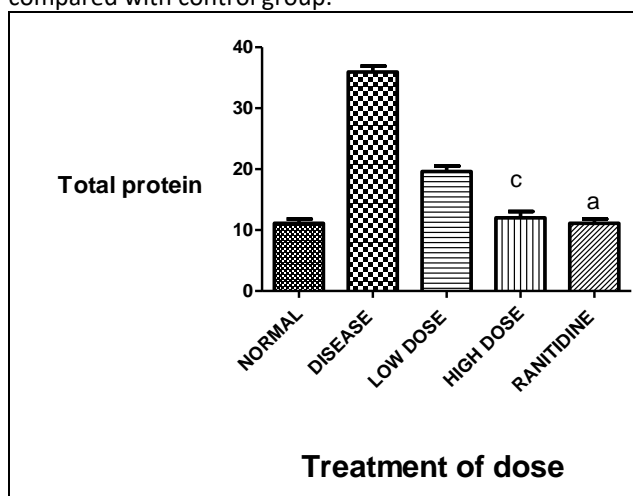


Figure 5:

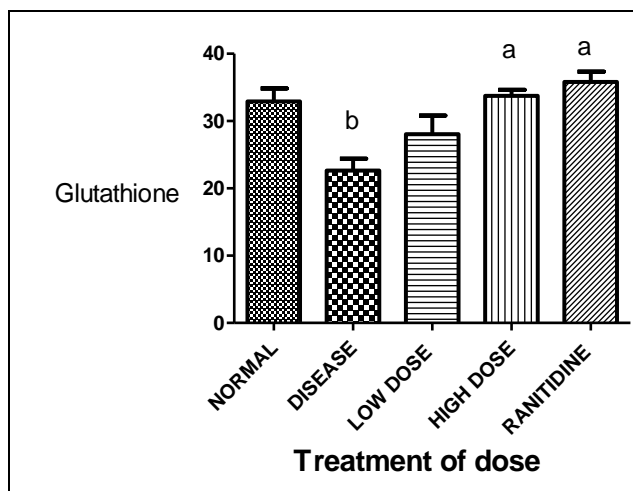


Figure 6:

Values are expressed as mean  $\pm$  SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to a=  $P < 0.001$ , c=  $P < 0.05$  in Fig:- 5, a=  $P < 0.001$ , b= $P < 0.01$  in Fig:- 6 when compared with control group.

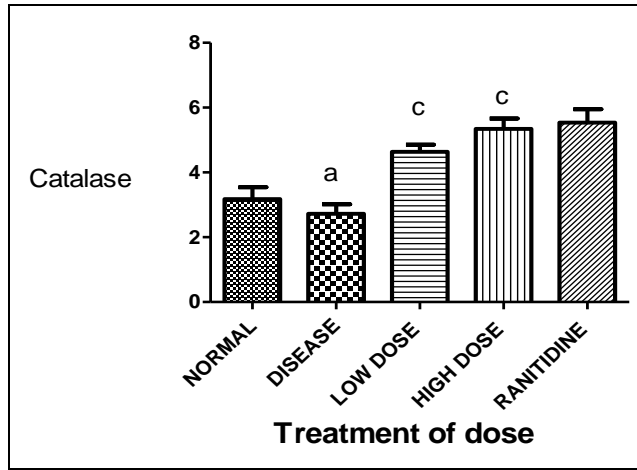


Figure 7:

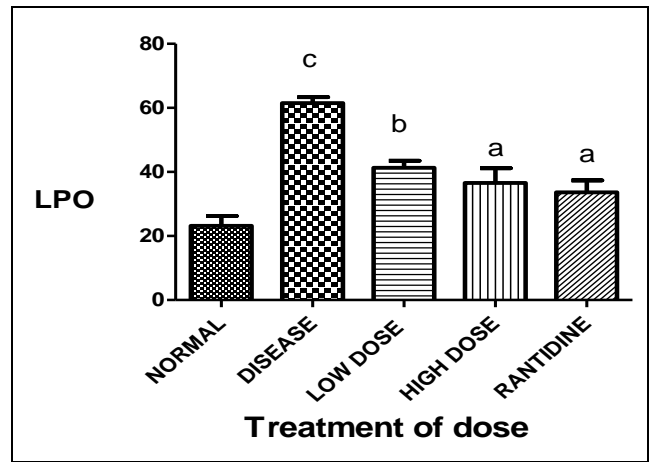
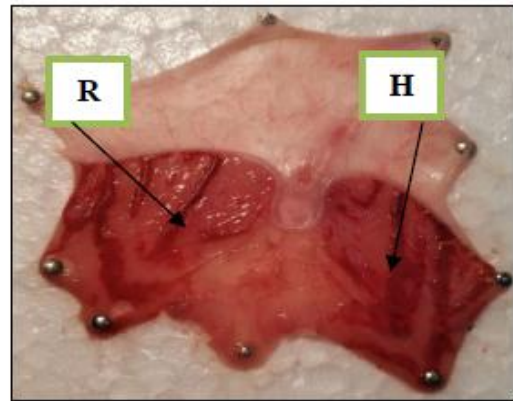


Figure 8:

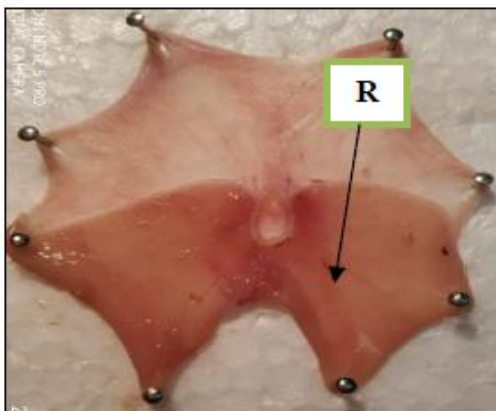
Values are expressed as mean  $\pm$  SEM of 6 animals in each group. Data analysed by One way ANOVA followed by Tukey's test Significant relative to a=  $P < 0.001$ , b= $P < 0.01$  in Fig:- 7, b= $P < 0.01$ , c=  $P < 0.05$  in Fig:- 8 when compared with control group.



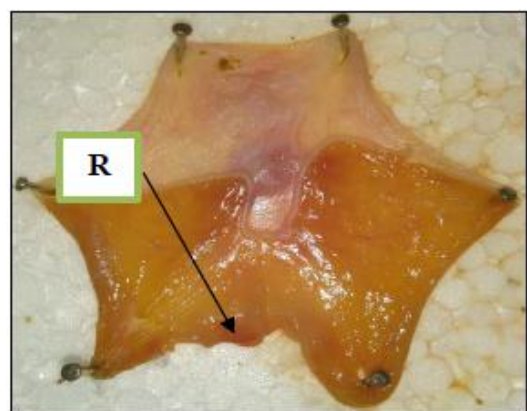
Normal group



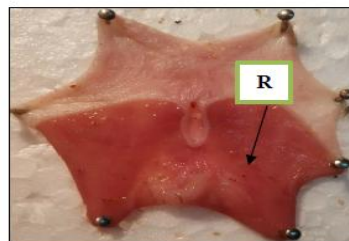
Disease group



Low dose group (200 mg/kg)



High dose group (400 mg/kg)

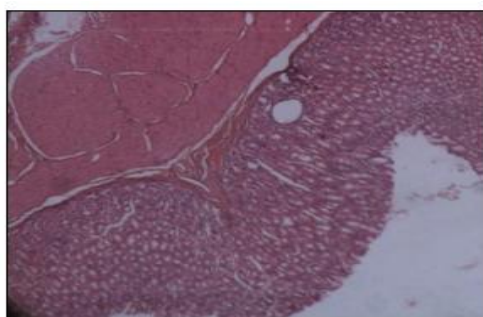


Ranitidine group (100 mg/kg)

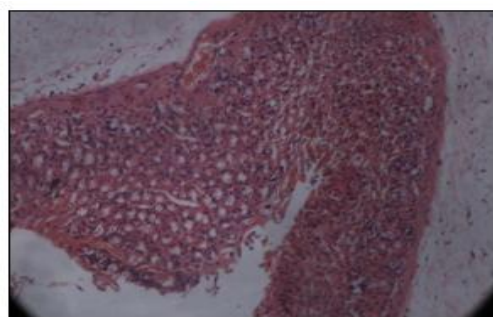
**R** → Redness

**H** → Hemorrhagic Streak

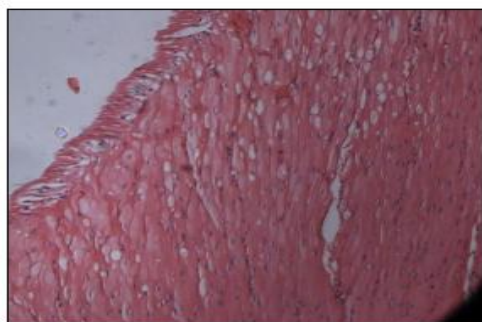
**Figure 9:** Stomachs of different groups of rats showing gastric ulcer induced by ethanol.



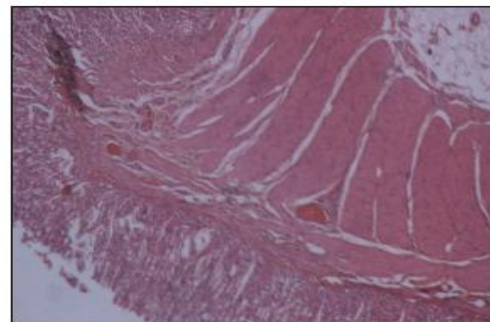
**Normal group**



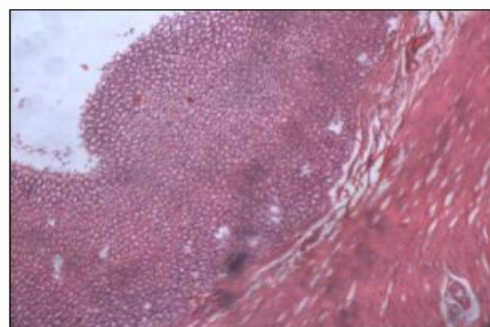
**Disease group**



**Low dose group (200 mg/kg)**



**High dose group (400 mg/kg)**



**Ranitidine group (100 mg/kg)**

**Figure 10:** Histopathological analysis of rat stomachs in ethanol induced ulcer model.



**Discussion:**

The present study is carried out to investigate the gastroprotective effect of Ethanolic extract of *Sesbania grandiflora* seeds in ethanol induced gastric ulcers. Two doses of SGEE 200 and 400 mg/kg are used in present ulcer index, acid gastric volume, pH, total acidity, glutathione, total protein, lipid peroxidase and catalase estimation is carried out in assessment of antiulcer activity. The present study, Ranitidine used as a standard drug. Ranitidine is a H<sub>2</sub> receptor blocker, is capable of reducing 90% of basal, food stimulated and nocturnal secretion of gastric acid, stimulated by histamine, gastrin, cholinomimetic drugs and vagal stimulation. Ranitidine exerts its antisecretory effect by inhibiting the histamine induced c-AMP dependent pathway<sup>9</sup>. Ranitidine also increase certain mucus component of gastric mucus in patients with duodenal ulcer<sup>10</sup>.

In the present study the ethanolic extract of *Sesbania grandiflora* Linn. At dose levels of 200 and 400 mg/kg marked gastroprotection in ethanol model showing more protection. The efficacy of SGEE over Ranitidine is observed in ethanol model.

In ethanol induced gastric ulcers model the 200mg/kg & 400mg/kg significantly reduced ulcer index, acid volume, total acidity, total protein content and lipid peroxidase. It also caused significant increase in pH, catalase and glutathione. Ranitidine 100mg/kg shown better activity compared to extract treated groups. Histopathological studies also confirmed that pre-treatment with SGEE inhibited the ethanol induced congestion, haemorrhage and necrosis in gastric mucosa. Ethanol has been shown to produce free radicals and induce peptic ulcers. While stress ulcers are mediated by brain gut axis and complex neural mechanism. Stress causes an ischemic condition in the gastric mucosa by the activation of the parasympathetic and sympathetic nervous system resulting in vasoconstriction, which in turn causes free radical generation. Further, stress has also found to inactivate mucosal prostaglandins known to favour the generation H<sub>2</sub>O<sub>2</sub>, which in turn inhibits the synthesis of prostaglandins known to favour the generation of reactive oxygen species. This in turn increases the influx of Ca<sup>2+</sup> ions, resulting in reduced membrane integrity of surface epithelial cells, thereby generating gastric ulcers<sup>11</sup>. From the above discussion it is evident that the SGEE pre-treatment in ulcer inducing model, i.e. Ethanol caused significant reduction in ulcer severity. In gastric ulcers model the antiulcer activity of SGEE was better than the standard drug used in present study. Based on the significant reduction in ulcer lesions in ethanol

model can be concluded that SGEE lowers the risk and incidence of ulcers as well as helps in treatment of active ulcers.

**Conclusion:**

The study has been done to investigate the gastroprotective effect of SGEE in ethanol induced gastric ulcer. It has been found that the SGEE possess significant gastroprotective effect in dose dependent manner. It has been observed that in ethanol model there is decrease in acid volume, ulcer index, total acidity, total protein content, lipid peroxidase and increase in pH and catalase, glutathione compared to control group. Histopathological studies also confirmed that SGEE has cytoprotective effect.

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