

Research Article

Ameliorative effect of *Albizzia procera* leaves extract against experimentally induced gastric ulcer models in Wistar albino rats

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Abstract

Albizzia procera belongs to family Fabaceae and has several phytoconstituents like flavonoids, terpenes, alkaloids, saponins etc. The plant is commonly used in traditional medicines. The objective of the present study was to assess the ameliorative effect of *A. procera* leaves extract against an experimentally induced gastric ulcer in albino rats. The antiulcer, antisecretory and cytoprotective properties of an ethanolic extract prepared from the leaves was evaluated. When given in a 200 mg/kg oral (per. os) dose, the extract produced 74% and 85% protection index in ethano-induced ulcer model and pylorus ligation model, respectively. Additionally, the extract also prohibited the formation of haemorrhage and edema, significantly lessened catalase activity ($p < 0.0001$) and the lipid peroxidation level ($p < 0.0001$) in the glandular tissue of Wistar albino rats. Furthermore, the extract also significantly diminished the total acidity ($p < 0.0001$) of the gastric fluid by increasing its pH ($p < 0.0001$). The occupancy of different phytoconstituents in the extract, such as flavonoids and tannins were identified that may be responsible for its gastro-protective activity. All these results provide a basis explaining the antiulcer ability of *A. procera* being useful in the management of gastric ulcers.

Keywords: Antiulcer, Gastroprotective, Pylorus-ligation, Safed siris, Ulcer index

INTRODUCTION

Clinically, peptic ulcer can be defined as the most regular and familiar gastrointestinal disorder (Akhtar *et al.*, 1992). These are the sore owing to the disparity between protective factors and damaging factors within gastroduodenal mucosa. Ulcer treatment is now primarily concentrated on reducing the harmful properties of invading secretion of acid, but novel search revived the development of safer medicine that can protect the gastric mucosa neither by disturbing acid secretion nor by deactivating intragastric acidity. Almost all the conventional drugs give rise to adverse effects like impotency, hematopoietic changes, gynecomastia and arrhythmias

(Ariypshi, 1986). On the other hand, plant extracts have exhibited some promising outcomes for managing gastric ulcers and are an interesting source for blooming new drugs (Pillai *et al.*, 1978).

Albizzia procera belonging to family Fabaceae, commonly known as Safed Siris, naturally occurs in India, northern Australia, southern China, including Hainan and Taiwan. It has also been instigated into few African countries, along with Panama and Puerto Rico. The phytochemical examination of *A. procera* is reported for various secondary metabolites such as saponins, terpenes, alkaloids and flavanoids. Few bioactive compounds separated and recognized were new alkaloids (budmunchiamines A, B, C), triterpenoid saponins

(julibroside), and two flavonol glycosides (quercitrin and/or isoquercitrin) (Sivakrishnan and Swamivelmanickam, 2019). The whole plant has utility in some way, such as leaves and pods are eatable portions, whereas its wood is a reasonable material for paper pulp. *A. procera* was stated to exhibit diverse pharmacological activities. The plant is used conventionally in convulsions, delirium, pain, and sepsis. The plant bark has spermicidal action (Azamthulla *et al.*, 2015) and is also given for rheumatism and haemorrhage. *A. procera* exhibits several pharmacological activities, for instance, antimicrobial activity, Central Nervous System activity, cardiogenic activity, antioxidant property, anticancer etc. (Khatoun *et al.*, 2014) (Kokila *et al.*, 2013). The objective of the present study was to evaluate the antiulcer, antisecretory and cytoprotective properties of the leaves extract of *A. procera* in Wistar albino rats,

MATERIALS AND METHODS

Plant material collection

A. procera plant leaves were possessed from Indo-Gangetic plain regions of Lucknow, India. The herbarium was prepared and submitted for authentication to the National Botanical Research Institute (NBRI) Lucknow. The Authentication voucher specimen number is 97824.

Ethanol extracts preparation

Fresh leaves of *A. procera* were collected, air-dried and powdered. Using a soxhlet extractor; 100gm of powdered drug was considered and extracted for 72h taking 90% ethanol as a solvent. The extracted drug was concentrated in a water-bath at temperature 50-55°C and dried in a desiccator (Kokate *et al.*, 2015). Ethanol extracts of *A. procera* leaves extracts (EEAP) were subjected to preliminary phytochemical analysis (Khandelwal, 2002).

Determination of total phenolic, flavonoid content and *in vitro* antioxidant property of extract

Leaf extract of *A. procera* was evaluated for total phenolic content (TPC) using Folin-Ciocalteu (FC) assay using Gallic acid standard curve. In the procedure, 0.5 ml of plant extracts were mixed with 1.5 ml of Folin-C reagent diluted 1:10 v/v. Then after 5 minutes 1.5 ml of 7% sodium carbonate solution was added. The final volume of the tubes was made upto 10 ml with distilled water and allowed to stand for 90 minutes at room temperature. Absorbance of the sample was measured against the blank at 750nm using Shimadzu UV-1600 Spectrophotometer and results were expressed in gallic acid equivalents (Ruchi and Rekha, 2017).

The total flavonoid content (TFC) of the leaf extract was determined by Aluminium chloride method using quercetin as the standard. 1 ml of the test substance and 4

ml of water was added to a 10 ml volumetric flask. Add 0.3 ml of 5% sodium nitrite, 0.3 ml of 10% aluminium chloride was added after 5 minutes. After 6 minutes of incubation period at room temperature, 1 ml of 1M sodium hydroxide was added to the reaction mixture. Immediately the final volume was made up to 10 ml of distilled water. The absorbance of the sample was measured against the blank at 510 nm using Spectrophotometer and results was measured as milligram of quercetin present per gram of dried extract sample (mg QE/g) (Pekal and Pyszynska, 2014).

The free radical scavenging activity of the extracts was determined by DPPH method. The 0.006% w/v of DPPH was prepared in 95% methanol. The ethanolic leaves extract was mixed with 95% methanol to prepare stock solution 1mg/ml. Freshly prepared DPPH solution was taken in test tubes and extracts were added by serial dilutions(100-1000µg) to every test tube. The final volume was 2ml and discolouration was measured at 517nm after incubation for 30 min in dark using Spectrophotometer. Ascorbic acid was used as standard (Kedare *et al.*,2011).

Experimental animals

150-200g healthy Wistar albino rats were purchased from the Laboratory Animal Services Division of Central Drug Research Institute, Lucknow. During the study period, the International guidelines of the Organization for Economic Cooperation and Development for the humane treatment of the animals used in the study were followed. For keeping the animals, polyacrylic cages were used and all the standard housing conditions were sustained (room temperature 24-27 °C). This was accompanied by a 12 h light and dark cycle. Food and water were accessible *ad lib*. Food was not permitted between an hour before and till the conclusion of the behavioral studies. All the experimental procedures described and performed were studied and permitted through the Institutional Animal Ethics Committee (IAEC) and Committee for control and supervision of Experiments on Animals (CPCSEA), Amity Institute of Pharmacy. Amity University Uttar Pradesh, Lucknow.

Treatment schedule

Wistar rats of either sex (150-250 gram) were taken and grouped into 4 groups: each having 4 animals viz. Group 1: Positive Control (0.9% normal saline); Group 2: Negative Control (Ethanol induced and pylorus ligation induced); Group 3: Standard (Ranitidine 45 mg/kg); and Group 4: EEAP (Ethanol *A. procera* leaves extract) (200 mg/kg). Two ulcer-inducing models, i.e., Ethanol induced ulcer model and Pylorus ligation induced ulcer model, were performed. Treatment was given orally for 14 days in all animals.

Ethanol induced gastric ulcer model

In this model, animals were administered orally with 1 ml (90% v/v) ethanol to produce the gastric ulcers. The animals were fasted 24 h before the induction but had free access to water. Thirty minutes before treating with ethanol, different groups of animals were given *A. procera* leaves extract (test group), 0.9% normal saline (negative control) and Ranitidine (standard group) orally and then sacrificed 30 minutes later the disease induction. The abdominal of the sacrificed animals was dissected out and the greater curvature of the stomach was observed for ulcer spots and hemorrhagic lesions. Ulcer score was then recorded accordingly. The length of each lesion was observed at 10 x magnification and measured in mm. Ulcer index per stomach was then totalized. Lastly, to learn the histopathological changes, all treated and ulcerated stomach were soaked in 10% formalin for 24 h (Kumar, *et al.*,2013; Misra *et al.*, 2012).

Method of ulcer rating

Ulcerated stomachs were noticed for the wounds with the support of 10 X lens and then rated 0 for the normal stomach, 0.5 for red pigmentation, 1.0 for 1-3 small lesions and ulcer spots, 2.0 for 3-5 lesions and hemorrhagic bands and 3.0 for more than 5 lesions.

Calculation of ulcer index

The average score of ulcers was calculated and stated as ulcer index for every individual group of animals. The extent of ulceration in the treated groups was equated to the Negative control groups in all the models.

Ulcer index = (UN + US + UP) × 1\10 ..Eq. 1
where.

UN= Average number of ulcer/animals

US = Average severity score

UP = % of animals with ulcers

% Ulcer inhibition= [Av. ulcer index for the Negative control group – Av. ulcer index for test group / Av. ulcer index for Negative control group] × 100

Histopathological evaluation

All the dissected stomachs were dipped for 24 h in Formalin (10%) to evaluate the possible modifications via Histopathological study. For tissue staining, Haematoxylin-eosin was used to review some transformations like gastric epithelium removal, infiltration of neutrophils, gastric erosions, inflammation and oedema (Brzozowski *et al.*,1998).

Pyloric ligation induced gastric ulcer model

The model involved the exposure of the animal's stomach under the light anesthesia ketamine (40mg/kg). The experimental animals were kept on fasting for 24 h before the test was conducted with unrestricted access to water. The stomach was opened by making a small

midline incision and the pylorus of the stomach was somewhat lifted out. Cautiously, the pylorus was replaced without affecting the blood supply after it was ligated. Lastly, the abdominal wall was sutured. Immediately following pyloric ligation, the animals were administered 0.9% normal saline (Group 2 : Negative Control), *A. procera* extract (Group 3: EEAP, Test Group) and Ranitidine (Group 4: Standard drug group, 45 mg/kg Body weight (bw) orally. Four hour after pyloric ligation the stomachs of the animals were dissected out after they were sacrificed with an excess of chloroform. All the gastric contents obtained from the dissected stomach were collected into graduated tubes. The contents were determined for their volume, total acidity, and pH. Erosions were witnessed and then the glandular part of the stomach was cut open to expose the inner surface for the examination of ulcers and washed under a stream of water. Based on ulcers' diameter, all the erosions were counted and scored on a scale of 1-3. Ulcer index was enumerated as the total diameter of ulcers for each stomach divided by a factor 10. Using the formula, the percentage of ulcer protection was calculated (Prabha *et al.*,2003).

Biochemical estimations

The collected gastric juice from the dissected stomachs was assessed for their volume and the pH. It was then centrifuged and evaluated for different biochemical estimations (Srivastava *et al.*,2010).

Determination of free and total acidity

Topfer's reagent (2-3 drops) was added into a 100 ml conical flask holding 1 ml of gastric juice. Titration for the mixture was done using 0.01N sodium hydroxide until the solution developed yellowish-orange. The amount of alkali added was observed, which corresponded to free acidity. For knowing the total acidity, the juice was again titrated by adding phenolphthalein solution (2-3 drops) and sustained up to the appearance of red colour. The total added amount of alkali was observed, which corresponded to total acidity.

Calculation of acidity

Acidity = Volume of NaOH added × Normality of NaOH × 100 / 0.1 (meq/Lit/100 gm)Eq. 2

Estimation of mucin

The stomachs were everted following the assemblage of gastric juice and immersed for 2 h in 0.1% alcian blue 8GX, which was prepared in buffered 0.16 M sucrose with 0.05 M sodium acetate whose pH was accustomed to HCl. Through two consecutive washes with 0.25 M sucrose solution at 15 and 45 min, the formed uncomplexed dye was withdrawn whereas the complex formed between the dye and mucus was immersed in 0.5 M MgCl₂ (magnesium chloride) (10 ml).

After 2 h of immersion, the developed blue solution with an equivalent volume of diethyl ether was agitated. Lastly, the aqueous phase was utilized for the determination of optical density using a Hitachi 1520 spectrophotometer at 605 nm (Saranya, *et al.*, 2011).

Estimation of catalase activity

0.4 ml water, 1.0 ml buffer and 0.2 ml enzyme were measured and put into 0.5 ml of the mixture (Reagent A + Reagent B). 2.0 ml acetic acid was added after incubating the complete mixture for 0, 30, 60, 90 seconds and then heated for 10 minutes. The colour established after heating was declaimed at 610 nm. Catalase activity was calculated in moles of decomposed H_2O_2 /min/mg protein (Aebi, 1984).

Evaluation of lipid peroxidation (LPO)

Sample (1.0 ml) was possessed and put into 2.0 ml TCA-TBA-HCL reagent. For 15 minutes, the mixture was heated in a steaming water bath. The heated solution was cooled and centrifuged at 1,000 g for 10 minutes. The flocculent precipitate was discarded, whereas clear supernatant was utilized. The readings were taken at 535nm and LPO was stated as nmoles of formed MDA/min/mg protein (Buege *et al.*, 1978).

RESULTS AND DISCUSSION

The total phenolic Content and total flavonoid content in each gram of extract of *A. procera* were found as milligram equivalent gallic acid 46.9 mg GAE/g and 35.3 mg QE/g, respectively, suggesting its antioxidant activity which is vital for the cytoprotection and ulcer healing potential of *Albizia* leaves' extracts. The DPPH scavenging activity of leaf extract was found to be 13.35 μ g/ml (Fig. 1). The antioxidant effect of *Albizia* was mainly due to the presence of phenolic components and flavones.

Oral administration of absolute 90% ethanol produced distinguishing lesion in rats of Negative control group, Standard group and EEAP group. The rat stomach's glandular portion showed normal architecture in positive control whereas Negative control showed redness, 8-10 red spots & hemorrhagic streaks. Ranitidine group showed 1-2 red spots & EEAP group showed 2-3 red spots (Fig 2). The ulcer index in Negative Control, Ranitidine (45mg/kg oral) and EEAP (200mg/kg per.os) was 9.25 \pm 0.15, 1.11 \pm 0.18, 1.85 \pm 0.15 respectively. The ulcer protection of EEAP was statistically significant by ANOVA test $P < 0.0001$, Positive control group does not play a significant role in ulcer index. *A. procera* showed a protection index of 74% with the dose of 200 mg/kg b.w compared to Negative control and Ranitidine as reference standard drug (Table 1).

Ethanol is the most common ulcerogenic agent producing acute gastric hemorrhagic erosions when adminis-

tered intragastrically to rats. Factors responsible for ethanol's gastric lesions were depletion of gastric mucus content and release of mucosal leukotrienes. Ethanol induces damage to the gastric mucosa via accumulated activated neutrophils that are a source of free radicals. Thus, Xanthine oxidase influences sufficient making of oxygen free radicals, which cause amplified LPO and cell membrane destruction (Dwivedi, *et al.*, 2014). Histopathological evaluation in Positive control group showed normal mucosa, Negative control showed oedema, inflammation, degeneration, haemorrhage, in the ethanol-induced model. The treatment with 200 mg/kg b.w *A. procera* group indicated regeneration and prevention of haemorrhage and oedema formation whereas Ranitidine group showed almost normal appearance (Fig. 3). The histopathological observations showed that ethanol-induced damages to the mucosal epithelium had a nearly normal structure, fewer haemorrhage and necrosis because of prior treatment with *A. procera* ethanolic extract guards the gastric cells by subsiding the necrosis and haemorrhage against ethanol-induced injury as also stated by Alvarez-Suarez *et al.* (2011) for strawberry polyphenols that attenuate ethanol-induced gastric lesions in rats by activation of antioxidant enzymes and attenuation of MDA increase.

When administered orally with *A. procera* leaves extract (200mg/kg), pylorus ligated animals displayed a decline in gastric acid production and change in its pH, decrease of total acidity, and free acidity well fall in ulcer index while comparing it with the control group, Positive control group doesn't play a significant effect in these estimations. The rat stomach's glandular portion showed normal architecture in positive control, whereas Negative control showed redness, 5-6 red spots & hemorrhagic streaks. Ranitidine group showed 1-2 red spots and EEAP group showed 2-3 red spots (Fig. 4). The ulcer index in Negative Control, Ranitidine (45mg/kg oral) and EEAP (200mg/kg p.o) was 9.62 \pm 0.19, 0.92 \pm 0.15, 1.48 \pm 0.25 respectively and was statistically significant $P < 0.0001$ (Table 2). *A. procera* had shown a protection index of 85% with the dose of 200 mg/kg b.w compared to Negative control and Ranitidine as the reference standard (Fig. 4). The p^H in Negative Control, Ranitidine and EEAP was 1.7 \pm 0.07, 2.9 \pm 0.10, 2.6 \pm 0.103 respectively and was statistically significant $P < 0.0001$. The gastric volume (ml) in Negative Control, Ranitidine and EEAP was 3.12 \pm 0.0846, 1.26 \pm 0.05426, 1.44 \pm 0.1565, respectively and was statistically significant $P < 0.0001$. The free acidity (Meq/ltr) in Negative Control, Ranitidine and EEAP was 21.66 \pm 1.944, 9.6 \pm 0.4282, 12.6 \pm 0.5578 respectively and was statistically significant $P < 0.0001$. The total acidity (Meq/ltr) in Negative Control, Ranitidine and EEAP was 82.0 \pm 2.176, 38.4 \pm 1.430, 51.2 \pm 1.167 respectively and was statistically significant $P < 0.0001$.

In the Pyloric-ligation model, treatment with *A. procera*

Table 1. Ulcer inhibition in ethanol induced ulcers in rats.

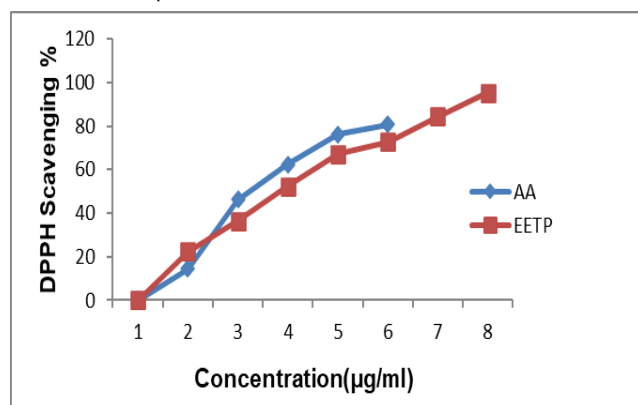
S. N.	Treatment	Ulcer index	% Ulcer Inhibition
1.	Negative Control (Group 2)	9.25 ± 0.15	-
2.	Ranitidine (45mg/kg b.w) (Group 3)	1.11 ± 0.18*	88 %
3.	EEAP (200mg/kg b.w) (Group 4)	1.85 ± 0.15*	74%

No. of rats in each group: 4; Values are expressed in terms of Mean ± S.E.M, (N=5) *p<0.0001 using one-way ANOVA followed by Dunnett's test

Table 2. Effect of EEAP on ulcer inhibition in pylorus ligation induced gastric ulceration in rats.

S. N.	Treatment	Ulcer index	Protection %	pH of gastric juice	Gastric juice (ml)	Free acidity Meq/l	Total acidity Meq/l
1.	Negative Control (Group 2)	9.62 ± 0.19	-	1.7±0.07	3.12±0.0846	21.66±1.944	82.0±2.176
2.	Ranitidine (45 mg/kg b.w) (Group 3)	0.92 ± 0.15*	90.4%	2.9±0.106*	1.26±0.05426*	9.6±0.4282*	38.4±1.430*
3.	EEAP (200mg/kg b.w) (Group 4)	1.48 ± 0.25*	85%	2.6±0.1033*	1.44±0.1565*	12.6±0.5578*	51.2±1.167*

No. of rats in each group: 4; Values are expressed in terms of Mean ± S.E.M, *p<0.0001 using one-way ANOVA followed by Dunnett's test when compared with control.

**Fig. 1.** DPPH scavenging activity of *A. procera* leaf extract (EEAP) in-vitro.

showed cytoprotective mechanism on gastric mucosa and thus inhibited ulcers through considerably reducing the secretion of basal gastric acid, which is characteristic for beginning of ulcers. The various factors regulate gastric acid secretion such as histaminergic and gastrinergic neurotransmissions, including proton pump, anxiety effect, vagal activity, and irritant receptors (Waldum et al., 2013). The current data evidently established that anti-ulcerogenic activity of *A. procera* may be linked to its action of inhibiting the aggressive factors (acid and pepsin). These aggressive factors are a chief feature in the expansion of peptic ulcer. The consequences obtained from the current studies also showed that *A. procera* had anticholinergic and vagolytic activity as well as the inhibitory effect on irritant receptors. The mucous content of rats treated with EEAP was increased at a dose level compared to the Negative control group. The mucous content (μg alcian blue/g wet glandular tissue) in Positive control, Nega-

tive Control, Ranitidine and EEAP was 320.1±0.47, 232.62±0.32, 309.2±0.14, 304.94±0.37, respectively. *A. procera* increased the mucin content with the dose of 200 mg/kg b.w compared to the Negative control and Ranitidine as reference standard drug and was statistically significant $P < 0.0001$ (Table 3).

Consequently, *A. procera* may exhibit gastric mucosal protection by the potential mechanism of partially strengthening the mucosal barrier, reducing the acidity of gastric fluid, and increasing its pH. Antiulcer drugs upsurge the mucus secretion consisting of mucin-type glycoproteins in the gastric mucosa. These glycoproteins could be determined by the quantity of alcian blue binding. The antioxidant enzyme catalase ($\mu\text{ml H}_2\text{O}_2/\text{min}/100 \text{ mg}$ of tissue) in Positive control, Negative Control, Ranitidine and EEAP was 25.58±1.72, 5.64±1.22, 22.8±1.95, 19.86±1.42, respectively. *A. procera* increased the catalase with the dose of 200 mg/kg b.w in comparison with the Negative control and Ranitidine as reference standard drug and was statistically significant $P < 0.0001$. The lipid peroxidation (nmol of MDA/mg protein) in Positive control, Negative Control, Ranitidine and EEAP was 24.82±0.66, 39.84±0.52, 26.42±0.06, 27.02±0.27, respectively. *A. procera* decreased the lipid peroxidation (LPO) with the dose of 200 mg/kg b.w in comparison with the Negative control and Ranitidine at 45mg/kg as reference standard drug and was statistically significant $P < 0.0001$ (Table 3).

Reactive oxygen species (ROS) are generated through numerous normal metabolic processes and are needed for the organism's malfunctioning. Various antioxidant enzymes like SOD and CAT reduces their accumulation. Any imbalance in these enzymes' activity normally

Table 3. Effect of EEAP on catalase activity, lipid peroxidation and mucous content.

Treatment	CAT ($\mu\text{ml H}_2\text{O}_2/\text{min}/100$ mg of tissue)	LPO (nmole of MDA/mg protein)	Mucous content (μg alcian blue/g wet glandular tissue)
Positive control (Group 1)	25.58 \pm 1.72	24.82 \pm 0.66	320.1 \pm 0.47
Negative control (Group 2)	5.64 \pm 1.22	39.84 \pm 0.52	232.62 \pm 0.32
Ranitidine (45 mg/kg b.w) (Group 3)	22.8 \pm 1.95*	26.42 \pm 0.06*	309.2 \pm 0.14*
EEAP (200mg/kg b.w) (Group4)	19.86 \pm 1.42*	27.02 \pm 0.27*	304.94 \pm 0.37*

No. of rats in each group: 4; Values are expressed in terms of Mean \pm S.E.M, * $p < 0.0001$ v/s ulcerated control group using one-way ANOVA followed by Dunnett's test

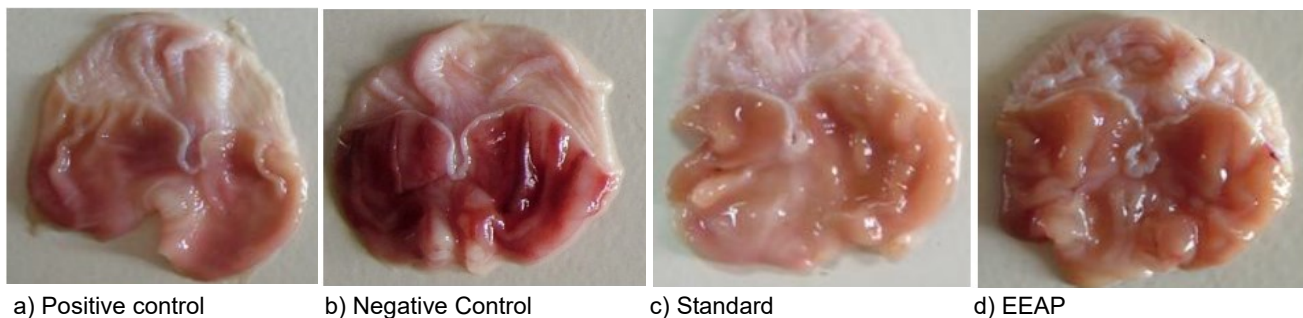


Fig. 2. Rat stomach glandular portion (a) Positive control showing normal architecture (b) Negative control (treated with 1m/kg b.w Ethanol) showing redness, 8-10 red spots & Hemorrhagic streaks. (c) Standard (treated with Ranitidine 45mg/kg b.w) showing 1-2 red spots (d) Test (treated with EEAP 200mg/kg b.w EEAP) showing 1-2 red spots.

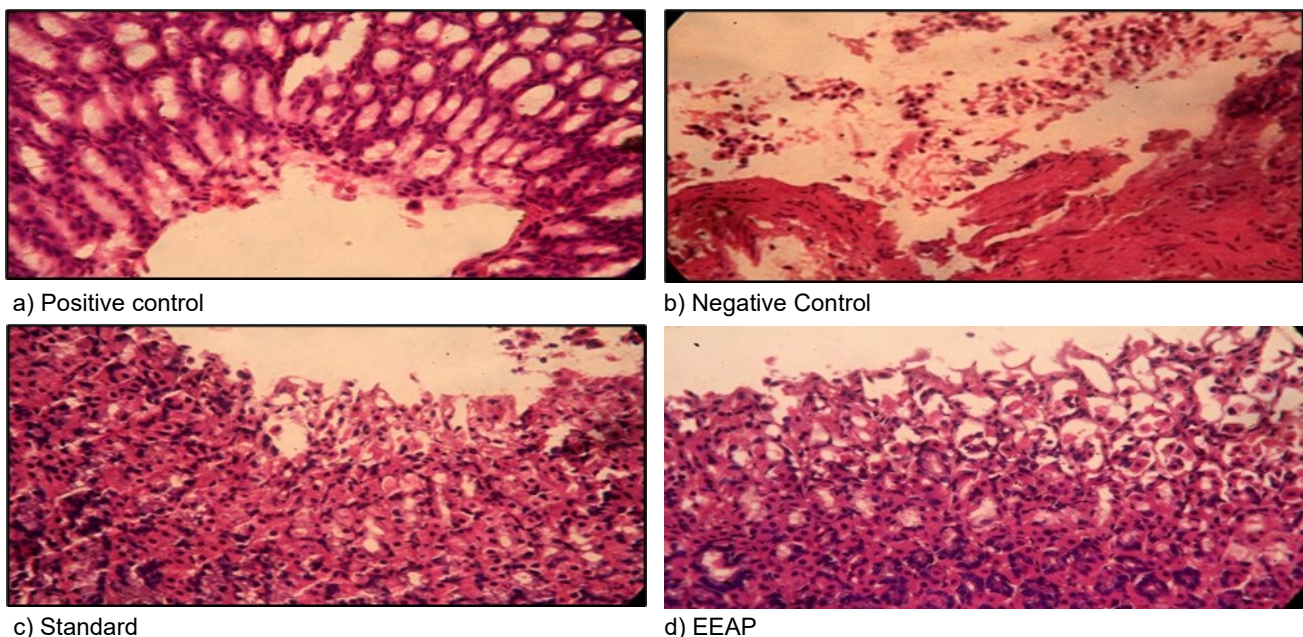
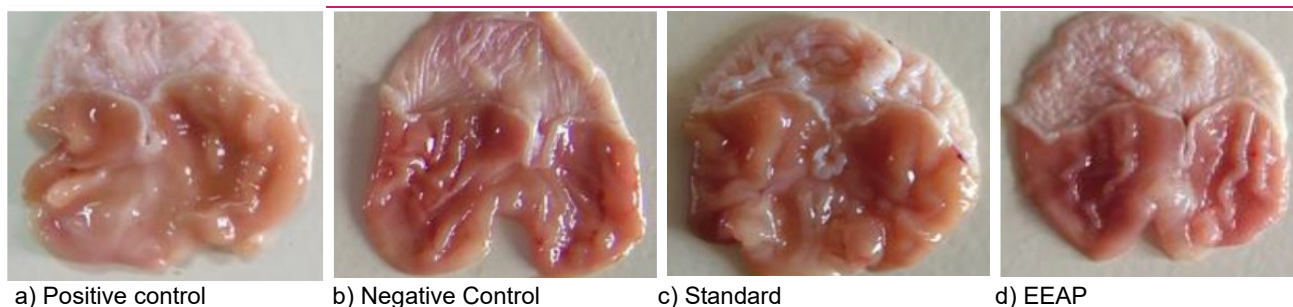


Fig. 3. Histopathological evaluation of Ethanol Induced Gastric Ulcers (a) Positive control showing normal mucosa (b) Negative control showing inflammation & mucosal ulceration control, (c) Standard drug Ranitidine (45mg/kg b.w) showing no significance change in histopathology almost normal appearance, (d) Test group EEAP (200mg/kg b.w) showing no significance change in histopathology almost normal appearance.

leads to faulty disposal of free radicals and their accumulation. These ROS are responsible for the oxidation of tissues leading to lipid peroxidation and tissue damage. The antiulcer effect was also reinforced by increasing catalase activity and decreasing LPO. The

antisecretory and antiulcerogenic activity of *A. procera* observed in the present study was done on the leaves collected from Indo-Gangetic region of India, which showed improvised quality of phytoconstituents due to changed chemotaxonomy which suggests the better



a) Positive control

b) Negative Control

c) Standard

d) EEAP

Fig. 4. Rat stomach glandular portion (a) Positive control showing normal architecture (b) Negative Control (Ulcer induced by pylorus ligation) showing redness, 5-6 red spots & hemorrhagic streaks (c) Standard (treated with Ranitidine 45mg/kg b.w and showing protected mucosal layer with 1-2 red spots only (d) Test (treated with EEAP 200mg/kg b.w and showing protected mucosal layer with 1-2 red spots only

antiulcer activity, cytoprotective activity, *in vitro* and *in vivo* antioxidant activities (Ambika and Jegadeesan, 2017). The mechanism behind its gastroprotective effect in wistar rats may be assigned to reduced vascular penetrability, decreased generation of free radicals and LPO in addition to strengthening of mucosal barrier. Besides, the occurrence of phytoconstituents in the plants, such as flavonoids and tannins, might be responsible for this activity.

Conclusion

The present study concluded that the leaves of *A. procera* possessed antiulcer, antioxidant, antisecretory and cytoprotective property and possibly acted via various mechanisms including free radical scavenging action. The leaves extract significantly showed ulcer healing potential. The leaves extract was found to contain a noticeable amount of total phenols and flavonoids, which might have played a major role in controlling the oxidation and exhibited cytoprotection. The current activity confirmed the antiulcer protective ability of *A. procera* being effective in managing gastric ulcers; therefore, further study is needed to isolate and identify the active principles present in the extract responsible for the antiulcer activity.

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Conflict of interest

The authors declare that they have no conflict of interest.

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