RESEARCH PAPER

Evaluation of Anti-Ulcer Activity of Ethanolic Extract of Parijoto Fruit-An in-Vivo Study

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ABSTRACT

This study investigates the anti-ulcer activity of the ethanolic extract of Parijoto fruit against Aspirin induced and Pylorus ligated ulcers. Gastric ulcer disease is caused by an imbalance between mucosal defense factors and injurious factors, such as acid and pepsin. Ulcers caused by pylorus ligation result from increased gastric acid and pepsin accumulation, leading to auto digestion of gastric mucosa and breaking down the gastric mucosal barrier. NSAIDs are frequently associated with peptic ulcers.

The anti-ulcer effect is supported by decreased aggressive factors like gastric volume, decreased free and total acidity, and increased resistance factors like pH. The anti-ulcer agent may protect the mucosa from acid effects by selectively increasing prostaglandins, which have a vital protective role. The mucosal defense mechanism may be due to the epithelial cells of the gastric mucosa, which are impermeable to H+ ions and form a physical barrier.

The ethanolic extract of Parijoto fruitwas evaluated using the aspirin induced ulcer model, showing dose-dependent inhibition percentages of 53.26% and 63.23% compared to the ulcer control. The standard drug Ranitidine HCL (20mg/kg) showed a percentage inhibition of 71.48% when compared to the ulcer control.

The study also found that the extract can reduce reactive free radicals that might cause oxidative damage to tissue. It also reduced polymorphonucleur infiltration, TNF expression, and ROS formation in peptic mucosa. The anti-ulcerogenic activity of the extract may add to its beneficial effect against peptic ulcer disorder.

Keywords: *Peptic Ulcer, Parijoto fruit, Acid Secretion, Inflammation, Mucosa*

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INTRODUCTION

Peptic ulcers are acid-induced lesions in the digestive tract, affecting 5-10% of the general population. They are characterized by denuded mucosa and defects. However, recent studies show a decrease in incidence, hospital admissions, and mortality. NSAID users and aspirin users have a four-fold increased risk of complications, and the use of these medications with other medications increases the risk of upper gastrointestinal bleeding.[1-3] NSAID-associated damage to the gastroduodenal mucosa occurs due to the systemic inhibition of cyclooxygenase-1, causing decreased mucosal blood flow, low mucus and bicarbonate secretion, and cell proliferation inhibition. Co-administration of exogenous prostaglandins and COX-2-selective NSAIDs reduces damage and ulcer risk.[4-7] Traditional medicine uses plants and herbs to treat gastrointestinal disorders, including gastric ulcers, with carbenoxolone being the first effective drug discovered through research on various plants. [8]

Materials and Methods: *Parijoto fruit was* obtained from a local nursery, Ethanol and CMC was obtained from S.D. Fine Chem, Mumbai, Distilled water was procured fromLoba Chem ,Mumbai. The standard Rantidine HCL

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was procured from Yarrow Chem, Mumbai. All other chemicals and reagents used were of analytical grade.

Preparation of Extract: The *Parijoto fruit* were shade dried and then ground till they became coarse powder in a mortar-pestle. The powdered material thus obtained was subjected to extraction using Ethanol. The extracts obtained were distilled to remove excess of the solvent and then evaporated at 40ºC to get a semi-solid mass. These extracts were subjected to phytochemical tests. [9-10]

Animals: Wistar rats of either sex (150-200gms) were housed in separate cages at controlled room temperature (24 ±2ºC; relative humidity 60-70%) in a 12hr light- dark cycle. They were fed with standard pellet diet and water *ad libitum*.

Determination of Acute Oral Toxicity (Ld_{50}) Of *Parijoto fruit*

Test Substance Details:

Name of the test substance: Ethanolic extract of *Parijoto fruit*.

Color: Greenish Black

Nature of the Test Substance: Gummy

Study Procedure: The Organization for Economic Cooperation and Development (OECD) guideline 425 methodology were used when assessing acute oral toxicity. A specifically made oral needle for mice was used to gavage the extract in a single dosage. The day before the dose, the animals fast (no water, only no food). (OECD Chemical Testing Guidelines, 425). Prior to conducting the study on animals, institutional animal ethics committee clearance was obtained for the handling of the animals and the procedures utilised in the study.

Housing and Feeding Condition: The temperature in the experimental animal room was kept 22±3ºC. Artificial lighting was provided. The animals were acclimatized to standard laboratory conditions of temperature (22 ± 30ºC) and maintained on 12:12 h light: dark cycle. The animals were housed in sanitized polypropylene cages containing sterile paddy husk as bedding. They were provided with regular rat chow diet and distilled water ad libitum. [11]

Preparation of Animals: The animals were randomly selected and kept in their cages for at least 5 days prior to dosing to allow for acclimatization to the laboratory conditions.

Extracts & Standards Used:

Extract used: *Parijoto fruit* Ethanolic extract.

Standard Used:

Ranitidine HCL: 20 mg/kg b.w.

Drugs, viz, Ranitidine HCL and the test extract of *Parijoto fruit* were suspended in 0.5% CMC and used for anti-ulcer studies. Each drug suspension was freshly prepared just before administration.

Preparation and Administration of Doses: The extract was solubilised in 0.5% Carboxy Methyl Cellulose prior to experimental use to obtain the desired concentrations (200 and 400 mg/kg body weight) in 1 ml. The test substances were administered in a single dose using a gastric intubation tube after fasting for 3 to 4 hrs.

Anti-Ulcer Activity: [12-15]

Aspirin Induced Gastric Ulcers: Albino rats were divided into four groups of six animals each. The weight of the animals chosen was between 150 and 180gms. The animals were fasted 36 hrs prior to the commencement of the experiment but were allowed free access to water.

- Group I (control): 0.5% CMC, Group II: 20mg/kg Ranitidine HCL, Group III: ethanolic extract at dose 400mg/kg, Group IV: ethanolic extract at dose 200mg/kg.

All the animals received 200mg/kg of aspirin orally to the rats. In the treatment group, drugs were administered orally 1hr before the administration of aspirin. After 2hrs of treatment with aspirin, animals were sacrificed by an excess dose of ether. The stomachs were removed, opened along the greater curvature and examined for lesions. Lesions severity was determined by ulcer index. The ulcers were scored according to the following scale:

- Normal Coloration – 0, Red Coloration – 0.5, Spot Ulcer – 1, Haemorragic Streaks – 1.5, Ulcer – 2, Perforation - 3

Mean ulcer score for each animal will be expressed as ulcer index. The percentage of ulcer protection was determined as follows:

Statistical Analysis: Statistical analysis was carried out using Graph Pad Prism5 software version 5.04 (Graph Pad prism software Inc.) The values were expressed as mean ± SEM. The statistical analysis was carried out by one way analysis of variance (ANOVA) followed by Dunnet's t-test. P values< 0.05 were considered significant.

Pylorus Ligation Induced Gastric Ulcers: Rats were divided into four groups of six animals each. All the animals selected for the study were of weight between 200-250gms.

- Group I (control), received, 0.5% CMC, Group II (reference standard) was treated with 20mg/kg Ranitidine HCL, Group III treated with 400 mg/kg ethanol extract, Group IV was treated with 200mg/kg ethanolic extract.

% Protection = Control Mean Ulcer Index- Test Mean Ulcer Index × 100/ Control Mean Ulcer Index

Animals in all the groups were fasted for 36 h after the respective assigned treatment and were anaesthetized with anaesthetic ether. The abdomen was opened by a small midline incision below the xiphoid process and pylorus portion of stomach was lifted out and ligated. Precaution was taken to avoid traction to the blood supply. The stomach was sutured with interrupted sutures. Animals were allowed to recover and stabilize in individual cages and were deprived of water during post-operative period. Four hours after the pyloric ligation, the animals were sacrificed by an excess dose of ether. The stomach was carefully removed and the gastric contents were collected. The gastric juice was centrifuged at 1000rpm and gastric volume was measured. Free and total acidites of the supernatant were determined by titration with 0.1 N NaOH and expressed as mEq/ L /100 gms. The stomach was cut open along the greater curvature and pinned onto a soft board for evaluating the gastric ulcers and to calculate ulcer index. Ulcer scoring is done according to the scale mentioned below.

Ulcer Index: After the incision of the stomach at the greater curvature the ulcers were observed. And the number of ulcers was counted using a magnifying glass and the diameter of the ulcers were measured using vernier calipers. The following arbitrary scoring system was used to grade the incidence and severity of lesions.

- Normal coloration – 0, Red coloration – 0.5, Spot ulcer – 1, Hemorrhagic streaks – 1.5, Ulcer – 2, Perforation - 3

Determination of Free Acidity and Total Acidity: The gastric contents were centrifuged at 1000rpm for 10mins. 1ml of supernatant was diluted with 9ml distilled water. A volume of 2ml diluted gastric juice was treated with 0.1 N sodium hydroxide run from a micro burette using 3-4 drops of Topfer's reagent as indicator until a canary yellow colour was observed. The volume of NaOH run down was noted. This corresponds to free acidity.

Further, 2-3 drops of phenolphthalein was added and titrated with NaOH until pink colour was restored. This gives total acidity. Free acidity and Total acidity are expressed in terms of ml of 0.1N HCl per 100 gms of gastric contents. This is the same as mEq/lit. [16] Acidity may be calculated by using the following formula:

$$\text{Acidity} = \text{Volume of NaOH} \times \text{Normality of NaOH} \times 100/0.1$$

Histopathological Evaluation: Gastric tissue samples were fixed in neutral buffered formalin for 24 hours, and histopathological examination was conducted to study the ulcerogenic and anti-ulcerogenic activity of Parijoto fruit's ethanolic extract. The tissues were processed, stained with hematoxylin and eosin, and examined for pathomorphological changes like congestion, haemorrhage, oedema, and erosions.[17]

Biochemical Studies: The ulcer induced part were utilized to perform the different biochemical studies for the assessment of myeloperoxidase, catalase (CAT), superoxide dismutase (SOD), lipid peroxidation (malonaldehyde MDA), protein carbonyl (PCO), and MPO.[18-21]

RESULTS:

Results of Preliminary Phytochemical Evaluation: The colour of the Alcoholic test extract (Ethonolic extract of ***Parijoto fruit***) was found to be dark green in colour and the consistency was found to be sticky. The extracts were subjected to phytochemical screening for the presence of type of phyto-constituents. The phytochemical screening revealed that ethonolic extract contains resins, alkaloids, Phytosterols, Phenols and Flavonoids. Of the phytosterols, the ones identified were triterpenes. The results shows that the ethanolic extract contains a mix of bioactive compounds like alkaloids, flavonoids, phenols, tannins, and triterpenoids, which are often associated with medicinal properties. However, some secondary metabolites like saponins, glycosides, and steroids were not detected, possibly due to solvent selectivity or their absence in the source material.

Acute Toxicity Study: Up to a dosage level of 2000 mg/kg body weight, the ethanolic extract of Parijoto fruitdid not cause any toxic symptoms or fatality. During the observations up to a 24-hour period for mortality, there was no evidence of toxicity or alteration in behavioural pattern. As a result, the extract was deemed safe for pharmacological analysis. The biological assessment was conducted using dosages of 200 and 400 mg/kg.

Toxicological evaluations of ethanolic extract of *Parijoto fruit:* Study on the effects of Parijoto fruitethanolic extract on mice revealed that, when given orally at doses of up to 2000 mg/kg, the extract did not cause any toxicity or death in the mice. The results shows that the tested substance showed no abnormalities (N) in alertness, roaming, sniffing, depression, gripping strength, writhing, pupils, urination, salivation, skin color, and lacrimation. However, some responses were absent (A), including grooming, anxiety, tremors, convulsions, scratching, and defecation, suggesting possible sedation or lack of stimulatory effects at the tested dose. No severe toxic reactions were observed.

Anti-Ulcer Evaluation:

Aspirin Induced Gastric Ulcers: Table 3 displays the apparent effect of Ranitidine HCL and Extract on the Ulcer Index and the level of mucosal damage in the stomach. In Group I (control animals), oral aspirin treatment resulted in distinctive lesions in the glandular area of the rat stomach, which showed as elongated bands of thick, black, and dark red lesions. Group II animals pretreated with the standard medicine, Ranitidine HCL, demonstrated significant protection from ulcers in the stomach mucosa, and in Group III and IV, EEAS considerably decreased the ulcer Index at 400mg/kg and 200mg/kg dosages, respectively. Ranitidine HCL, as a reference, provided ulcer prevention. Pretreatment considerably decreased the severity of haemorrhage and lesions, demonstrating EEAS' protective impact.

Table 1: Results of Anti-Ulcer Evaluation by Aspirin Induced gastric Ulcers

Groups	Ulcer Index	% Protection
Group I- Control	12.24±0.06	--
Group II- Standard (Ranitidine HCL)	3.49±0.08***	71.48 %
Group III – *Parijoto fruit Linn* Extract 400mg	4.5±0.10***	63.23%
Group IV – *Parijoto fruit Linn* Extract 200mg	5.72±0.05*	53.26 %

All values represent Mean ± SEM, n=6 in each group. P < 0.05. Control group (Group I) is compared with standard and extract doses, * represents significance.

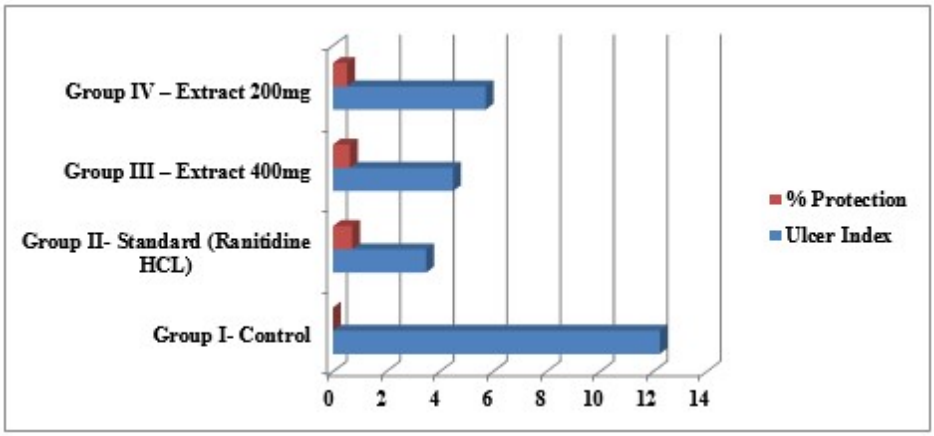

Figure 1: Graphical Representation of Anti-Ulcer Evaluation by Aspirin Induced gastric Ulcers

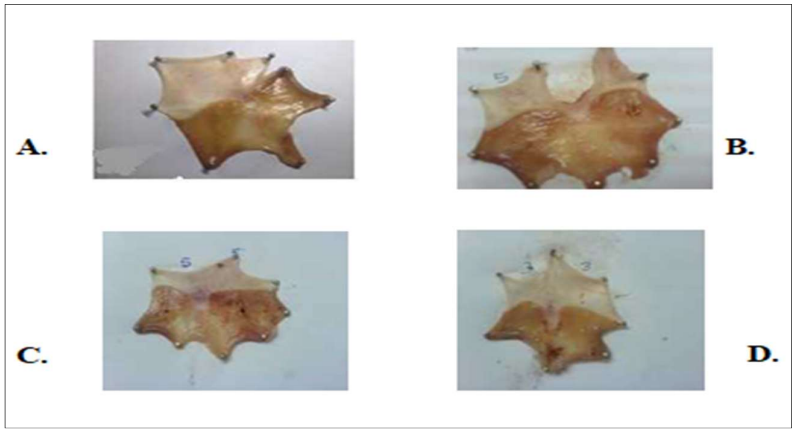

Figure 2: Aspirin Induced Ulcers and their Treatment

(A) – Control; (B) – Standard; (C) – Extract 400mg/kg; (D) – Extract 200mg/kg

Pylorus Ligation Method: In the pyloric ligation induced ulcer model, oral administration of EEAS at two distinct dosages (200mg/kg and 400mg/kg) resulted in a substantial reduction in ulcer index, stomach volume, free acidity, and total acidity when compared to the control group. In comparison to the control, EEAS showed a protection index of 52.3% and 36.28% at doses of 400mg/kg and 200 mg/kg, respectively, whereas Ranitidine HCL, the reference standard medicine, showed a 70% protection percentage. The ulcer protection percentage of EEAS at 200mg/kg is 36.28%, which is regarded less significant in the context of the research.

Table 2: Results of Anti-Ulcer Evaluation by Pylorus Ligation Method

Groups	Ulcer Index	% Protection
Group I- Control	11.3 ± 0.11	--
Group II- Standard (Ranitidine HCL)	3.39 ± 0.07***	70%
Group III – *Parijoto fruit Linn* Extract 400mg	5.48 ± 0.07***	52.3%
Group IV – *Parijoto fruit Linn* Extract 200mg	7.2 ± 0.06*	36.28%

All values represent Mean ± SEM, n=6 in each group. P < 0.001. Control group is compared with standard and extract doses.

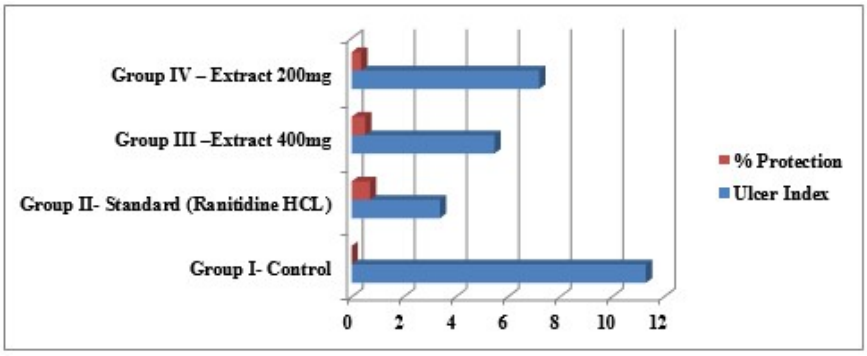

Figure 3: Graphical Representation of Anti-Ulcer Evaluation by Pylorus Ligation Method

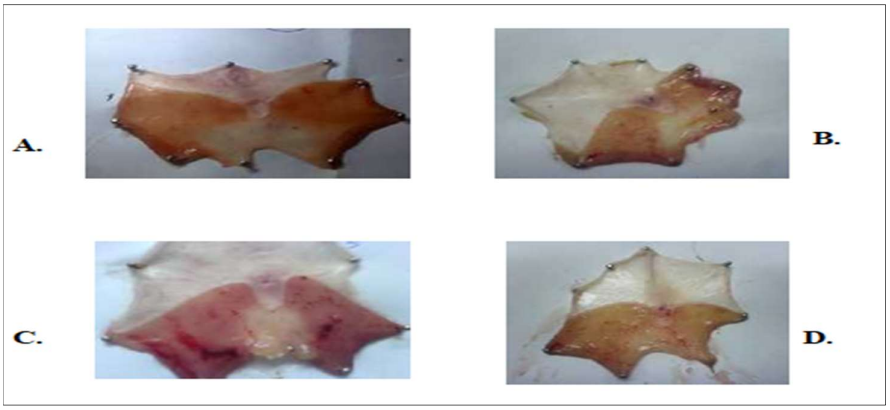

Figure 4: Pylorus Ligation Method and their Treatment

(A) – Control; (B) – Standard; (C) – Extract 400mg/kg; (D) – Extract 200mg/kg

Histopathology Examination: The photos and reports show a submucosal edoema with mixed inflammatory infiltrate, including lymphocytes and neutrophils. The muscularis propria appears to be normal, and macrophage and neutrophil aggregation can be detected on the epithelial cell lining. In Group II, the conventional group demonstrated no stomach mucosal injury. The histological sections of group III treated with EEAS 400 mg/kg indicate mucosal moderate edoema, scattered chronic inflammatory infiltration, and congested vascular spaces. The muscularis propria seems normal. The slice of IV-group rats demonstrates mucosal edoema with inflammatory infiltrate. The muscularis propria looks to be normal.

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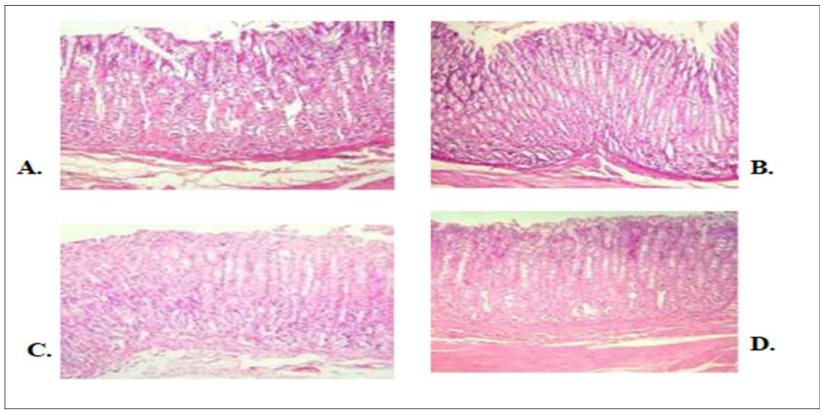

Figure 5: Histopathological Study of Aspirin Induced Ulcers and their Treatment

(A) – Control; (B) – Standard; (C) – Extract 400mg/kg; (D) – Extract 200mg/kg

The histological analysis of the rats' stomachs revealed a more detailed picture of the gastric lesions and mucosal damage. Acute stomach ulceration was seen in Group I, while Group (A) (control group rats) had mucosal ulcers comprised of necrosis, cellular debris, neutrophils, and degenerated epithelial cells. Group II(B) includes stomach mucosa with intact epithelium, lamina propria, and muscularis mucosa. Group (C) demonstrates an intact mucosa with lots of regenerating epithelial cells. Group (D) exhibits localised mucosal ulceration composed of degraded cells and a few regenerated epithelial cells.

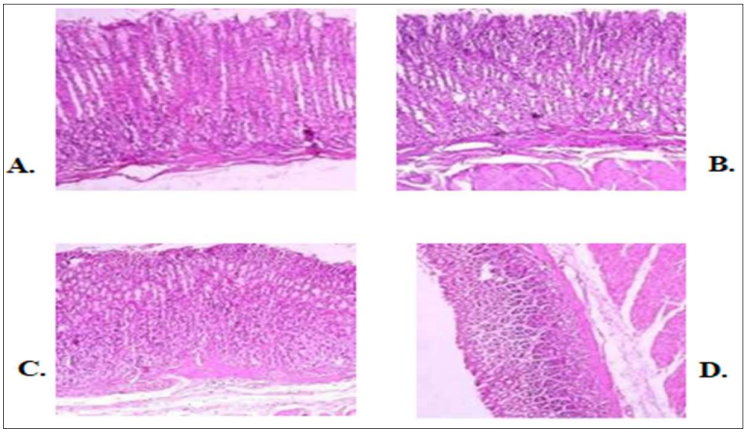

Figure 6: Histopathological Study of Pylorus Ligation Method and their Treatment

(A) – Control; (B) – Standard; (C) – Extract 400mg/kg; (D) – Extract 200mg/kg

Gastric volume, Free Acidity and Total Acidity: The study shown in Table 5 examines the effects of acid secretory parameters on rats induced by ligation-induced gastric ulcers. The control group showed higher estimates, but EESP administration significantly reduced all parameters, equivalent to the standard medication Ranitidine HCL 20 mg/kg. The ethnologic extract also showed a reduction in mean gastric juice at both 400 and 200mg/kg, indicating an anti-secretary mechanism and a dose-dependent action of EEAS.

Table 3: Results of Gastric volume, Free Acidity and Total Acidity

Group	Gastric Volume	pH	Free Acidity	Total Acidity
Control	3.52 ± 0.02	1.41 ± 0.12	55.01 ± 2.28	67.79 ± 1.31
Std (Ranitidine HCL)	1.28±0.05***	5.21±0.13***	20.90±0.76***	32.06±3.316***
Extract 400mg	1.598±0.03**	3.5±0.08***	29.86 ± 0.77**	41.96±0.715***
Extract 200mg	1.86 ± 0.04**	4.63±0.14***	39.66 ± 1.14*	55.56 ± 0.99***

All values represent Mean ± SEM, n=6 in each group. P < 0.001. Control group is compared with standard and extract doses.

Biochemical Studies: The study found that a formulation of enzymes, specifically Catalase, can reduce reactive free radicals and inflammation in ulcerative colitis. Acetic acid, which causes ulcerative colitis, was found to have elevated

levels of LPO. The formulation also reduced myloperoxidase, an inflammation marker in neutrophil granules. The antioxidant defense system was found to be functioning, with reduced inflammation and potential protection against ulcerative colitis.

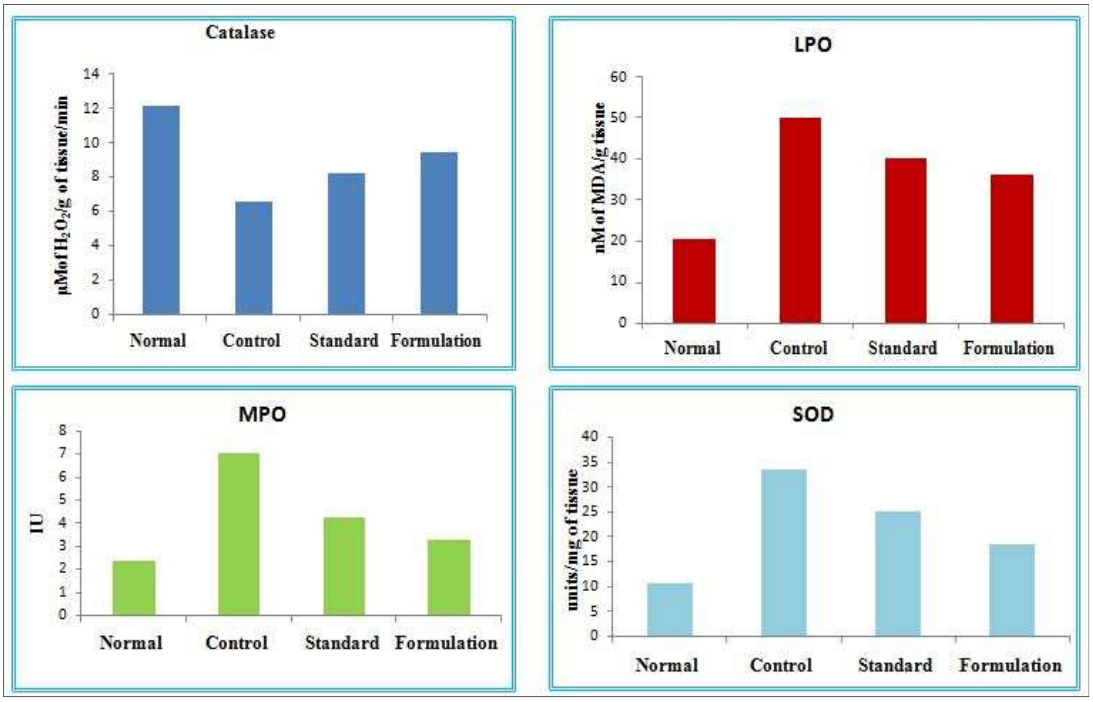

Figure 7: Various Biochemical Study in Peptic Ulcer

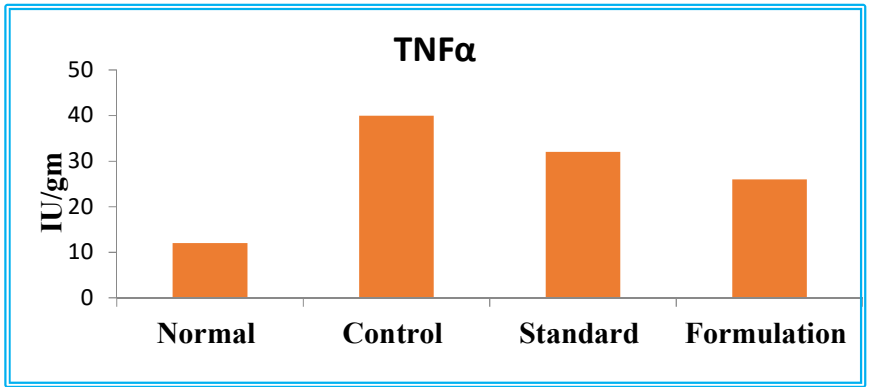

Figure 8: TNFα activity in Peptic Ulcer

Acetic acid, when used in a formulation, can suppress free radical formation by restoring the redox state of the peptic mucosa. This anti-ulcerogenic efficacy is supported by its anti-inflammatory and antioxidant properties. The study assessed Catalase, SOD, MPO, and TNFα levels, finding significant differences between the created and Control groups.

DISCUSSION:

The present study demonstrates that the ethanolic extract of *Parijoto* fruit possesses appreciable anti-ulcer activity in experimental rat models of aspirin-induced and pylorus ligation-induced gastric ulcers. The observed activity appears to involve more than a single mechanism, as the extract reduced ulcer severity while also influencing gastric secretion, acidity, oxidative stress, and inflammatory responses. The preliminary phytochemical screening identified flavonoids, phenols, alkaloids, tannins, phytosterols, and triterpenes in the extract. These constituents may collectively contribute to the gastroprotective effect because several classes of plant-derived phenolic and flavonoid compounds are associated with antioxidant and mucosal protective activities.

The aspirin-induced ulcer model provided clear evidence of the protective potential of the extract. Aspirin administration produced marked gastric lesions in the control animals, whereas pretreatment with the *Parijoto*

fruit extract reduced the severity of mucosal damage. The 400 mg/kg dose produced an ulcer index of 4.50 ± 0.10 and 63.23% protection, while the 200 mg/kg dose produced an ulcer index of 5.72 ± 0.05 and 53.26% protection. Although both doses were effective, the higher dose produced greater protection, indicating a dose-related response. The standard drug ranitidine showed 71.48% protection, suggesting that the activity of the higher extract dose approached that of the reference treatment.

The findings from the pylorus ligation model further support the gastroprotective action of the extract. Pylorus ligation promotes accumulation of gastric secretions and increases exposure of the mucosa to acid, thereby facilitating ulcer formation. In the present study, the 400 mg/kg extract reduced the ulcer index to 5.48 ± 0.07 and produced 52.3% protection, whereas the 200 mg/kg dose produced 36.28% protection. Ranitidine produced 70% protection. Importantly, treatment with the extract also reduced gastric volume, free acidity, and total acidity while increasing gastric pH. At 400 mg/kg, gastric volume decreased to 1.598 ± 0.03, compared with 3.52 ± 0.02 in the control group, while pH increased from 1.41 ± 0.12 to 3.50 ± 0.08. These findings suggest that suppression of aggressive gastric secretory factors may contribute substantially to the observed anti-ulcer effect.

Histopathological observations provide additional support for these findings. Control animals exhibited pronounced mucosal injury, including necrosis, cellular debris, inflammatory-cell infiltration, and degeneration of epithelial cells. In contrast, the standard-treated group showed essentially intact gastric architecture. Animals receiving 400 mg/kg extract demonstrated comparatively preserved mucosa with regenerating epithelial cells, whereas the 200 mg/kg group showed localized ulceration with fewer regenerated cells. The relatively better tissue preservation at the higher dose is consistent with the biochemical and macroscopic ulcer findings.

The biochemical observations indicate that antioxidant and anti-inflammatory mechanisms may also participate in the protective effect. The study evaluated catalase, SOD, MPO, TNF-α, lipid peroxidation, and related oxidative parameters, with the findings indicating differences between ulcerated and treated groups. The extract's phytoconstituents may help limit oxidative damage and inflammatory responses within gastric tissue. Thus, the overall gastroprotective action of *Parijoto* fruit extract may result from a combination of reduced acid secretion, improved mucosal resistance, antioxidant activity, and attenuation of inflammation.

The acute toxicity findings also provide preliminary evidence of tolerability, as no mortality or major toxic behavioral changes were observed up to 2000 mg/kg, allowing 200 and 400 mg/kg doses to be used for pharmacological evaluation. Overall, the findings suggest that ethanolic *Parijoto* fruit extract has promising gastroprotective potential. However, further studies involving compound isolation, molecular investigations, standardized extract preparation, and longer-term safety evaluation are necessary before its therapeutic relevance can be established.

CONCLUSION: The study reveals the potential of Parijoto fruit as a natural remedy for managing ulcers and inflammation. It emphasizes the need for further research to understand its mechanisms and determine effective dosage and administration strategies. The results suggest a promising future for incorporating the extract as a treatment option for ulcers, but further studies are needed to assess its long-term safety and efficacy profile for safe and reliable integration into clinical practices.

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