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# Evaluation of Gastro Protective Activity of Ethanolic Leaf Extract of*Cassia grandis* Linn against Aspirin Induced Peptic ulcer on Swiss albino Wistar Rats

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## Abstract

This study examines the gastro protective activity of ethanolic leaf extract of*Cassia grandis* Linn against aspirin induced peptic ulcers on swiss albino wistar rat. Oral aspirin treatment (300 mg/kg body weight) caused ulceration. Omeprazole (standard) was given to swiss albino wistar rats at a dose of 20 mg/kg body weight, and *Cassia grandis* Linn was given at doses of 200 and 400 mg/kg body weight once daily for 14 days prior to the development of ulcers. Gastric secretions parameters were assessed after the experiment. After treatment with ethanolic leaf extracts of *Cassia grandis* Linn, efficiently reduced the elevated ulcer index, stomach volume, and pepsin activity.Additionally, the extract significantly increasingthe mucin concentration and lowered the pH activity in the ulcerated rats.The plant extract also showed gastro protective activity of 60.6% and standard drug Omeprazole was showed 76.5% of activity. These results point that the extract has the potential for gastro protective activity.

***Keywords:****Cassia grandis* Linn, gastro protective, ethanolic extract, Omeprazole andAspirin.

## INTRODUCTION

The most prevalent gastrointestinal ailment in the world with high mortality and morbidity is peptic ulcer disease (PUD) [1]. It is a digestive tract disease (GIT) that can cause both gastric and duodenal ulcers. Pepsin, stomach acid, and Helicobacter pylori are among the variables that are out of balance, whereas prostaglandins, bicarbonate ions, mucin, growth factors, and nitric oxide are among the defensive ones [2]. The main consequence, which involves a significant risk of mortality and morbidity, is a perforated stomach ulcer. A 5% prevalence of perforation affects over 4 million patients annually worldwide. Stomach mucosal injury brought on by damaging causes such as gastric acid (HCl), reactive oxygen species (ROS), ethanol, anti-inflammatory medications, and pathogenic attack of *H. Pylori* results in gastric ulceration.Drinking too much alcohol compromises the integrity of the stomach mucosa, which results in gastric ulceration. In India, the burden of peptic ulcer disease accounted for 0.66% of all fatalities, according to WHO (World Health Organization, 2019). In 2013 [3], it killed over 301,400 people worldwide. In India, patients who underwent surgery for PUD reported that 86% had duodenal ulcers while the remaining 14% had gastric ulcers. Surgery was required for the primary problems, such as perforation (35%), bleeding (7%), obstruction (30%), and chronic case (28%), and the overall death rate was 5.7% [3].

Non-steroidal anti-inflammatory drugs (NSAID) usage and H. Pylori were frequent etiologic factors for PUD. By lowering the levels of COX-1 and prostaglandins through their extended activity, NSAIDs' anti-inflammatory and analgesic actions severely harm the GI tract. PUD risk factors include stress, alcohol use, smoking, the Zollinger Ellison syndrome, and age-related declines in prostaglandin levels as a severe gastrointestinal illness, PUD necessitates a therapy strategy that is well-targeted. There are many medications that can be used to treat PUD, including proton pump inhibitors, $H_2$ receptor antagonists, and anti-acids. Antiulcer medications now on the market can have negative side effects such hypersensitivity, tachycardia, impotence, gynecomastia, haematological abnormalities, and kidney disease. Significant drug-drug interactions caused by these medications also have a negative impact on the usage of these substances [4]. Globally, primary healthcare is largely provided through traditional medicine. Because traditional medicine is more socially and culturally acceptable, it is more compatible with the human body, and it has less negative side effects, approximately 75–80% of people in underdeveloped nations use it [5].

## MATERIALS AND METHODS

### Plant material

The fresh leaves of *Cassia grandis* Linnwere collected from Viralimalai, Puthukottai District, Tamilnadu, India. The plant was authenticated by Botanist Dr. P. Radha, Research Office. The 500 g plant materials sliced into small circular pieces and were shade driedfor 6 days. The dried pieces subjected for grinding to coarse powder. The coarse powder wasextracted with various solvents by continuous hot percolation method and the Percentage yields of various extracts were calculated.

### Phytochemical analysis

The preliminary phytochemical screening of the variousleaves extract of *Cassia grandis* was performed for the qualitative analysis of alkaloid, flavonoid, glycoside, steroid, polyphenols, saponin, reducing sugar,and tannin [6,7]

### In-vitro antioxidant Activity

### DPPH scavenging activity

A solution of 0.1 mM DPPH (2,2-diphenyl-1-picrylhydrazyl) in methanol was prepared, and 2.4 ml of this solution was mixed with 1.6 ml of extract in methanol at different concentrations (20–100 μg/ml). The reaction mixture wasthoroughly mixed and kept in the dark at room temperature for

30 min. Free radical scavenging ability of the extracts was tested by DPPH radical scavenging assay. The hydrogen atom donating ability of the plant extractives was determined by the decolourization of methanol solution of DPPH. DPPH produces violet/purple colour in methanol solution and fades to shades of yellow colour in the presence of antioxidants. The absorbance of the mixture was measured spectrophotometrically at 517 nm [8,9]. Ascorbic acid was used as standard. Percentage DPPH radical scavenging activity was calculated by the following equation:% inhibition of DPPH radical = $[A_{br} - A_{ar}] / A_{br} \times 100$

Where $A_{br}$ is the absorbance before reaction and $A_{ar}$ is theabsorbance after reaction has taken place.

**Ferric reducing-antioxidant power (FRAP) assay**

The Benzie and Strain method can be used to conduct antioxidant assay[10]. 3ml of prepared FRAP reagent was mixed with 100 ml of diluted sample andincubated for 30 minute at 37 °C. Using a diode-array spectrophotometer, the change in absorbance at 593 nm was measured to track the decline.By using this technique, antioxidants' capacity to scavenge ferric iron was measured. Its reduction of the ferrous form of the combination of ferric iron and 2,3,5-triphenyl-1,3,4-triaza-2-azoniacyclopenta-1,4-diene chloride (TPTZ) at 3.6pH.FRAP values, which are given as mM of Fe2+ equivalents per kg of sample, can be calculated by comparing the absorption change in the test combination with those obtained from increasing concentrations of Fe3+.

**Determination of Total Phenolic and Flavonoids Content**

**Total Phenolic Content.**

Folin-phenol Ciocalteu's reagent (0.2 ml) was added to the test sample (0.2 ml) along with 0.6 ml of water. After 5 minutes, the mixture was added 1 ml of saturated sodium carbonate solution (8% w/v in water), and the volume was then increased to 3 ml with distilled water. After centrifuging the reaction for 30 minutes in the dark, the absorbance of blue hue in various samples was measured at 765 nm. Using the Folin and Ciocalteu reagent, the total phenolic content of the extracts was calculated. Sample and standard readings were taken using a spectrophotometer at 765 nm in comparison to the reagent blank. On the basis of a gallic acid standard curve, the phenolic content was estimated as gallic acid equivalents GAE/g of dry plant material [11].

**Total Flavonoids Content**.

The total flavonoid content of the sample was determined using the aluminium chloride colorimetric technique [12]. Quercetin was utilised to create the standard calibration curve for the measurement of total flavonoid concentration. The standard quercetin solutions were made by serially diluting quercetin with methanol (5-200 g/ml) after the stock quercetin solution was made by dissolving 5.0 mg of quercetin in 1.0 ml of methanol. Standard quercetin extracts or solutions in a volume of 0.6 ml were separately combined with 0.6 ml of 2% aluminium chloride. The mixture was then incubated for 60 minutes at room temperature. The absorbance of the reaction mixtures was measured against a blank at 420 nm. The calibration plot was used to compute the concentration of total flavonoid content in the test samples, which was then represented as mg quercetin equivalent (QE)/g of dried plant material. [13].

**In-vitro Evaluation of Antiulcer Activity**

**Acid Neutralizing Capacity:**

The acid-neutralizing capacity (ANC) is the amount of acid that can be neutralized by an antacid. The ethanolic leafextracts of *Cassia grandis* Linn at various concentrationsof 100 mg, 500 mg, 1000 mg, and 1500 mg were measured. Magnesium hydroxide (500 mg) and aluminium hydroxide have been compared for the standard. Taken 5ml of the extract and adding the 65ml of water, the total volume was 70ml. This was blended for one minute. The 30ml of 1.0 N HCl was added to the

standard and test preparation and swirled for 15 minutes. Then, phenolphthalein was added. The HCl was titrated until the pink colour was achieved using 0.5N sodium hydroxide. The moles of acid neutralized is calculated by, Moles of acid neutralized = (vol. of HCl × Normality of HCl) - (vol. Of NaOH × Normality of NaOH) Acid neutralizing capacity (ANC) per gram of antacid = moles of HCl neutralized divided by Grams of Antacid/Extract [14].

**Assay of H + -K + ATPase activity**

Taken a 1 mlvolumeof the ethanolic leafextract of*Cassia grandis* Linn (20–100 µg/ml) was incubated with 5ml reaction mixture (40 mMTris–HCl buffer, pH 7.4, 2mM $MgC1_2$, and 10 g membrane protein). The reaction was then initiated using 2 mM ATP Tris salt, and this preparation was incubated for 20 min at 37 °C. By adding 1 ml of ice-cold, 10% v/v trichloroacetic acid, the process was stopped. Different doses of the extract and omeprazole were tested to measure the H +-K + ATPase activity. The amount of inorganic phosphate released from ATP was measured using spectrophotometry at 400 nm [15,16].

***In-vivo* of Antiulcer Activity**

**Experimental Animals**

Swiss albino wistar rats (200–250 g, 12–16 weeks) were used in the experiment. These animals were kept in the animal house of the Department of Pharmacology at Vinayaka Mission's College of Pharmacy in Salem and purchased from the Government Erode Medical College atPerundhurai. The experiment was subjected toCPCSEA approval and was cleared by the same(P.col/42/2022/IAEC/VMCP).

**Experimental design**

30 male healthy swiss albino wistar rats were used for this study. The animals were divided into 5 group each group contains 6 animals. Group Iwas normal Control they received normal saline for 14 days. Group II wasUlcer controltreated with Aspirin(300mg/Kg), orally for last 4 day.Group III was pre-treated withOmeprazole (20mg/kg) for 14 day and received Aspirin (300mg/Kg) orally. Group IV waspre-treated with EECG (200 mg/kg) for 14 day and received Aspirin (300mg/Kg) orally.Group V waspre-treated with EECG (400 mg/kg) for 14 day and received Aspirin (300mg/Kg) orally.

The Aspirin was administered orally to group III to group V animals an hour after the previous dose of standard and sample extract for last 4 days.On the 15th day the animals were sacrificed, stomachs were isolated and then dissected out. The estimation of gastric content and microscopic evaluation was performed [17].

**Estimation of gastric content**

***Volume of gastric content***

Volume of gastric contents was measured by pouring gastric contents carefully in graduated cylinder.

***Determination of pH***

A pH metre was used to measure the pH of a solution made by diluting 1 ml of gastric juice with 9ml of distilled water.

***Determination of total acidity***

A 50 ml conical flask was filled with an aliquot of 1 ml of gastric juice that had been diluted with 9ml of distilled water. Two drops of the phenolphthalein indicator were then added, and the mixture was titrated with 0.01N NaOH until a persistent pink colour was seen. It was observed how much

0.01N NaOH was utilised [18,19]. The following formula was used to determine the total acidity, which was represented as mEq/L:

$$\text{Acidity} = \frac{V_{NaOH} \times N \times 100\text{mEq/L.}}{0.1}$$

Where V is volume and N is normality

***Determination of Gastric Mucus Content.***

The gastric glandular mucous was measured by the method of Corne*et al*[20]. The gastric glandular portion of the stomachs were transferred for 2 h to 0.1% alcian blue dissolvedin buffer solution containing 0.1 mol/L sucrose and 0.05 mol/L sodium acetate (pH adjusted to 5.8 with hydrochloric acid). After washing the stomach twice in 0.25 mol/L sucrose (15 min and 45 min), the dye complexed with mucous was eluted by immersion in 10 mL aliquots of 0.5 mol/L $MgCl_2$ for 2 h. The resulting blue solution was shaken with equal volumes of diethyl ether and the optical density of the aqueous phase was measured spectrophotometrically at 605 nm.

**Microscopic Evaluation of Stomach.**

The stomachs were then dissected along the larger curvature, cleaned with ordinary saline to remove gastric contents, and looked at under a 10x magnification to check for ulceration. The ulcer score, ulcer index and % inhibition of ulcer was calculated [21].

**Histopathological evaluation**

Stomach samples were fixed in 10% formalin and then embedded in paraffin wax for histological analysis. The embedded stomach blocks were sliced into 3-5mm sections with thehelp of an ultramicrotome and processed for haematoxylin and eosin (H & E) staining. Thepathological alterations in stomach tissue were assessed under light microscopy [22].

**Statistical Analysis**

Results were expressed as mean ± SEM and were analyzed using Windows SPSS version 20.0. Comparisons were made between negative control, positive control, and treatment groups of varied doses using ANOVA following by Post hoc Tukey's HSD multiple comparison tests. At a 95% confidence interval, $p < 0.01$ was considered as statistical significance [23].

**RESULTS**

**Phytochemical evaluation:**

**Preliminary Phytochemical screening of leaves extract of *Cassia grandis* Linn.**

The extractive valve of 500 g of the powdered leaves material of *Cassia grandis* on various solvents extracts of Petroleum ether, Chloroform,Acetone, Ethanol and Aqueous were 5.3%, 4.5%, 5%,10% and 7.8% were obtained.

From the results of Phytochemical screening of various extracts, it showed the presence of alkaloids, carbohydrates, flavonoids, glycosides, phenolic compounds, saponins, tannins, and terpenoids in the Petroleum ether extract.Chloroform extract showed the presence ofalkaloids, carbohydrates,glycosides, terpenoids and protein and animo acid.Acetone extractshowed the presence ofalkaloids, carbohydrates, flavonoids, glycosides, phenolic compounds,saponins, tannins, and terpenoids.Ethanolic extract showed the presence ofalkaloids, carbohydrates, flavonoids, glycosides, phenolic compounds,Saponins, tannins, terpenoidsand protein and animo acid.Aqueous extract showed the presence ofalkaloids, carbohydrates, flavonoids, glycosides, phenolic compounds, saponins, tannins, and terpenoids.

Based on the results of extractive value and phytochemical analysis, the ethanolic extract of *Cassia grandis* (EECG) was chosen for the evaluation of Pharmacological activity.

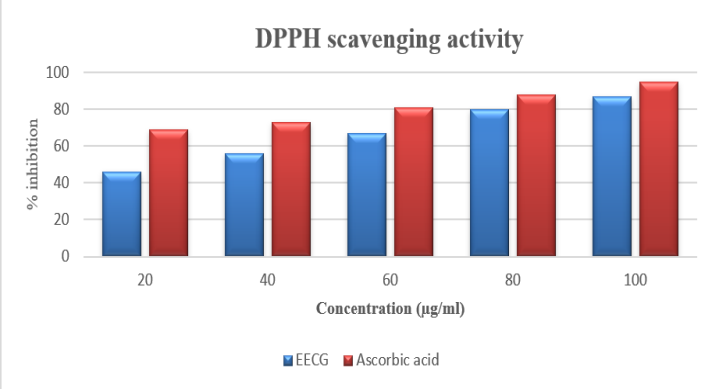
## PHARMACOLOGICAL STUDIES

### *In-vitro* anti-oxidant studies

### DPPH scavenging activity

The *in-vitro* anti-oxidant effect of the EECG was studied at various concentrations (20µg, 40µg, 60µg, 80µg and 100µg) and compared with standard Ascorbic acid. The EECG has the significant activity. The activity was concentration dependant. Maximum % inhibition activity of EECG was87.90±1.61% at a concentration of 100µg, and Ascorbic acid showed 95.37±0.85%.

**Figures 1:**Effect of EECG on DPPH scavenging activity




### FRAP Assay

The *in-vitro* total antioxidant capacity was evaluated by FRAP assay method using Ascorbic acid as standard. The EECG and the Ascorbic acid showed the FRAP activity of 743.33±13.01µmol Fe (II)/g. and 1026.67±17.15 µmol Fe (II)/g.

### Total phenolic and flavonoid contents.

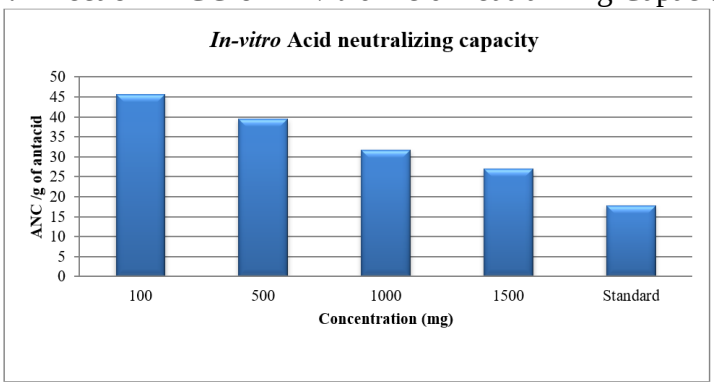
The Total phenolic and flavonoid content of EECG was evaluated. The Total phenolic content present in the EECG was 56.93±3.93 mg of gallic acid equivalent/ g dry materialand Total flavonoid content present in the EECG was 52.66±3.20mg of quercetin equivalent/g dry material.

### *In-vitro* anti-ulcer studies

### *In-vitro* Acid Neutralizing Capacity:

The *in-vitro* Acid neutralizing effect of the EECG was studied at four concentrations (100mg, 500mg, 1000mg, and 1500mg) were compared with standard Aluminium Hydroxide + Magnesium Hydroxide (500mg). The activity was concentration dependant. Maximum ANC of EECG was 26.94 ANC/gram of Antacidat a concentration of 1500mg, and the Standard showed 17.8ANC/gram of Antacid.

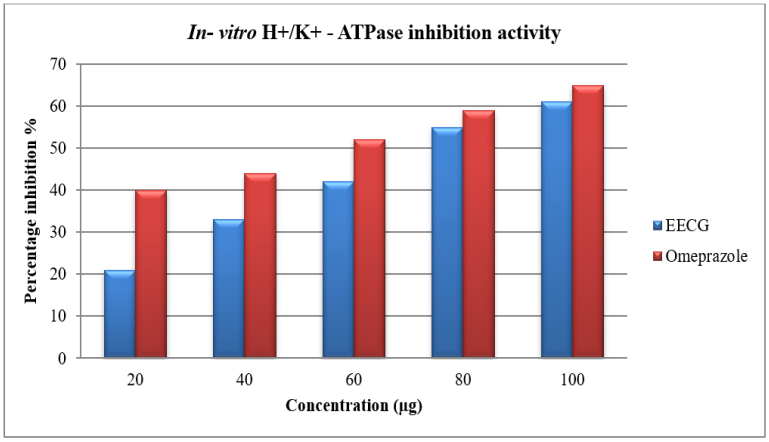
**Figures 2:** Effect of EECG on *in-vitro* Acid Neutralizing Capacity (ANC)




***In-vitro* H+/K+ - ATPase Inhibition Activity:**

The H+/ K+ - ATPase inhibition activity of EECG at various concentrations (20µg, 40µg, 60µg, 80µg, and 100µg) were compared with Omeprazole. The EECG demonstrated significant proton pump inhibitory activity in the gastric mucosal homogenate. The inhibitory activity was concentration dependent. Maximum percentage inhibitory activity was observed at a concentration of 100µg of EECG was 61.45±2.51% and standard Omeprazole showed 65.37±0.85% inhibition.

**Figures 3:** Effect of EECG on *in- vitro* H+/K+ - ATPase inhibition activity




***In-vivo* antiulcer studies**

In the *in-vivo*evaluation, the gastro productive activity of EECG was evaluated against aspirin induced peptic ulcer followed by the gastric content estimation and microscopic evaluation are done by following methods.

**Aspirin induced Ulcer model**

**Volume of gastric juice**

The EECG demonstrated significant reduction in the gastric volume at concentration dependent. The Volume of gastric juice of EECG at a 400mg/Kg was found to be 2.0±0.21*ml/100mg, Omeprazole showed 1.72±0.23*ml/100mg and Aspirin showed 2.8±0.14*ml/100mg.

**pH of gastric juice**

The EECG demonstrated significant reduction in the gastric pH at concentration dependent. The pH of EECG at a 400mg/Kg was found to be 4.3±0.05*, standard Omeprazole showed 5.06±0.06*and Aspirin showed 2.36±0.05*.

**Total Gastric acidity**

The total gastric acidity was decreased in high dose of EECG. The EECG demonstrated significant reduction in the total gastric acidity. This total gastric acidity was concentration dependent. Total Gastric acidityofEECG at a 400mg/Kg was found to be 30.94±0.12* mq/L/100mgand standard Omeprazole was 26.96±0.18*mq/L/100mg.

**Mucin content**

When compared to the normal group, aspirin treated group significantly decreased the mucin content of the gastric juice of the rats. The EECG at high dosesignificantly increased the mucin content. Mucin contentpresent in EECG at a 400mg/Kg was 290.37±0.87* and the standard Omeprazole showed 330.36±0.54*.

**Tables 1:** Effect of EECG on gastric contents

| S.No | Group | Vol. of Gastric juice (ml/100mg) | pH | Total Gastric acidity (mq/L/100mg) | Mucin content (µg/ml) |
|---|---|---|---|---|---|
| 1. | Control (Normal saline-1ml) P.O | 1.50±0.13 | 4.65±0.16 | 23.34±0.46 | 380.50±0.25 |
| 2. | Ulcer control (Aspirin 300 mg/kg) P.O | 2.82±0.14* | 2.36±0.05* | 55.6±0.76* | 185.42±0.63* |
| 3. | Omeprazole (20 mg/kg) P.O | 1.72±0.23* | 5.06±0.06* | 26.96±0.18* | 330.36±0.54* |
| 4. | EECG (200mg/Kg) P.O | 2.4±0.13* | 3.81±0.06* | 46.28±0.37* | 200.65±0.26* |
| 5. | EECG (400mg/Kg) P.O | 2.0±0.21* | 4.3±0.05* | 30.94±0.12* | 290.37±0.87* |

All the values were expressed as mean ± SEM. Each group contain six animals. All the data were analysed by one way ANOVA followed by TUKEYS – teat HSD method. $P < 0.01$ Considered to be significant (*=$P < 0.01$)

**Ulcer score**

The EECG demonstrated significant activity in the ulcer score. Ulcer scoreofEECG at a 400mg/Kg was 2.0±0.2* and the standard Omeprazole showed 1.2±0.16*.

**Ulcer index**

The effects of EECG on the ulcer index on experimental animals were measured. Oral administration of 300 mg/kg of aspirin caused a significant increase in the degree of ulceration (ulcer index) in the rats. A significant decreasing in the ulcer index was observed in the EECG treated animals. Ulcer indexof EECG at a 400mg/Kg was 7.8±0.28* and the standard Omeprazole was 5.2±0.34*.

**Degree of protection or % inhibition of ulcer**

The effects of EECG on % inhibition of ulcer were measuredon the experimental animals. Oral administration of 300 mg/kg of aspirin caused a significant increase in the degree of ulceration. A significant increasing in the level of inhibition of ulcer was observed in the EECG treated animals. Maximum % inhibition of ulcerof EECG at a 400mg/Kg was60.77±0.6*% and the standard Omeprazole was 76.23±0.5*%.

**Tables 2:** Effect of EECG on Ulcer score, ulcer index and % Inhibition of ulcer

| S.No | Group | Ulcer score | Ulcer index | % inhibition of ulcer |
|---|---|---|---|---|
| 1. | Control (Normal saline-1ml) P.O | 0 | 0 | 0 |
| 2. | Ulcer control (Aspirin 300 mg/kg) P.O | 3.4±0.2* | 15.4±0.56* | 0 |
| 3. | Omeprazole (20 mg/kg) P.O | 1.2±0.16* | 5.2±0.34* | 76.23±0.5* |
| 4. | EECG (200mg/Kg) P.O | 2.8±0.2* | 10.4±0.60* | 32.46±0.7* |
| 5. | EECG (400mg/Kg) P.O | 2.0±0.2* | 7.8±0.28* | 60.77±0.6* |

All the values were expressed as mean ± SEM. Each group contain six animals. All the data were analysed by one way ANOVA followed by TUKEYS – teat HSD method. P < 0.01 Considered to be significant (*=P < 0.01)

**Histopathological studies**

Histopathologic examination of stomach of various groups of animals was performed. Figure 4-A shows normal architecture with intact mucosa, submucosa, muscularis and serosa. The presence of inflammation, congestion and epithelial damage revealing successful induction of ulcers in aspirin induced model was shown in Figure 4-B. The standard drug omeprazole was effective in healing ulcer at 20 mg/kg p.o.Figure 4-C shows effect of *C. grandis* against aspirin-induced ulcers. The lower dose of EECG 200 mg/kg exhibited presence of ulceration, inflammation, and congestion in histology of animals Figure 4-D. However, the higher dose of EECG 400 mg/kg was significantly restored stomach damages and confirms the gastro protective effect of *C. Grandis*Figure 4-E.

**Figure 4**: Effect of EECG on stomach of aspirin induced peptic ulcer rats

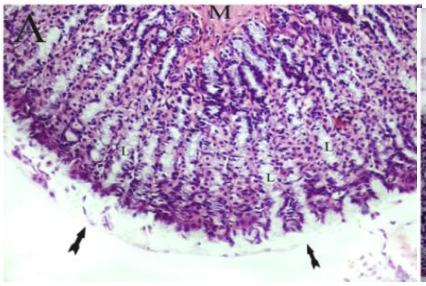



**Fig 4 – A** (Normal control)

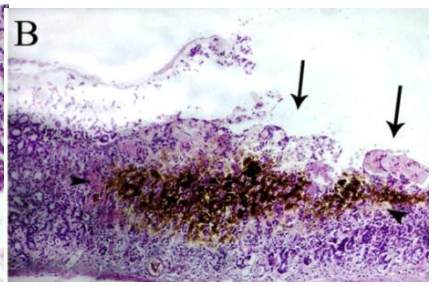



**Fig 4 – B** (Aspirin 200mg/Kg)

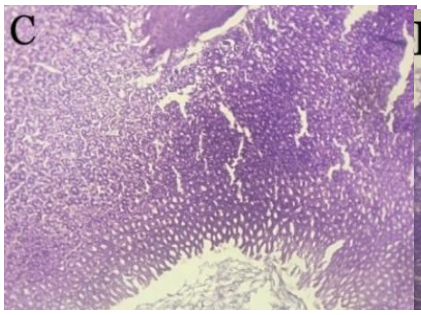



**Fig 4 – C** (Omeprazole 20mg/Kg)

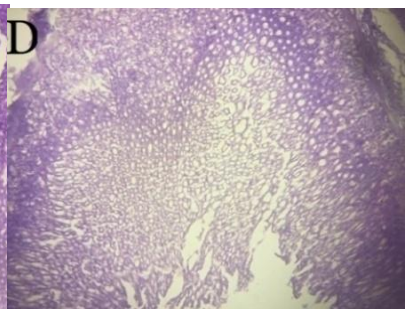



**Fig : 4 – D** (EECG 200 mg/Kg)

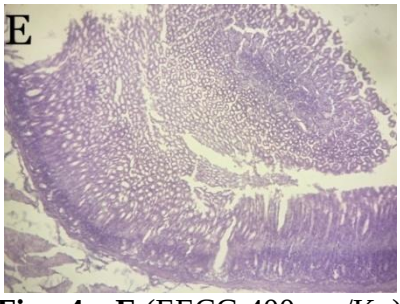



**Fig : 4 – E** (EECG 400 mg/Kg)

**DISCUSSION**

Gastric and duodenal ulcers are together referred to as peptic ulcer disease. It is one of the common gastrointestinal tract illnesses. Acid, pepsin, and *H. pylori* are examples of offensive factors, and mucin, prostaglandin, bicarbonate, nitric oxide, and growth factors are examples of defensive factors. These imbalances lead to peptic ulcers. Reducing stomach acid production and encouraging

the growth of the stomach's mucosa are two common strategies for treating peptic ulcers today. Histamine $H_2$ receptor blockers, Proton pump inhibitors, antacids, prostaglandin analogues, and anti-*H. Pylori* drugs are common medications used to treat peptic ulcers. The likelihood of tolerance, relapse, and adverse effects cast doubt on their use.

In the current work, ethanolic *Cassia grandis* Linn leaf extract was examined for potential anti-oxidant and anti-ulcer action in both *in-vitro* and *in-vivo* investigations.

One of the most commonly used analgesics in the world is aspirin. However, it has a number of undesirable side effects, including stomach mucosal damage and gastric ulcers. Aspirin inhibits prostaglandin synthesis, boosts acid secretion, and results in back diffusion of H+ ions. Mucus and bicarbonate are secreted by the stomach thanks to prostaglandins. Prostaglandins control mucosal blood flow and mucosa renewal as well. Aspirin's suppression of prostaglandin synthesis increases the vulnerability to mucosal damage, which worsens the prognosis of ulcers.

There are several gastric glands in the stomach. Gastric acid secretion is controlled by parietal cells. In parietal cells, a gastric proton pump transports protons to the stomach where they are then hydrolysed in the cytoplasm. One of the main reasons of acidity and ulcers is the over secretion of this enzyme. Thus, inhibiting the stomach proton pump is a simple way to manage problems associated with acidity and lower gastric acid output, making it a unique target for the treatment of peptic ulcers. Acid peptic disease should be treated with proton pump inhibitors. However, its use is restricted due to side effects and safety concerns.

The DPPH scavenging activity and FRAP assay of EECG was investigated it showedpromising anti-oxidant property. The in vitro Acid neutralizing capacity (ANC) and H + -K + ATPase inhibitory activity of EECG was investigated in this work. The study's findings showed that the EECG has goodacid neutralizing effect and H + -K + ATPase was inhibited in a dose-dependent manner.

In the current investigation, administration of an EECG to aspirin-treated rats model caused a decrease in ulcers, as shown by a decrease in ulcer index. Additionally, ethanol leaf extract reduced the overall acidity of the stomach's contents, minimized gastrointestinal damage, and increased the amount of gastric mucus. Similar types of antiulcer reports were observed with other Cassia species also. It was reported that the ethanol leaf extract of *Cassia fistula* showedantiulcer activity [24].So this study further confirms the antioxidant & antiulcer activityof *Cassia grandis.*

## CONCLUSIONS

From the result of the present study, it was concluded that, that the ethanolic leaf extract of *Cassia grandis* Linn shown promising anti-oxidant and antiulcer activity against aspirin induced peptic ulcer on swiss albino wistar rat. The antiulcer activity along with its safety profile could make the leaves of *Cassia grandis* Linn a good candidate for the treatment of PUD in humans.

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