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ORIGINAL ARTICLE

ANTIULCER ACTIVITY OF *CAYRATIA PEDATA, ENICOSTEMMA AXILLARE* AND *TERMINALIA CHEBULA* ON ETHANOL INDUCED ALBINO WISTAR RATS

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ABSTRACT

The aim of this study is to evaluate the anti-ulcer activity of ethanolic plant extract of *Cayratia pedata, Enicostemma axillare* and seed of *Terminalia chebula*. The ethanolic extract of *Cayratia pedata Enicostemma axillare* and seeds of *Terminalia chebula* was investigated for anti-ulcer activity against ethanol induced gastric ulcers in rats. Omeprazole (20 mg/kg) was used as standard drugs. A significant ($p<0.05$) anti-ulcer activity was observed in rat models. The plant extract of *Cayratia pedata, Enicostemma axillare and Terminalia chebula* produced gastroprotective effect in ethanol induced rats showed the decreased level of ulcer index, increased level of percentage of protection and also gastric volume, pH, free acidity, total acidity, mucin content, pepsin content, total protein were calculated from the effective concentration (200 mg/kg). This present study indicates that, *Cayratia pedata, Enicostemma axillare* and *Terminalia chebula* leaf extract have potential anti-ulcer activity in this tested model.

Keywords: *Cayratia pedata, Enicostemma axillare* and *Terminalia chebula,* Ethanol, Omeprazole.

1.INTRODUCTION

Ulcers are an open sore of the skin or mucus fluid layer portrayed by sloughing of aggravated dead tissue. There are numerous sorts of ulcer, for example, mouth ulcer, throat ulcer, peptic ulcer, and genital ulcer. Of these peptic ulcer is seen among numerous individuals. Gastric ulcers are situated in the stomach, portrayed by agony; ulcers are basic in more seasoned age bunch. Eating may expand torment as opposed to assuage torment. Different manifestations may incorporate sickness, spewing, and weight reduction. Despite the fact that patients with gastric ulcers have typical or reduced corrosive creation, yet ulcers may happen even in complete absence of corrosive (Vyawahare *et al.,* 2009). Duodenal ulcers are found toward the start of small intestine tract and are portrayed by serious torment with copying sensation in upper belly that stirs patients from rest. By and large, torment happens when the stomach is vacant and diminishes in the wake of eating. Duodenal ulcer is more basic in more youthful people and prevalently influences guys. In the duodenum, ulcers may show up on both the foremost and back dividers (Brooks, 1985). Now, peptic ulcer can be life debilitating with side effects like bleeding stool, serious stomach torment and issues alongside vomiting blood

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The Peptic ulcer is a perpetual, non-harmful incendiary infection portrayed by ulceration in the upper gastro-intestinal tract (stomach and duodenum) where parietal cells area found, which are usually acidic and thus, extremely painful. Several factors - such as improper digestion, metabolism, elimination of food, mental and physical stress enhance the growth of peptic ulcers. A number of drugs are available for the treatment of peptic ulcers, but the medical evaluation of these drugs indicates high incidences of side effects and drug interactions. The pathophysiology of peptic ulcer disease involves an imbalance between offensive and defensive factors. Approximate 15,000 deaths occur with peptic ulcer disease. The pathophysiology of peptic ulcer disease involves an imbalance between offensive (acid, pepsin, and Helicobacter pylori) and defensive factors (mucin, prostaglandin, bicarbonate, nitric oxide, and growth factors) (Hoogerwerfand, 2001.) Today, most of the world population depends upon plant based drugs for their primary health care needs. (Ahmed *et al.,* 2008) World Health Organization (WHO) estimates that 80% of the people living in developing countries almost exclusively use traditional medicines plant.

A new drug provides from plants sources, which have historical background. Although modern medicines may be available, due to socio-economical, cultural and historical reasons, these drugs have maintained their importance.

(Sartori *et al*.,1999). A number of drugs are used widely for the treatment of ulcer (Soll, 1990). The drugs show their affect mostly by inhibiting the Cox enzyme, protective mechanism, neutralizing mechanism and so on. Inspite of its curative affects they show some side effects (Chan, 2000). Indian Medicinal plants and their derivatives have been an invaluable source of therapeutic agents to treat various disorders including peptic ulcer.

Cayratia pedata Lam. is a climber belonging to the Family Vitaceae and it grows in shrubberies of India, Andaman Islands, Ceylon and Malasiya. Traditionally whole plant is used in the treatment of Anti-diarrhoeal and refrigerant, useful in burn and hysteria (Joy *et al*., 1998). In folklore claim, it is used for treating ulcer, diarrhoea, burns, refrigerants, uterine, flukes, a stringents low diuretic activity and has been a reputed remedy for cough, bronchitis, asthma, joint pain and to check uterine reflexes. The decoction of the leaves was used to check uterine and other fluxes (Patil, 2000). The plant has also found to possesses anti-inflammatory and antinociceptive activities (Veeradass Rajendran, 2011).

Enicostmma axillare (Lam.) Raynal. Syn.*E. littorale* Blume (Family - Gentianaceae), locally called as *Chota chirayita* in Marathi and *Mamajaka* in Sankrit, has been used traditionally for many diseases. Ayurvedic literature survey, the fresh juice of leaves has been used as a bitter tonic, to control arthritis, to reduce typhoid fever and as cooling agent. It is also used as stomachic and laxative, blood purifier in dropsy, rheumatism, abdominal ulcers, hernia, swellings, itches and insect poisoning. Plant extracts were reported for the biological activities such as antidiabetic, anti – inflammatory, stimulant, astringent and diuretic and useful in skin disease. The plant possesses stimulant, astringent, diuretic anthelmintic properties (The wealth of India, 2002, Raghu Bir, 2006). It is also acts as ethno medicine for snake bite (Garg, 2000).

Terminalia chebula Retz. belongs to the family "Combretaceae", commonly known as black myrobalan. *T. chebula* is a medium- to large-sized tree distributed throughout tropical and sub-tropical Asia, including China and Tibet. This tree is found in the forests of northern India, Uttar Pradesh and Bengal, and is common in Tamil Nadu, Karnataka and southern Maharastra. The traditional Indian systems of Ayurveda and Siddha medicines support the importance of medicinal plants to treat diseases (Beusher *et al*., 1994). *T. chebula* is routinely used as traditional medicine by tribals of Tamil Nadu to cure several ailments such as fever, cough, diarrhea, gastroenteritis, skin diseases, candidiasis, urinary tract infection and wound infections (Dash and Bhagwan, 1991). Plant fruits appear to have evolved complex antibiotic compounds to cure various diseases like cancer, cardiovascular, digestive and pathogenic bacteria. Antibacterial activity of *T. chebula* extracts against several bacterial strains have been reported (Malckzadeh *et al*., 2001; Kim *et al*., 2006; Chattopadhyay *et al*., 2007; Bag *et al*., 2009). It is effective in inhibiting *Helicobactor pylori* (Malckzadeh *et al*., 2001), *Xanthomonas campestris*pv.*citri* (Afzalakhtar *et al*., 1997) and *Salmonella typhi* (Rani and Khullar, 2004). In the present study it was aimed to investigate of ethanolic extract in poly herbal formulation of ethanol induced ulcers rats.

2.MATERIALS AND METHODS

Animals

Male Wistar albino rats (150-200 g) were procured from Rajah Muthiah Medical College and Hospital, Annamalai University, Chidambaram, Tamilnadu, India and were housed in polycarbonate cages in an animal room for 12 hours day – night cycle. The animals were allowed free access to tap water and standard laboratory rat food. The animal treatment and protocol employed were approved by the Institutional Animal Ethics Committee, Annamalai University (Registration Number - 1102 / 2015 /CPCSEA).

Collection of plant materials

The leaves of *Cayratia pedata, Enicostemma axillare and seed of Terminalia chebula* were collected from kolli hills, Nammakal District. South India. Plant leaves were dried under the shadow. The dried leaves were fine powdered and stored in polythene bags at room temperature (30° C) It was authenticated by Dr. V. Chelladurai, Research officer-Botany, Central council for Reasearch in Ayurveda & Siddha, Govt of India. The voucher specimen was kept in our research laboratory for further reference.

Preparation of extracts

The leaves of *Cayratia pedata , Enicostemma stemma axillare and* seed of *Terminalia chebula* were cleaned and washed in running water and dried at room temperature for two weeks and then coarsely powdered it with the help of Hand mill. The fine powder was extracted successively in soxhlet apparatus the boiling point of ethanol (1500 ml) was set up at 78° C. The solvent was recycled, thereby extracting the compounds present in the sample. They were continuously extracted until the solvent loses its color. All the extracts were carefully evaporated in a rotary evaporator under Controlled temperature and reduced Pressure to get the extracts was stored in the refrigerator.

Ulcer index and percentage of protection

Experimental design

The animals were randomly divided into nine groups of six animals each. The plant extract of *Cayaratia pedata, Enicostemma axillare* , seed extract of *Terminalia chebula* and Omeprazole were administrated orally by intubation once in a day in the morning hours for 6 days.

Group I -	Normal control
Group II -	Ulcer induced rats (ethanol)
Group III-	Omeprazole (20 mg /kg b.w).
Group IV-	*Cayaratia pedata* (200 mg/kg b.w)
Group V -	*Cayaratia pedata* (400 mg/kg b.w)
Group VI-	*Enicostemma axillare* (200 mg/kg b.w)
Group VII-	*Enicostemma axillare* (400 mg/kg b.w)
Group VIII-	*Terminalia chebula* (200 mg/ kg b.w)
Group XI-	*Terminalia chebula* (400 mg/ kg b.w)

Group-I (normal control) was treated with normal saline. Group-II animals were treated with ethanol orally (1 ml). Group-IV to IX rats were pretreated with *Cayratia pedata, Enicostemma axillare* and seed *Terminalia chebula* (200 and

400 mg/kg b.w,), respectively for 1 week. The animals of Group III were subjected to Omeprazole (20 mg /kg b.w) pretreatment for 1 week. The animals of all Groups were first fasted in individual cages for 36 h and were subjected to ethanol administration (1ml/ kg b.w.) in order to induce ulcers Animals were sacrificed 60 min later. The stomach was excised and then cut along the curvature and washed with 0.9% NaCl (5.0 ml). The ulcers were scored in the glandular portion of the stomach. The ulcer index was calculated by adding the total number of ulcer per stomach. (Hollander *et al*., 1985).

Measurement of ulcer index

Each stomach was opened along the greater curvature. Samples of gastric contents were analyzed for hydrogen ion concentration by pH-meter titration with 0.1 N NaOH solutions using digital pH meter. The acid content was expressed as mEq/l. Gastric mucus production was measured in the rats that were subjected to absolute ethanol-induced gastric mucosal injury. The gastric mucosa of each rat was gently scraped using a glass slide and the mucus obtained was weighed using a precision electronic balance (Tan, *et al*., 2002).

Any ulcers would be found in the gastric mucosa, appearing as hemorrhagic lesions of the stomach. Each gastric mucosa was examined for damage. The length (mm) and width (mm) of the ulcer on the gastric mucosa were measured by a planimeter (10 × 10 mm^2 = ulcer area) under dissecting microscope (1.8 x). The area of each ulcer lesion was measured by counting the number of small squares, 2 × 2 mm, covering the length and width of each ulcer band. The sum of the areas of all lesions for each stomach was applied in the calculation of the ulcer area (UA) where the sum of small squares × 4 × 1.8 = UA mm^2 as described by (Kauffman and Grossman 1978) with slight modification. The inhibition percentage (I %) was calculated by the following formula as described by (Njar *et al*., 1995).

(I %) = [(UA control – UA treated) ÷ UA control] × 100.

Anti-ulcer activity studies

Experimental design

Based on the results of Ulcer index and % protection, the animals were regrouped, and only the effective concentration of 200mg/kg b.w of the plant extract of *Cayaratia pedata, Enicostemma axillare,* and seed extract of *Terminalia chebula* and Omeprazole were retained for further study. For experimental purposes, the groups were reorganized as follows:

Group I - Normal control
Group II - Ulcer induced rats (ethanol)
Group III- Omeprazole (20 mg /kg b.w).
Group IV- *Cayaratia pedata* (200 mg/kg b.w)
Group V - *Enicostemma axillare* (200 mg/kg b.w)
Group VI- *Terminalia chebula* (200 mg/ kg b.w)

Ethanol induced ulcer model

Thus, Group-I (normal control) was treated with normal saline. Group-II animals were treated with ethanol orally. Group-IV,-V and-VI rats were pretreated with *Cayratia pedata, Enicostemma axillare* and seed *Terminalia chebula* (200 mg/kg b.w,), respectively for 1 week. The animals of Group III were subjected to Omeprazole (20 mg /kg b.w) pretreatment for 1 week. The animals of all Groups were first fasted in individual cages for 36 h and were subjected to ethanol administration (1ml/ kg b.w.) in order to induce ulcers Animals were sacrificed 60 min later. The stomach was excised, the gastric content was collected and centrifuge for 5 min at 2000 x g and the supernatant was separated. The gastric volume, pH, free acidity, total acidity, mucin content, pepsin content and total protein of gastric fluid were determined for the effective concentration only. The samples were further processed for antioxidants parameters and histopathological estimation (Hollander *et al*., 1985).

BIOCHEMICAL ESTIMATIONS

Determination of gastric volume

The rats were sacrificed, the stomach portion was removed and The gastric contents were transferred into the centrifuge tube. The gastric juice was centrifuged at 1000 RPM for 10 minutes. The supernatant liquid was then transferred to a measuring cylinder, and the volume was measured.

Determination of pH of gastric content

1 ml of the gastric juice was collected, and pH was directly measured by using pH strip for pH (Arun and Asha 2008).

Determination of free acidity and total acidity

The total volume of gastric content was measured after gastric contents were centrifuged and filtered. One ml of the gastric juice was pipetted out and the solution was titrated against 0.1N sodium hydroxide and using 2 to 3 drops of Topfer's reagent as indicator, to the endpoint when the solution turned to yellowish orange colour was observed. This indicated the volume of NaOH required neutralizing the free hydrochloric acid present in the gastric juice. Then 2 to 3 drops of phenolphthalein solution was added and titration have continued until a definite red tinge reappears. The difference between the two readings indicated the volume of NaOH required neutralizing the combined acid present in the gastric juice. The sum of the two titrations was the total acid present in the gastric juice (Muhammad jan *et al*.,2004)

Acidity was calculated by using formula;

$$\text{Acidity} = \frac{\text{Vol. of NaOH} \times \text{Normality of NaOH}}{\text{Vol. of Gastric juice used}} \text{ m. Eq. /dl.}$$

Determination of mucin content

The barrier mucus of gastric tissue was estimated by the method of (Corne *et al*., 1974). The dissected stomach was soaked for 2h in 0.1 % alcian blue. Dye complexed with mucus was diluted by immersion in 10 ml aliquots of 0.5 M $Mgcl_2$ for 2h. The resulting blue solutions were shaken with equal volumes of diethyl ether and the density of the aqueous phase was measured at 605 nm. The barrier mucus was expressed of µg of Alcian blue/g of glandular tissue.

Determination of pepsin content

Pepsin was assayed according to the method of (Shay *et al*., 1945) using hemoglobin as substrate. The absorbance of the

solution was read at 650 nm. The pepsin content was expressed as µm of tyrosin liberated/ml.

Estimation of total proteins

Reagents

Alkaline copper reagent

Solution A: 2% sodium carbonate in 0.1N sodium hydroxide

Solution B: 0.5% copper sulphate in 1% sodium potassium tartarate. 50 ml of solution A was mixed with 1 ml of solution B just before use. Folins phenol reagent. One volume of folins reagent was diluted with two volumes of distilled water, just before use.

Standard bovine serum albumin

20 mg of bovine serum albumin was dissolved in 100 ml of distilled water and Few drops of NaOH for complete dissolution of bovine serum albumin and to avoid frothing, it was allowed to stand overnight in a refrigerator.

Procedure

The dissolved proteins in gastric juice were estimated by adding 90 % of alcohol with gastric juice in 9:1 ratio respectively. Take 0.1 ml of alcoholic precipitate of gastric juice was dissolved in 1 ml of 0.1 N NaOH and from this 0.05 ml was taken into another test tube and add 4 ml of alkaline copper reagent kept for 10 minutes. Then 0.5 ml of phenol reagent was added and waits for 10 minutes to color development. Reading was taken against distilled water blank at 640 nm. The total protein content was calculated against a standard curve prepared with bovine albumin and has been expressed in terms of µg/ ml of gastric juice (Lowery, 1951; Rosenbrough *et al*., 1993).

3.RESULT

Group I- Normal control showed the 100 % of protection. Group II-Ulcer induced rats (ethanol) showed the increased ulcer index and the percentage of protection is zero. Group III- Omeprazole (20 mg /kg b.w) was used to standard drugs, it shows decreased level of ulcer index and percentage of protection was high when compared to ethanol treated group. Group-IV to IX rats were pretreated with *Cayratia pedata*, *Enicostemma axillare* and seed *Terminalia chebula* (200 and 400 mg/kg b.w) showed the decreased level ulcer index and increased level of protection when compared to ethanol treated group (Table 1).

Table 1. Ulcer index and % of protection of ethanolic plant extract of *Cayratia pedata*, *Enicostemma axillare* and *Terminalia chebula* on ethanol induced ulcer in rats

Groups	Dose	Ulcer index (mm^2)	% of Protection
Group I		0.00^{a}	100.02 ± 7.62^{c}
Group II	Ethanol (1 ml)	30.58 ± 2.34^{d}	0.00 ± 0.00^{a}
Group III	Omeprazole (20 mg)	6.62 ± 0.50^{bc}	78.34 ± 5.97^{b}
Group IV	*Cayratia pedata* (200mg)	5.62 ± 0.43^{b}	81.64 ± 6.25^{b}
Group V	*Cayratia pedata* (400mg)	5.58 ± 0.43^{b}	81.21 ± 6.22^{b}
Group VI	*Enicostemma axillare* (200 mg)	6.98 ± 0.53^{c}	77.19 ± 5.91^{b}
Group VII	*Enicostemma axillare* (400 mg)	6.79 ± 0.52^{c}	77.19 ± 77.79^{b}
Group VIII	*Terminalia chebula* (200 mg)	7.68 ± 0.59^{c}	74.90 ± 5.73^{b}
Group 1X	*Terminalia chebula* (400 mg)	7.36 ± 0.56^{c}	75.94 ± 5.81^{b}

Values are expressed as Mean ± SD for six animals in each group. Values not sharing a common superscript (a, b, c..) are significantly different from each other ($p < 0.05$).

gastric volume, pH, free acidity, total acidity and total protein presented were calculated from the effective concentration 200m/kg bw and/or standard drug Omeprazole treated groups, and also for ethanol alone treated group (Table 2).

The level of gastric volume was increased in the Group- I (ethanol treated) when compared to standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm axillare* and *Terrminalia chebula*

The level of pH was decreased in the Group-II (ethanol treated) when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm* axillare and *Terrminalia chebula*

The effect of free acidity was increased in the Group-II (ethanol treated) when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm* axillare and *Terrminalia chebula*

Table 2. Gastric volume, pH, Free acidity, Total acidity, Mucin content, Pepsin content and Total Protein in stomach of ethanolic plant extract of *Cayratia pedata*, *Enicostemma axillare* and *Terminalia chebula* treatment on ethanol induced ulcer in rats

Tretment	Gastric volume (ml/100mg)	pH	Free acidity (mEq/L)	Total acidity (mEq/L)	Mucin content (µg/g)	Pepsin content (µmol/h)	Total Protien (mg/ml)
Group I Ethanol	3.27 ± 0.25^{c}	1.26 ± 0.10^{a}	36.88 ± 2.82^{d}	46.30 ± 3.54^{c}	1.78 ± 0.14^{a}	0.86 ± 0.07^{d}	11.27 ± 0.86^{a}
Group II Omeprazole	1.38 ± 0.11^{a}	2.26 ± 0.17^{cd}	13.26 ± 1.01^{ab}	26.30 ± 1.92^{ab}	4.39 ± 0.33^{c}	0.28 ± 0.02^{ab}	26.06 ± 1.98^{c}
Group III *Cayratia pedat* (200 mg)	1.20 ± 0.09^{a}	2.48 ± 0.19^{d}	11.24 ± 0.86^{a}	25.02 ± 1.92^{a}	4.86 ± 0.37^{d}	0.24 ± 0.02^{a}	29.64 ± 2.27^{d}
Group IV *Enicostemma axillare* (200 mg)	1.41 ± 0.11^{a}	2.10 ± 0.16^{c}	14.59 ± 1.12^{bc}	28.03 ± 2.15^{ab}	4.01 ± 0.31^{c}	0.31 ± 0.02^{bc}	25.63 ± 1.96^{bc}
Group V *Terminalia chebula* (200 mg)	1.78 ± 0.14^{b}	1.86 ± 0.14^{b}	16.21 ± 1.24^{c}	3.27 ± 0.25^{b}	3.59 ± 0.27^{b}	0.36 ± 0.03^{b}	23.04 ± 1.70^{b}

Values are expressed as Mean ± SD for six animals in each group. Values not sharing a common superscript (a, b, c..) are significantly different from each other ($p < 0.05$)

The level of total acidity was increased in the Group-II (ethanol treated) when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm* axillare and *Terrminalia chebula*

The level of mucin content was decreased in the Group-II (ethanol treated) when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm* axillare and *Terrminalia chebula*

The level of pepsin content was increased in the Group-II (ethanol treated) when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm* axillare and *Terrminalia chebula*.

The level of protein content was increased in the ethanol treated rats when compared to the standard drug omeprazole and plant extracts of *Cayratia pedata, Enicostemm axillare* and seed of *Terminalia chebula*

4.DISCUSSION

In the present study, *Cayratia pedata, Enicostemm axillare* and seed of *Terminalia chebula* antiulcer activity was evaluated by employing ethanol induced gastric ulcer models in albino rats. *Cayratia pedata, Enicostemm axillare* and seed of *Terminalia chebula* pre-treatment showed significant antiulcer activity against gastric ulcers in increasing order of doses. Ethanol prompted gastric lesion formation may be because of stasis in gastric blood stream which adds to the advancement of the discharge and necrotic parts of tissue injury. Alcohol rapidly penetrates the gastric mucosa clearly creating cell and plasma film harm prompting expanded intra cell layer permeability to sodium and water. The gigantic intracellular accumulation of calcium speaks to a noteworthy stride in the pathogenesis of gastric mucosal harm. Liquor instantly infiltrates the gastric mucosa obviously bringing about cell and plasma layer harm driving to increased intra cell film penetrability to sodium and water. The huge intracellular collection of calcium speaks to which leads to cell death and exfoliation. There is much evidence that the ethanol damage to the gastrointestinal mucosa starts with micro-vascular injury, namely disruption of the vascular endothelium resulting in increased vascular permeability, oedema formation and epithelial lifting. These effects are secondary to ethanol induced slowing or cessation of gastric mucosal flow. Ethanol also induces a marked compression of the roundabout muscles of rodent fundic strip. Such a withdrawal may prompt mucosal pressure at the site of the best mechanical anxiety, at the peaks of mucosal folds prompting corruption and ulceration.

The etiology of peptic ulcer is obscure in a large portion of the cases, yet it is by and large acknowledged that it results from unevenness between forceful components and the support of mucosal integrity through the endogenous resistance systems. To recover the parity, diverse helpful operators including plant concentrates may be utilized (Raju, 2009).

A. indicum concentrate is one such home grown medication utilized as a part of the present concentrate principally to assess the counter ulcerogenic in pylorus ligation and ethanol impelled ulcers in rats. This prompts cell demise and shedding in the surface epithelium. It was seen in this study that the concentrate diminished altogether ethanol-actuated ulcer. This may be because of cytoprotective impact of the concentrate by means of cancer prevention agent impacts. The concentrate indicates insurance against trademark injuries delivered by ethanol organization this antiulcer impact of MEAI may be because of both diminishments in gastric corrosive emission and gastric cytoprotection. The antiulcer property of *A. indicum* in pylorus ligation model is clear from its huge decrease in free acidity, all out sharpness, number of ulcers and ulcer record. *A. indicum* treated creatures essentially repressed the arrangement of ulcers in the pylorus ligated rats furthermore diminished both the fixation and expanded the pH it is recommended that *A. indicum* can smother gastric harm impelled by forceful elements.

The system of activity of omeprazole is such that it ties particularly to a solitary subunit of the H +, K +-ATPase at the secretory surface of parietal cell and inactivate it (Munson *et al*., 1995). It reduces acid secretion regardless of the source of secretory stimulation. By expanding intragastric pH through hindrance of corrosive discharge, PPIs hinder initiation of pepsin. They are successful in treating peptic ulcer malady and gastroesophageal reflux with both short and long haul use (Schneeweiss *et al*., 2006). Ethanol upsets the gastric mucosal obstruction and reason significant smaller scale vascular changes with solid vaso-tightening joined by arteriolar dilatation in charge of engorgement of mucosal capillaries (Cho and Ogle, 1992).

As it has been mentioned in (Wasman, *et al*., 2010) by Goel and Sairam, peptic ulcer is a popular gastrointestinal medical problem despite its vague etiology but the imbalance between aggressive factors like acid and pepsin and defensive factors which lead to maintenance the unity of mucus has been admitted as the main cause of peptic ulcer. Stated by Sezabo, Marhuenda and Mutoh in (Mahmood, *et al*., 2010) ulcer induction by ethanol in experimental rats causes severe injuries in stomach which will begin with micro vascular damages and leads to enhancing in vascular permeability. Direct toxic effect of ethanol prompts necrotic damages to gastric mucosa by decreasing mucus production and diminishing effect on bicarbonates discharge.

The results from the present study suggest that the various extract of *Cayratia pedata* Lam, *Enicostemma axillare* and seed of *Terminalia chebula* exhibited significant Anti-ulcer effect. The most broadly utilized essential test to screen anti-inflammatory agent is to measure the ability of a compound to reduce local oedema actuated in rodent paw taking after the infusion of aggravations, for example, carrageenan (Winter, 1962).The chloroform extract of *Cayratia pedata* increased absorption of water and electrolyte from the gastrointestinal tract. Consumption of excessive alcohol usually elevates the risk of gastric mucosal damage by generating oxygen-derived free radicals such as superoxide anions, hydroxyl radicals and lipid peroxides (Li *et al*., 2008 and Pan *et al*., 2008).

In the present study that the ethanol leaf extract of *Cayratia pedata, Enicostemm axillare* and seed of *Terminaliachebula* has an ulcer healing property against experimentally induced ulcers in rats. Hence this study confirms benefits in treatment of ulcer.

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