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EVALUATION OF THE ANTI-ULCER ACTIVITY OF ETHANOLIC LEAVES EXTRACT OF *EUTERPE OLERACEA*

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ABSTRACT

Peptic ulcers are a prevalent and widespread health issue. Primary reasons include an elevation in harmful substances affecting the mucosal lining, a decrease in the body's ability to defend the mucosal lining, or the generation of free radicals. Various plants and their derivatives, including Allium sativum, Bacopa monnieri, Moringa oleifera, Aloe vera, Aegle marmelos, Carica papaya, Terminalia chebula, Sesbania grandiflora, Shorea robusta, Azadirachta indica, Ocimum sanctum, Annona squamosa, Mimosa pudica, and several others, have demonstrated the ability to prevent or treat peptic ulcers. The Euterpe oleracea plant was selected for assessment of its anti-ulcer properties because it contains choline esters, which have the ability to regulate mucus production. The plant is recognised for its many phytoconstituents, including as flavonoids, terpenoids, steroids, and coumarin. At first, plant leaves were gathered and verified for authenticity. The Soxhlet device was used to do the extraction. The pre-phytochemical screening indicated the presence of several phytoconstituents, such as flavonoids, terpenoids, cardiac glycosides, steroids, and coumarin. The anti-ulcer effects of Euterpe oleracea leaves were investigated using a model where ulcers were generated by aspirin. Pretreatment with the ethanolic extract of Euterpe oleracea substantially lowered the ulcer index, free and total acidity, and glutathione GSH serum levels ($p< 0.001$). In addition, the levels of amylase, MDA, and amylase serum showed an increase. This was further confirmed by histological examinations. In conclusion, our experiment showed that the ethanolic extract of Euterpe oleracea has a similar ability to cure ulcers as the test substance. Therefore, this plant has the capacity to be used for the purpose of identifying natural substances that may possibly serve as a reservoir of ideas for new pharmaceutical goods.

KEYWORDS: Peptic ulcers, Helicobacter pylori, *Euterpe oleracea*, Antacids.

1.1 INTRODUCTION

Ulcers are a common gastrointestinal disorder with several origins; they impact 10% of the global population. Persistent alcohol use, smoking, extreme stress, long-standing use of non-steroidal anti-inflammatory medications, and infection with H.pylori bacteria are the most general causes of peptic ulcers, which may cause inflammation, mucosal bleeding, and stomach pain in patients Ulcers like this may form when the stomach's protective mucus, bicarbonate, and prostaglandins are out of sync with the harmful chemicals like acid, pepsin, bile ions, and Helicobacter pylori bacteria. The most recent method for treating peptic ulcers involves preventing acid production, encouraging gastro-protection, reducing cell death, and increasing the number of epithelial cells to speed up the healing process. Histamine receptor antagonists, prostaglandin analogues, proton pump inhibitors, cytoprotective medicines, antacids, and anticholinergics are among the common medications used to treat ulcers. However, it is important to note that these treatments might mix with other pharmaceuticals or have unpleasant side effects. Use of these medications completed a prolonged period of time have the probable to alter biochemical reactions in the body. Consequently, herbal medications are often used for the long-term treatment of chronic conditions.

The muscularis mucosae lining the gastrointestinal tract may become infected with erosions or sores called peptic ulcers (PU). Cell death in its various phases, localised white blood cell infiltration, reduced blood flow, elevated amounts of oxygen molecules with detrimental effects, and inflammation are common symptoms of these disorders.[1] Symptoms of a non-life-threatening illness known as a peptic ulcer (PU) often include recurrent pain in the upper abdomen. Patients may experience significant worry, disruption of daily tasks, and mental anguish due to these symptoms, but they may sometimes be alleviated by eating or taking bicarbonate.

Stomach sores, which form along the abdomen's slight curvature, and duodenal ulcers, which procedure in the intestinal tuber—the area most showing to gastric acid—are the main ways the disorder is classified anatomically.[3] Peptic ulcer disease is characterised by an unevenness between the stomach lining's defensive components—such as prostaglandins, mucus, the bicarbonate barrier, and enough blood flow—and damaging chemicals, such pepsin and HCl, according to recent research.[4] At first, it was thought that all ulcers in the oesophagus and gastric tract were produced through the antagonistic attitude of pepsin and stomach acid towards the mucosa. On the other hand, there has been recent evidence connecting the term "peptic ulcer" to Helicobacter pylori infection, a condition that becomes worse with the ongoing use of NSAIDs and acetylsalicylic acid (ASA).[5]

In this report, we will take a close look at peptic ulcers, their symptoms, clinical features, epidemiology (particularly as it pertains to H. pylori infection), the pharmacological agents that have proven effective in managing these ulcers, and last but not least, we will discuss the current opportunities and challenges surrounding the use of phytochemicals from plants as antiulcerogenic agents, building on recent advances in this area. Protecting the integrity of plant-based goods was a top priority. The goal was to guarantee a high degree of competency in managing health-related systems and to encourage people to improve their skills in this area. A complex network of organs housed in tubes makes up the human digestive system. When it comes to metabolism, the stomach is the most important organ.[6]

2.1 MATERIAL AND METHOD

2.4 Extraction Preparation

The leaves of Euterpe oleracea were washed, dried at ambient temperature, and then ground into a fine powder using a grinder. The plant powder was measured and 100g was put in a container. The extraction process was carried out three times during a span of two days, at a temperature ranging from 45 to 60℃. A volume of 750 ml of methanol was used as the solvent for extraction. The extract was condensed using a water bath after the solvent was fully evaporated using arotatory evaporator right after the extraction process. The whole dehydrated extract was then quantified, conserved, and readied for future study.

2.5. Histopathological examination

The animals were classified into five distinct groups, with each group consisting of five rodents. The prescribed order of therapy for all animals is as follows.

Group I- Normal control treated group: give the normal saline to all animal for 1 week.

Group II- give ranitidine (20 mg/kg) followed 30 minutes after by asprin (200 mg/kg) according to body weight of animal for 1 week.

Group III- ulcer induce group: give aspirin (200 mg/kg) according to body weight of animal for 1 week.

Group IV- 200 mg/kg Euterpe oleracea leaves extract treated group: Received 200 mg/kg Euterpe oleracea ethanolic leaves extract after 30 minutes administration of aspirin (200 mg/kg) within 1 week

Group V: 400 mg/kg Euterpe oleracea leaves extract treated group: Received 400 mg/kg Euterpe oleracea ethanolic leaves extract after 30 minutes administration of aspirin (200 mg/kg) within 1 week

The animals were euthanized via cervical dislocation on the seventh day, and their stomachs were removed for further study of the severity of the ulcer.

2.6. Normal control (Normal saline)

This region of the stomach did not exhibit any edoema, necrosis, haemorrhage, or necrosis.

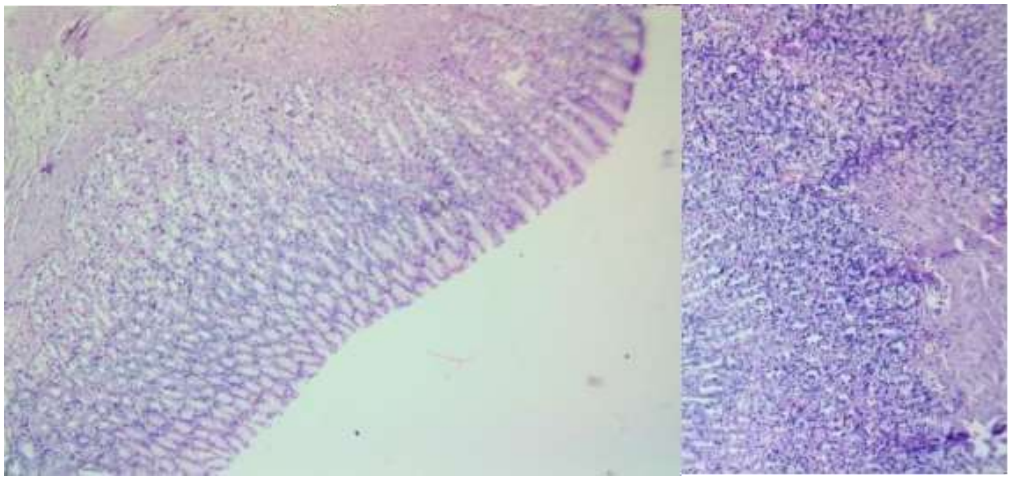

Figure 1.1.: Histopathological examination of control group with normal saline.

2.7. Microscopically investigation

Figures 3.15 depict the lining of the stomach and the area where the oesophagus meets the stomach, known as the gastroesophageal junction. The stomach foveolar and glandular layer seem normal and do not show any notable abnormalities. No significant inflammatory cells

are seen in the lamina propria. The muscular layer is essentially ordinary. The slim specimens exhibited no signs of necrosis, edoema, or desquamation, making them inconspicuous. However, the lamina propria of the mucosa showed a small number of clogged capillaries. Lymphocytes and eosinophils demonstrate localised infiltration of the sub-mucosa and muscle layer. Lymphocytes and eosinophils show localised infiltration of the muscular layer. The muscular layer does not show any presence of inflammatory infiltrates.

2.8. Ulcer induce group: Treated with Aspirin

In this part of the stomach, the whole width of the mucosa was clearly visible, along with significant surface erosion. In addition, there were regions of tissue death, swelling, and areas of bleeding.

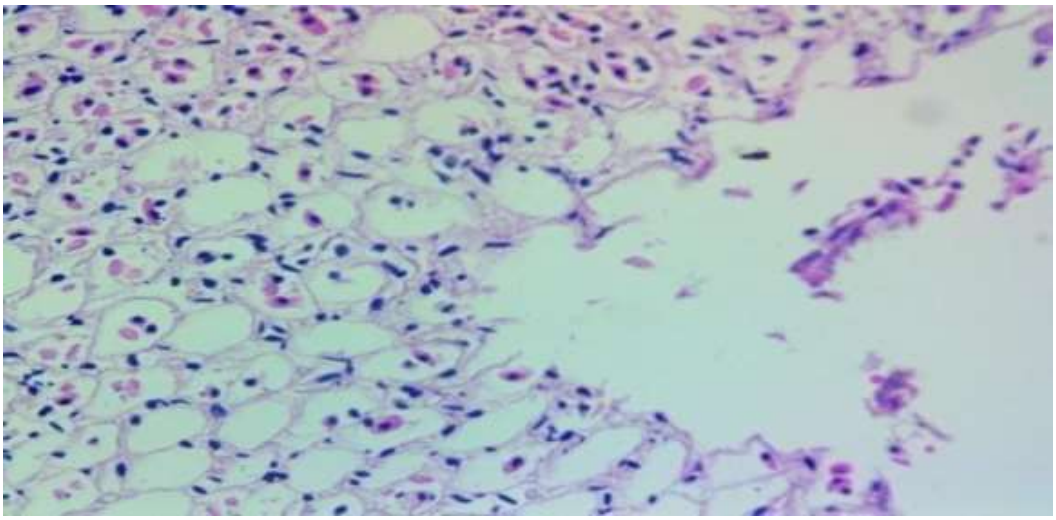

Figure 1.2: Histopathological examination of Ulcer induce group Treated with Aspirin.

2.9. Microscopically investigation

Figures 3.16 illustrate the lining of the stomach and the point where the stomach meets the oesophagus, known as the gastroesophageal junction. The foveolar and superficial glandular layers of the stomach's mucosa show a significant level of necrosis, ranging from partial to total, along with a few spots of bleeding. A few sites were discovered where the region of total necrosis extended deeply into a glandular layer. There were areas of tissue loss and accumulations of cells involved in inflammation on the surface of the mucosa. However, the muscular layers, serosa, and pariserosal tissue showed no significant abnormalities. Edoema, necrosis, and surface erosion (desquamation) are present in the superficial layers of the mucosa. Necrosis was reported to penetrate the glandular layer in a few specific areas. Furthermore, there was a little region of bleeding.

2.10. Standard group treated with Ranitidine

The mucosa and gastro-esophageal system of this part of the stomach showed an inflammatory infiltration consisting of eosinophils and lymph mononuclear cells in the sub-mucosal and serosal layers. There was no swelling or tissue death noted; nonetheless, there were clogged blood vessels.

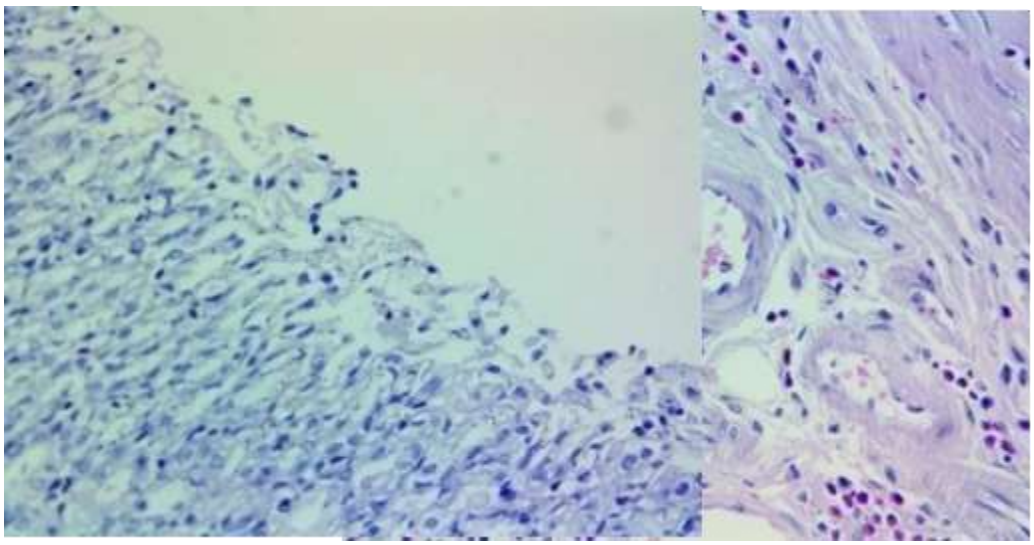

Figure 1.3. Histopathological examination of Standard group treated with Ranitidine.

2.11. Microscopically investigation

Figures 3.17 show the stomach mucosa and gastroesophageal junction. The foveolar layer has ulceration in many sites. The lamina propria has a multitude of engorged capillaries. The presence of many enlarged organs indicates regenerative activity. Visible areas of surface degradation may be seen. The submucosa shows a moderate presence of eosinophils, along with a small number of lymphocytic cells and an eosinophilic inflammatory infiltrate. There is no significant swelling or bleeding.

2.12. Test group treated with EEEOL (200 mg/kg)

Although there were no signs of edoema, haemorrhage, or extensive tissue death, a few small areas of surface erosion were seen in this specific part of the stomach, including the mucosal and gastro-esophageal regions.

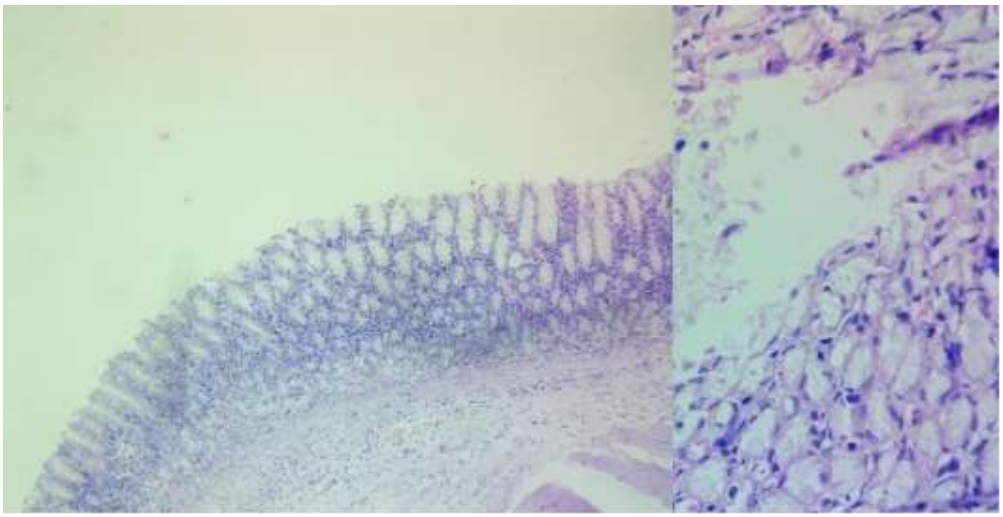

Figure 1.4.: Histopathological examination of Test group treated with EEEOL (200 mg/kg).

Microscopically investigation

Figures (3.18) depict the stomach mucosa and gastro esophageal junction. The foveolar and glandular layers seem to be undamaged, as shown by the slice. However, there are a few small regions of surface ulceration. The glandular layer is essentially ordinary. However, there is a significant presence of eosinophils and lymphoid mononuclear cells in the sub-mucosa and deeper layers of the mucosa. Usually, there is no significant tissue death, bleeding, or swelling.

2.13. Test group treated with EEEOL (400 mg/kg)

The stomach's mucosal, gastro esophageal, and typically ulcer-free regions did not exhibit any edoema or necrotic symptoms.

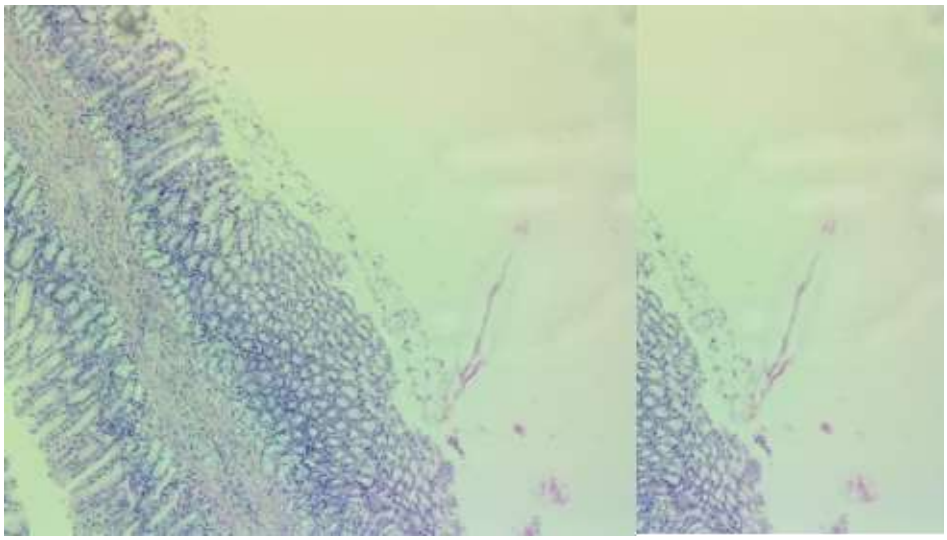

Figure 1.5. Histopathological examination of Test group treated with EEEOL (400 mg/kg)

Microscopically investigation

Figures 3.19 illustrate the stomach mucosa and gastroesophageal junction. The portions in many places indicate that the foveolar and glandular layers remain undamaged. Some areas show little erosion of the surface. The subadjacent glandular layer is unimpressive.

There are no notable abnormalities seen in the submucosa, muscular layer, or serosal. Typically, necrosis, edoema, or desquamation are absent.

3.1. DISCUSSION

Nonsteroidal anti-inflammatory medicines have the potential to cause stomach ulcers, as shown by the effectiveness of anti-secretory and cytoprotective treatments. NSAID-induced stomach ulcers are the second most prevalent cause of gastric ulcers, behind ulcers caused by Helicobacter. This might explain the reason for their extensive use. NSAIDs are linked to ulcers because they may interfere with prostaglandin synthetize in the COX-1 pathway. This disrupts the release of HCO3- and mucus, affects the blood flow in the mucosal lining, and disrupts the turnover and healing of mucosal cells.

The intestines have a preventative role. As a result, NSAIDs decrease the production of prostaglandins, leading to an increased risk of stomach ulcers and damage to the mucosal lining. NSAIDs exert an effect on Cox enzymes, particularly COX-1 and COX-2, resulting in decreased production of mucin and bicarbonate, impaired platelet aggregation, changes in the structure of small blood vessels, damage to protective layers, and decreased adherence of angiogenic or growth-promoting white blood cells. NSAIDs lead to oxidative damage to the mucosal tissue. NSAIDs may cause oxidative damage to the mucosal lining by raising the amounts of H2O2 and hydroxyl-ions, which in turn suppresses gastric peroxidases. NSAIDs, especially acidic ones, may directly damage epithelial cells.

The production of gastric acid in oxyntic glands is triggered by the release of histamine from cells such as enterochromaffin, whereas G cells produce gastrin. As a result, when given to the animals, the ethanolic extract showed anti-histaminic properties and blocked the H2 receptor in the stomach. These data indicate that EEEOL is a very efficient treatment for peptic ulcers caused by the excessive acidity of the stomach. Administering EEEOL at a dosage of 400 mg/kg showed protective effects on the stomach lining, whereas a lower dosage of 200 mg/kg resulted in little inflammation and surface ulcers. This suggests that EEEOL has gastro protective properties. This research predicted the antioxidant effect, as the treatment with EEEOL restored the level of GSH and decreased the level of MDA in a way that depended on the dosage. Overall, reactive oxygen species (ROS) worsen ulceration, which is a causative element in the substantial harm to cells, DNA, and lipids. As a result, it is aiding in the process of lipid peroxidation. Furthering clinical research, such as activity-guided fractionation, would provide further data for aspiring researchers. In the future, there will be a greater focus on studying the phytoconstituents in biological research.

4.1. CONCLUSION

Administration of EEEOL at a dosage of 400 mg/kg exhibited gastro protective properties via stimulating protective effects in the gastric mucosa. On the other hand, when the dosage was 200 mg/kg, just a little amount of inflammatory cells and surface ulcers were seen.

ROS, which may cause many types of damage to cells, DNA, and lipids, usually worsen ulceration. Thus, it is expediting the process of lipid peroxidation. Therefore, this research predicted the antioxidant impact by decreasing the level of MDA and replenishing the level of GSH using EEEOL treatment in a way that varied according to the dosage.

The study found that the ethanolic extract of Euterpe oleracea leaves shown anti-ulcer properties against stomach ulcers produced by aspirin in albino Wistar rats. It had a significant impact on the normalization of blood-related symptoms and changes in the tissue structure of the stomach.

The presence of antioxidants in this plant may enhance its ability to prevent ulcers. Hence, the possibility of using this plant as an alternate treatment for stomach ulcers is now being investigation.

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