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EVALUATION OF THE GASTROPROTECTIVE ACTIVITY OF LEAVES ETHANOLIC EXTRACT OF SENNA ALATA IN RAT MODEL

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ABSTRACT

This research evaluates the antiulcer properties of Senna alata root extract by employing a mouse model of pylorus ligation-induced ulcers. When given at dosages of 100, 200, and 300 mg/kg body weight over a five-day period, Senna alata significantly decreased the ulcer index. The maximum degree of protection was seen at a dosage of 300 mg/kg. The extract markedly decreased the amount of stomach fluid, levels of free acidity, and levels of total acidity, while concurrently elevating pH levels. In addition, it reduced the levels of pepsin and DNA, indicating a reduction in cell discharge, while simultaneously increasing the concentration of mucosal glycoproteins and mucin secretion. The findings suggest that Senna alata improves the protective function of the stomach mucosa, which is similar to the effects of Ranitidine. This indicates that Senna alata might be a practical treatment choice for managing ulcers.

KEYWORDS: Ulcer, Senna, Ranitidine, Decoction, hyperacidity, Gastroprotective activity.

1.1. INTRODUCTION

Phytochemical substances found in plants have long been used as therapeutic components and as a source of food for humans.[1] In the distant past, thriving civilizations such as the Indians, Babylonians, and Sumerians were all around.

Traditional remedies based on botanicals and plants were within the reach of the culture. The features of production, natural products, and their interactions with other living systems have been steadily and rapidly improved throughout the last two centuries, thanks to scientific and technological achievements.[2]

Approximately seventy-five to seventy-five percent of the global population will need traditional medicine for healthcare in the 21st century, according to the World Health Organisation (WHO). Instead of manufactured medications, this method will put an emphasis on medicines derived from plants. At least one plant extract is anticipated to be the active phytochemical component in almost 25% of the medications administered in India. The assertions described above are backed by research and demonstrate the significance of natural treatments produced from plants in the quest for novel medications.[3]

It is often believed that the imbalance between the mucosal membrane's defences and the assaults of acid secretions is the root cause of peptic ulcer sickness Peptic ulcer problems have been treated using medicines derived from plants. Moreover, it was shown in experimental animals that were purposefully given ulcers that the plant components had an anti-ulcer effect; this finding was subsequently validated and published. The phytochemical extracts include flavonoids, which have significant biological implications and were shown to strongly suppress ulceration in experimental mice.[4]

2. MATERIAL AND METHOD

2.1. Preparation of Extract

A granular powder was prepared by air drying A. farnesiana and pulverising it using a mechanical grinder. The finely ground material underwent a gradual extraction process utilising a heated seepage method in a soxhlet extractor, using methanol solvents. The solvent was volatilized, resulting in the formation of a viscous substance as the various concentrations decreased in volume at a temperature of 450°C. The obtained concentrations were analysed to identify their phytochemical components. The determined that the overall yield of the A. farnesiana methanolic extract was around 54.5-55%. But it was saved for until we looked into it further.[5]

2.2. General extraction process

Various techniques must be used to extract or purify raw pharmaceuticals derived from different sources, such as plant materials, depending on the physicochemical features and composition of the substances involved. The

methods for preparing mixture (a liquid extract made by steeping or soaking, known as infusion), decoction, and digesting are now prohibited by law. As a result, they are only used infrequently for extracting medicine, with a few rare cases. The maceration and permeation methods are very significant, and most pharmacopoeias cite these techniques for extracting crude pharmaceuticals.[6]

2.3. Experimental animal

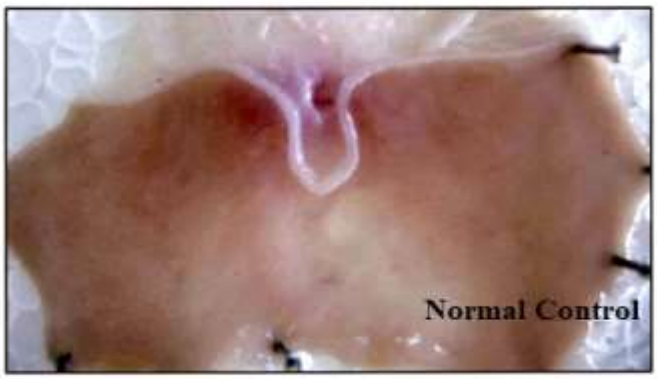
Male albino Wister rats between 1 to 2 months of age and weighing 125-150 g were procured from (Sri Venkateswara enterprises, Bangalore) and maintained in the animal house of Sree Siddaganga College of Pharmacy (123/PO/C/99/CPCSEA), Tumkur. The animals were cared for according to the guidelines outlined in the Animal User's Manual, which was issued by the National Institute of Nutrition (NIN). Over the course of a week, the animals were gradually adjusted to the conditions of our animal housing, which maintained a temperature range of 20 to 240 degrees Celsius. The light source in the animal room was regulated by a 12-hour light cycle, which was then followed by a 12-hour dark cycle. Each 41 × 28 x 14 cm cage housed two to three animals. The bedding consisted of paddy husk, which was regularly replenished and meticulously cleaned with water, Domex (a detergent), and a disinfectant every other day. The rats were provided with water ad libitum, coupled with a standard pellet diet obtained from Sai (Durga Feeds and Foods Bangalore).

2.4. Study on the antiulcer efficacy of the pylorus ligation model conducted for duration of 4 hours

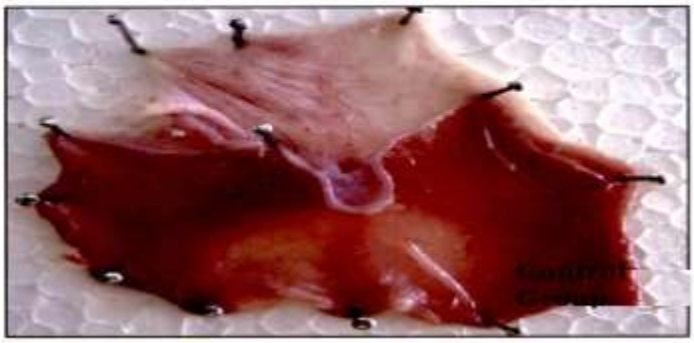
For this investigation, we followed the approach proposed by Shay and his colleagues (1945) for the treatment of ulcers. The selected criteria were examined to investigate the protective impact of selenium.

The present work was undertaken following methodology of Shay and his associates (1945) for the induction of gastric and following parameters have been chosen to investigate the protective effect of Senna alata.

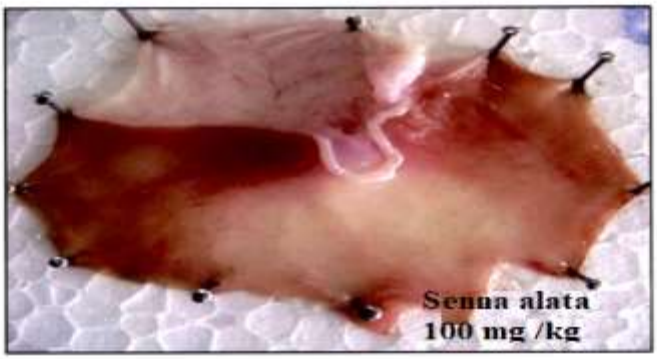
a) Stomach epithelium of normal saline treated albino rats before 4 h pyloric ligation



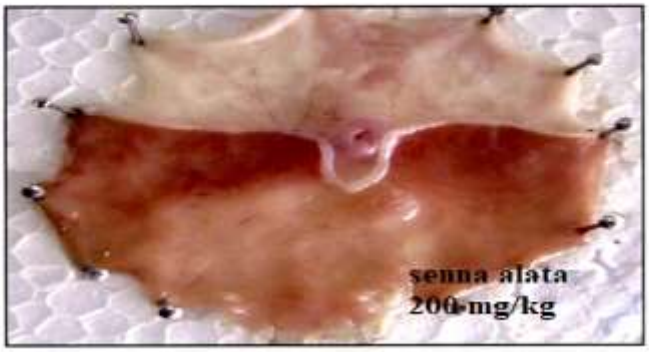
b) Stomach epithelium with gastric ulcer induction in albino rats after 4 h pyloric ligation



c) Stomach epithelium of pylorous ligated albino rats pretreated with Senna alata 100 mg/kg

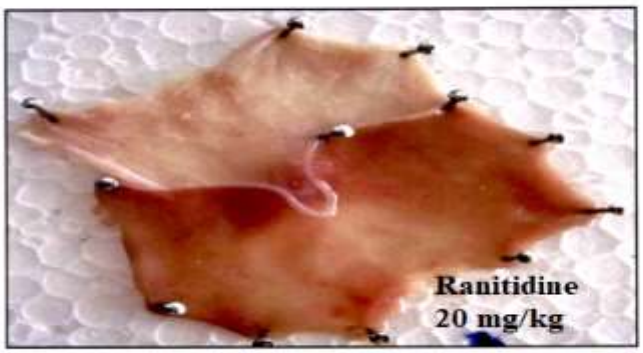


d) Stomach epithelium of pylorous ligated albino rats pretreated with 4 Senna alata 200 mg/kg



e) Stomach epithelium of pylorous ligated albino rats pretreated with Seena alata 300 mg/kg

e) Stomach epithelium of pylorous ligated albino rats pretreated with 20 mg/kg Ranitidine



2.5. Evaluation of gastro protective activity

- The impact on the capacity, pH, free acidity, total acidity, pepsin content, DNA content, and mucin secretion in gastric juice.
- Concentration of mucosal glycoproteins m the gastric mucosa.

A research was undertaken to evaluate the effectiveness of Senna alata root extract in treating ulcers. The study used a pylorus ligation-induced ulcer model and administered the extract at dosages of 100, 200, and 300 mg/kg body weight. The uraia picta root extract is given intravenously to all three dose groups. On the sixth day, mice that had been disadvantaged of food for 24 hr were exposed to the tests once a day for five consecutive days. The ulcer index decreased, and the antiulcerogenic effect of Senna alata was shown to rise in a way that depended on the dosage. The most favourable outcome was seen while administering a dosage of 300 mg per kilogramme of body weight. The ulcers were evaluated and examined using the same methods as previously explained.

3.1 Ulcer index

In the 4h pylorus ligation model, the ulcer size in the groups treated with Ranitidine has dropped to 13.06, associated to the control group which has a value of 22.33. Ulcer index in the groups treated with Senna alata was decreased to 18.2, 13.89 and 11.23 ($P<0.05$) at dosages of 100, 200 and 300 mg/kg, singly, associated to the control group. The percentage of protection is 39.45%, and there is a significant difference in the results at a dosage of 300 mg/kg compared to the control group (Table 3.2 and Figure 3.1).

Table 3.1: Outcome of Senna alata on Ulcer index in 4h PL model.

Sr. No.	Treatment/Groups	Dose mg/kg	Ulcer index	Percentage Protection
1	Control	saline	22.33	……..
2	Ranitidine	20	13.06	42.02%
3	Senna alata	100	18.23	17.06%
4	Senna alata	200	13.89	18.67%
5	Senna alata	300	11.23	39.45%

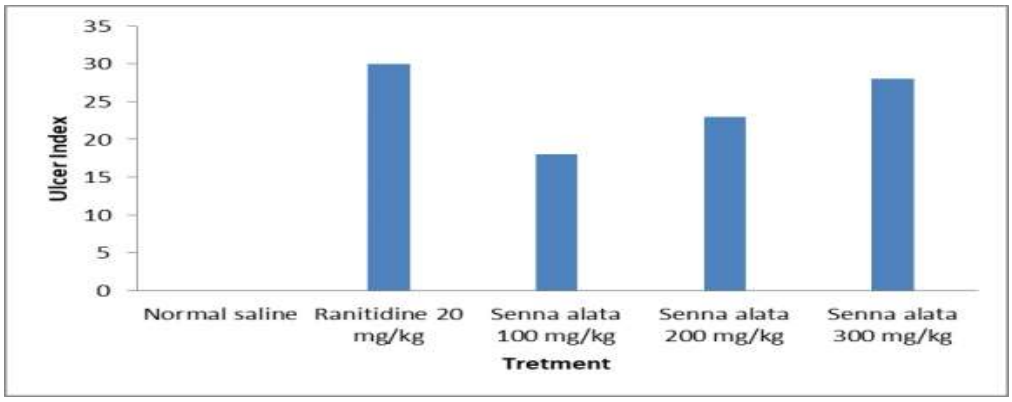


Figure 3.1: Outcome of Senna alata on Ulcer index in 4h PL model.

3.3.1 Effect on volume of gastric juice secretion

The Ranitidine cured group saw a substantial reduction in gastric juice secretion volume, dropping to 1.85±0.08 ml associated to the control group. The volume of the control group is 2.77 ±0.11 ml. Associated to the saline control, the groups treated through Senna alata showed a decrease in gastric juice volume to 2.65±0.13ml, 2.02±0.02 and 1.88±0.07ml at dosages of 100, 200 1nd 300 mg/Kg, singly (Table 3.3 and Figure 3.4).

Table 3.2: Outcome of Senna alata on volume of gastric juice in 4h PL model.

Sr. No.	Treatment/ Group	Dose mg/kg	Volume of gastric juice (ml/100mg) mean± SEM
1	Control	Saline	2.45±0.01
2	Ranitidine	20	1.85±0.08*
3	Senna alata	100	2.65±0.13
4	Senna alata	200	2.02±0.02
5	Senna alata	300	1.88±0.07*

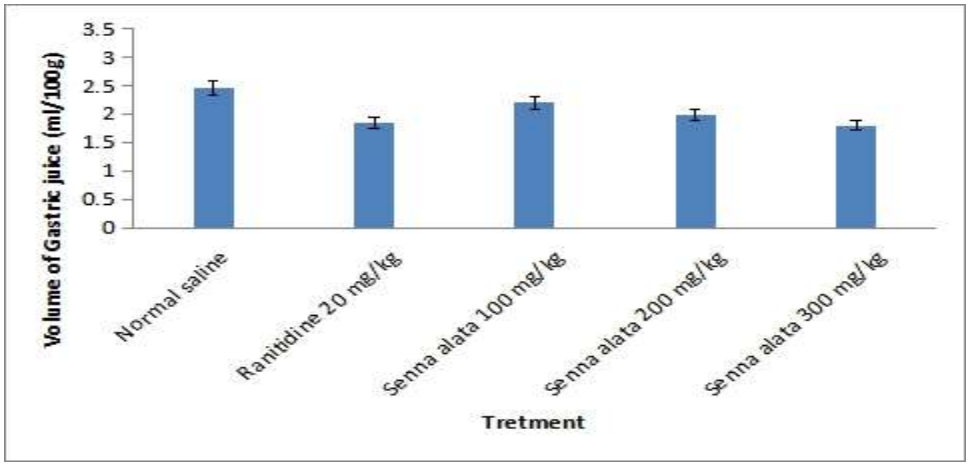


Figure 3.2: Outcome of Senna alata on volume of gastric juice in 4h PL model.

3.3.2 Effect on pH of Gastric juice

The pH of stomach fluid in the Senna alata-treated groups was higher compared to the control group. At dosages of 100, 200 1nd 300 mg/kg, the pH rose to 1.82±0.06, 1.99±0.04 and 2.54±0.07, respectively (P<0.05). The pH of the stomach fluid in the group treated with Ranitidine has risen to 2.65±0.07.

Table 3.3: Effect of Senna alata on pH of Gastric juice in 4h PL model.

Sr. No.	Treatment/ Group	Dose mg/kg	pH mean± SEM
1	Control	saline	1.66±0.07
2	Ranitidine	20	2.65±0.07
3	Senna alata	100	1.82±0.06
4	Senna alata	200	1.99±0.04
5	Senna alata	300	2.54±0.07

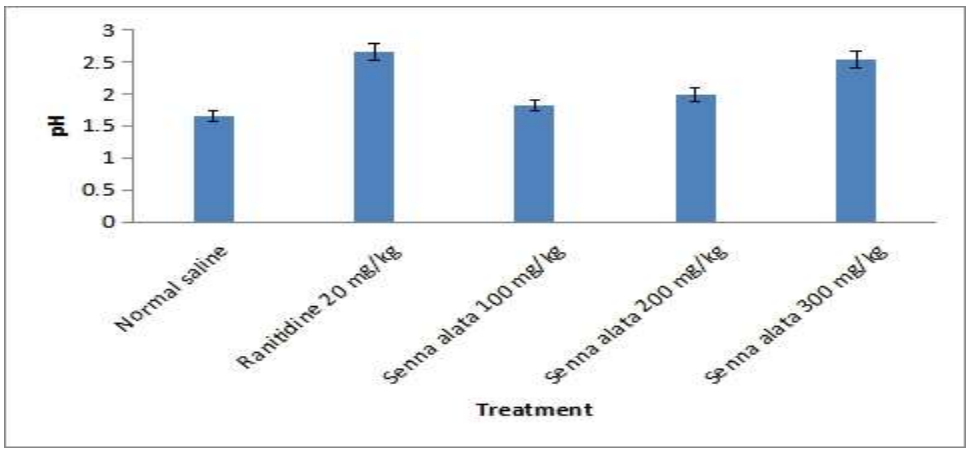


Figure 3.3: Effect of Senna alata on pH of Gastric juice in 4h PL model.

3.3.3 Effect of free acidity

The control group had a free acidity of 61.76±1.4Meq/1, whereas the Senna alata-treated groups had a free acidity of 57.12±1.7Meq/1, 49.07±1.8 Meq/l and 46.36±1.99Meq/1 at dosages of 100, 200 1nd 300mg/kg body weight, respectively. The Ranitidine-treated group had a free acidity of 43.65±1.07Meq/L, which is lower than the free acidity of the control group.

Table 3.4: effect of Senna alata on free acidity in the gastric juice in 4h PL model.

Sr. No.	Treatment/ Group	Dose mg/kg	pH mean± SEM
1	Control	saline	61.76±1.4
2	Ranitidine	20	43.65±1.07
3	Senna alata	100	57.12±1.7
4	Senna alata	200	49.07±1.8
5	Senna alata	300	46.36±1.99

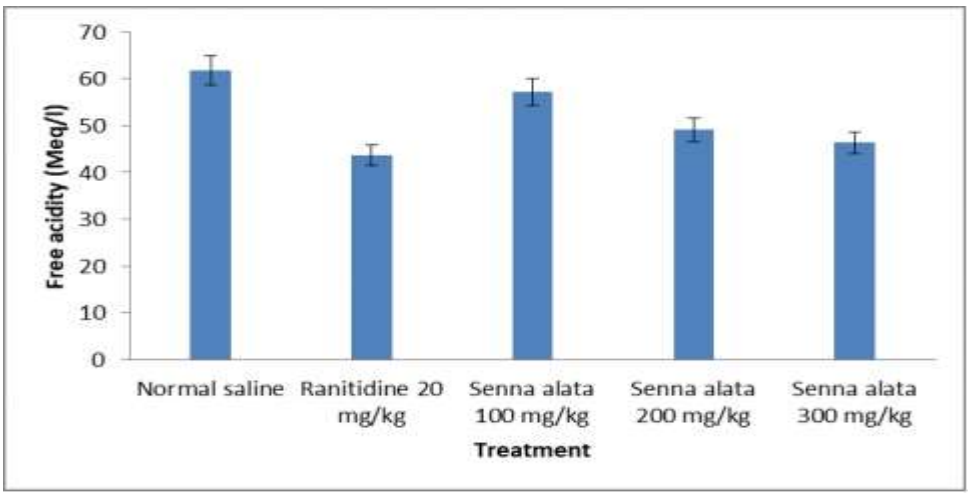


Figure 3.4: Effect of Senna alata on free acidity in the gastric juice in 4h PL model.

3.3.4. Effect of Total acidity

The Senna alata-treated groups showed a decrease in total acidity to 87.23±1.93Meq/1, 80.15±1.97 Meq/1 and 71.77±2.04Meq/1 at dosages of 100, 200 1nd 300mg/kg body weight, respectively. In comparison, the control group had a total acidity of 92.22±1.7Meq/1. The reference medication Ranitidine-treated group exhibits a decrease in total acidity to 71.66±1.14 Meq/1.

Tablet 3.5: Effect of Senna alata on total acidity in the gastric juice in 4h PL model.

Sr. No.	Treatment/ Group	Dose mg/kg	pH mean± SEM
1	Control	saline	92.22±1.7
2	Ranitidine	20	71.66±1.14
3	Senna alata	100	87.23±1.93
4	Senna alata	200	80.15±1.97
5	Senna alata	300	71.77±2.04

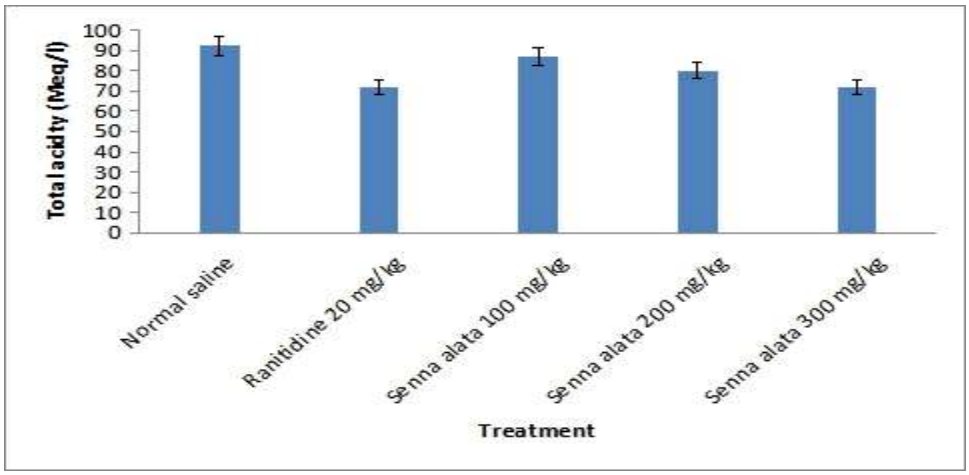


Figure 3.5: Effect of Senna alata on total acidity in the gastric juice in 4h PL model.

3.3.5 Effect on pepsin cotent

Compared to the animals in the control group, the pepsin concentration in the groups treated with Senna alata was decreased to 245.34±23.67pmol/ml, 205.18±29.9 pmol/ml and 167 ±31.9pmol/ml at dosages of 100, 200 1nd 300mg/kg body weight, respectively, whereas it was 290.3±32.8pmol/ml. The administration of Ranitidine to animals leads to a decrease in the content of pepsin in their blood to a level of 183.7±30.3pmol/ml. At a dosage of 100, 200 mg/kg body weight, selenium has little impact on the amount of stomach fluid, pH, and acid concentration. Administering a dosage of 300 mg/kg body mass results in a substantial inclination to reduction the amount of gastric fluid, elevate the pH level, and lower the concentration of acid-pepsin. The reference chemical, Ranitudine, which is a cytoprotective agent, likewise shown the ability to decrease the volume, pH, and acid content of stomach fluid.

Table 3.6: Effect of Senna alata on on gastric mucosal glycoprotein I 4h Pl rats, Mucosal carbohydrates and proteins in µg/ml.

Sr. No.	Treatment	Dose (mg/kg)	Total hexoses	Hexose amine	Fucose	Slalic acid	Total carbohydrate	Total Proteins	Tc:P ratio
1	Control	saline	272.34±14.56	180.6±7.8	63.55±2.8	24.99±0.56	537.23±12.30	523.98±0.9	1.06
2	Ranitidine	20	321.6±12.34	182.34±5.1	67.60±0.88	33.55±1.80*	600.66±14.3*	431.65±8.6*	1.55±0.06*
3	Senna alata	100	300.88±6.78	180.33±5.8	66.89±1.56	31.77±1.67	576.30±6.5	499.90±21.98	1.19±0.05
4	Senna alata	200	307.34±3.98	187.12±1.45	69.23±1.86	35.78±1.34	582.56±6.9	505.01±1.99	1.45±0.18
5	Senna alata	300	315.23±2.34	192.45±1.78	71.67±1.90	39.43±1.78	592.45±6.2	525.78±1.67	1.55±0.05

4.1. DISCUSSION

An imbalance between the stomach's defensive systems and the digestive tract's damaging chemicals is the frequent cause of gastric hyperacidity and ulcers. There has been new hope for the treatment of this terrible sickness because to research developments in this domain since 1990. The gastroduodenal mucosa's defensive cytoprotective system has been the focus of attention. Breakthroughs in diet and medicine that might one day prevent peptic ulcers have emerged in the past 20 years, according to study. Desai et al. (1997) outlined strategies for achieving this goal, however they focused on minimising aggressive acid-pepsin release rather than enhancing the defensive cytoprotective mechanisms of the gastroduodenal mucosa. It is unclear whether decreased mucosal defences in cytoprotection or increased acid pepsin synthesis has a more significant impact in the aetiology of peptic ulcers. Forty to seventy percent of patients with duodenal ulcers have normal acid secretion. Peptic ulcers do not always form in people with Zollinger-Ellison syndrome, even though all of these patients have excessive acid secretion. Consequently, it is expected that these people must possess an exceptionally robust mucosal defence system (Kang et al 1993). As a defensive mechanism, acid eliminates toxic substances and germs from food we consume on a daily basis. The stomach produces its own acid as a protective mechanism against self-digestion, according to Szabo (1981). Here are the included aspects: safeguarding the epithelial mucosa, enhancing blood flow to the mucosa, enhancing endogenous prostaglandin synthesis, and secreting mucosal bicarbonate that functions as a barrier.

After a four-hour ligation, this study intends to examine the effects of Senna alata on several gastric secretion activities, such as volume, pH, coffee acidity, overall acidity, pepsin concentration, DNA content, mucus secretion, and concentration of mucosal glycoproteins.

There are a number of ways in which synthetic NSAIDs, such as aspirin, can harm the mucosal lining. These include rupturing the protective barrier, encouraging the backward movement of H+ ions, increasing acid production, and interfering with the manufacture of prostaglandins (Vane, 1971; St John, 1973). The mucosal mast cells, which are located on the mucous membrane's outer layer, are susceptible to disruption by ethanol. Histamine, platelet activating factor, LTC4/D4, and other vasoactive mediators are produced as a result of this disturbance. According to Wallace et al. (1982) and Miller and Henagan (1984), these chemicals damage the mucosa lining the gastrointestinal tract.

In pathogenic processes, alterations in gastric secretion, abnormalities in abdominal gastric motility, and disruptions in gastric mucosal microcirculation have been associated with stress-induced stomach mucosal lesions (Garrick et al, 1986; Tariq et al, 19g6).

According to Goel and Bhattacharya (1991), peptic ulcers may develop when the stomach's fluids digest themselves, the mucosa receives less blood, and the protective mucosal barrier breaks down. For the purpose of screening and evaluating antiulcer medications, the Shay model was established in 1945 by Shay et al. and is a dependable and readily repeatable methodology. It has a stellar reputation for being quite predictable. It doesn't rely on any outside elements that may impede the process or employ any outside chemicals that might induce ulcers. The conventional wisdom is that ulcers develop when the mucosal lining's defensive function is overwhelmed by acid production. Studies have shown that the body's natural prostaglandin synthesis helps in mucosal adaptability (Konturek et al., 1982; Robert et al., 1983). Aspirin (Brzozowski et al., 1995), necrotizing chemicals (Robert et al., 1983), and stress (Konturek et al., 1990) were the first NSAIDs to show signs of adaptation in stomach mucosa. However, nobody has looked at how this adaptation happens or how it affects

the stomach mucosal defence mechanism against necrotizing agents.

Goel and Bhattacharya (1991) conducted a comprehensive investigation to investigate the function of PGs in controlling the mucosal defence of the gastroduodenum. A large body of evidence indicates that prostaglandins (PGs) protect the gastroduodenal mucosa, making it more resistant to harm and less susceptible to the apparent damage that many chemicals may cause. The actions of prostaglandins, which suppress acid secretion, are not relevant to this function. It is anticipated that the potential for PGs to directly increase cellular resistance to injury would not significantly impact their overall effect. So, to better capture the impact of PG on the duodenal and stomach defences, the term "mucosal protection" seems to be the most appropriate and correct choice.

As shown by Peskar et al. in 1991, there are other studies that imply chemicals other than PG may aid adaptive cytoprotection. Senna alata may preserve the mucosal lining's endogenous prostaglandins (PGs) and other protective components, according to our study.

Although the exact mechanisms by which PGs exert their beneficial benefits remain a mystery, research suggests that they may boost mucus formation (Johansson and Kollberg, 1979) and bicarbonate secretion (Ross and Tumberg, 1983). This holds truest when it comes to PGE2. To protect cells from potentially dangerous stimuli, prostaglandins (PGs) may block ion transport and potential difference changes. In addition to reducing acid production, they may improve blood flow to the mucosal lining. Research by Robert et al. (1976), Whittle (1977), Chaudhury and Jacobson (1978), and Tepperman et al. (1978) has shown these effects. The PGE and PGF series were able to demonstrate protection against mucous membrane damage induced by numerous hazardous chemicals. This characteristic, however, has nothing to do with acid in the stomach. Ranitidine and other cytoprotective medications are synthetic prostaglandin (PG) derivatives that have shown promise in the prevention of stomach ulcers.

Continued research has shown that mucosal glycoproteins and mucus formation contribute to Senna alata's adaptive cytoprotection, offering supplementary defensive mechanisms. In 1908, Kaufmann first proposed the notion of investigating the causes of peptic ulcers by analysing the properties of stomach mucus. Glass (1953) characterised visible mucus as an intricate gel. Investigating how ulcerogenic and ulcer-healing medications influence the formation and make-up of gastrointestinal mucus is the current focus of molecular pathophysiology research into ulcers.

Mucus is an organism's built-in defensive mechanism. But it's a shame nature didn't think of every bad thing that may ruin mucus' defensive qualities in good health and in illness. Although it might be annoying when there's too much or too enough mucus, it's essential for the proper operation of several bodily processes. The evolutionary history of mucus is extensive, and it performs several roles, such as lubricating, waterproofing, and protecting the organism from potentially harmful fluctuations in osmotic pressure. When it comes to the digestive system, mucus is like the skin: it does its job just as well, if not better, than the skin on your body. The gastrointestinal mucosa is also protected from the stomach's powerful acid and proteolytic discharges by this factor.

The stomach fluid is contained behind the cohesive protective barrier that created by the mucus, which safeguards the living lining of the gastrointestinal tract. It is resistant to biochemical damage and bacterial infection because to its strong integrity. In this field, medical researchers and doctors may work together to control secretions for disease prevention and treatment by conducting in-depth investigations. To prevent stomach ulcers, the mucus layer is the main line of defence. As a protective barrier, mucus is composed of complex molecules. Stress and certain drugs, including aspirin, may interfere with it. Glycosylation and the subsequent production of mucus glycoprotein may be impeded by these circumstances.

In order to find out how mucin and mucosal glycoprotein were doing, researchers measured various percentages of mucosubstances in the stomach fluid and mucosa, including total hexoses, hexosamine, fucose, sialic acid, and proteins. According to Goel and Bhattacharya (1991), the ratio of total carbohydrates to protein is a valid indicator of mucosal resistance, which in turn reflects the functional integrity of the gastric mucosal barrier.

As a reference chemical, Ranitidine was used. Clinically, Ranitidine helps critically sick patients prevent stress ulcers. According to clinical studies, Ranitidine helps stomach ulcers heal faster and reduces the likelihood that they will return. One possible explanation for these side effects is the thickening of stomach mucus.

The cytoprotective effects of Senna alata on mucosal glycoprotein concentrations are the focus of this study. The results show that the ulcer index decreased and the glycoprotein content increased. The ratio of total carbohydrates to protein in mucosal glycoproteins and stomach fluid mucoproteins increased significantly after 4 hours after ligation. Senna alata was responsible for this effect. The obtained result is consistent with Senna alata's ulcer-protective characteristics, as shown by an increase in the TC:P ratio. The effectiveness of selenium in preventing stomach ulcers was enhanced because it led to a discernible shift in the concentration of mucin and mucosal glycoproteins in the stomach fluid.

An essential metric for assessing the degree of damage or disintegration of gastric mucosa cells is the quantity of DNA in stomach fluid, according to Mukhopadhyay et al. (1987). When exposed to ulcer genic chemicals, this indicator rises, and when exposed to ulcer protective agents, it falls. The decrease in DNA content in the stomach fluid thus demonstrated the substantial protection of the gastric mucosa by Senna alata. Evidence suggests that prostaglandins (PGs) play a unique function in Senna alata's cytoprotective effects. Also, Senna alata bolstered the stomach's defenses by increasing glycoprotein concentration and mucus synthesis, two essential components of stomach lining defenses.

3.5. DISCUSSION

Sections of gastric mucosa gotten from Senna alata root extract pre-treated doses at 50, 150 and 300 mg/kg, correspondingly. Red arrow: surface epithlium damage; Black arrows: inflammatory infiltrate composed of polynuclear eosinophyles.

When compared to rats given ethanol as a control, animals given Senna alata (300 mg/kg) had a much lower ulcer index. Intestinal mucosa irritation and bleeding may occur in both animals and people when exposed to ethanol and some NSAIDs, such aspirin. In 1981, Kontrruek et al. published the referenced work. The first stage in the development of ethanol-induced damage to the stomach lining, according to research on the subject (Kontrruek et al., 1990), is the breakdown of the lining of the blood vessels. This leads to increased permeability of the blood vessels, swelling, and a rise in epithelial cells.

The cytoprotective action of Senna alata seems to have a role in the present study's defence against stomach ulcers caused by ethanol. Its effect on mucus production lends credence to this. A key component in determining the state of mucosal resistance is the condition of mucus production. In 1987, Green et al. investigated the process by which ethanol causes gastric mucus to gel on the mucosal surface. In order to see this process, they used alcian blue, a basic pigment that forms bonds with acidic mucopolysaccharides. A reduction in stomach mucus content is one of numerous causes that lead to ethanol-induced gastric lesions. The production of secretions from the stomach wall was shown to decrease following ethanol administration, according to Al Harbi et al. (1997). The present study shows that Senna alata, when administered at a dose of 300 mg/kg, successfully inhibited the ethanol-induced decrease in stomach mucus.This protective mechanism of Senna alata against necrotizing substances like ethanol implies that endogenous prostaglandins play a role.

We saw several long, red bands of lesions in the stomach mucosa after injecting ethanol straight into the stomach. Observed in the This is seen in Figure 10. In the groups that received Senna alata at dosages of 100, 200 mg/kg and 300 mg/kg, respectively, the levels of TBA reactive chemicals in the stomach mucosa, which indicate lipid peroxidation, were found to be 10.28±0.3 nmol/mg protein and 8.86±0.84 nmol/mg protein, respectively. There was a statistically significant reduction in TBA reactant concentration in the selenium-treated groups.

These findings support the hypothesis that Senna alata may protect against ethanol-induced damage. Results show that 300 mg/kg of Senna alata has a protective effect that increases with dosage. Gastric mucosal damage was significantly reduced when the necrotizing substance was administered into the stomach. At the same time, we found that the concentration of lipoperoxide had dropped significantly.

In 1998, Martin et al. found that oxygen-free radicals have a direct role in the development of ethanol-induced mucosal injury. The veins constrict strongly and rapidly after ethanol injection, and the tiny arteries enlarge shortly thereafter. Mucosal capillary dilatancy is caused by a conglomeration of microvascular events (Oates and Hakkinen, 1988). The formation of ethanol-induced stomach lesions is influenced by the reactive oxygen species that are produced during the hypothesised ischemia-reperfusion intervals. Superoxide ions and other reactive radicals may be elevated in the mucosa as a result of the hypoxia (Salim, 1990). A probable reason why ethanol causes free radicals to develop might be because it speeds up the breakdown of purines. This could be because it switches from the dehydrogenase pathway to the xanthine oxidase pathway (Glavin and Szabo, 1992). Disruptions to the cell membrane's function are caused by the generation of lipid hydroperoxides and alkoxy radicals. Results that were achieved since the stomach mucosa was exposed to the necrotizing agent, who resulted in a considerable increase in lipid peroxidation levels, the results is consistent with our experimental circumstances.

De Groot et al. (1996) found that substances with antioxidant qualities might potentially attach to cell membranes and inhibit lipid peroxide formation, therefore preventing caused illnesses. A powerful antioxidant with properties that limit lipid peroxidation, selenium methylthionine has gained widespread recognition. Our results are in line with previous findings, as the treatment groups showed a substantial and gradual reduction in lipid peroxide levels compared to the ethanol-only group.

3.6 CONCLUSION

The study's results indicate that Senna alata has a notable antiulcer effect in the pylorus ligation model. The findings demonstrate that there is a reduction in the ulcer index that is directly proportional to the dosage, with the most effective outcome obtained at a dosage of 300 mg/kg. Senna alata shown significant efficacy in reducing gastric juice volume, free acidity, total acidity, and pepsin content, while simultaneously elevating pH

levels. These findings indicate that Senna alata may have a beneficial impact in protecting the stomach mucosa.

In addition, the extract increased the production of mucin and the level of mucosal glycoproteins, which are essential for preserving the integrity of the stomach lining. The effects of Senna alata are similar to those of Ranitidine, a recognized antiulcer drug. This demonstrates the possibility of Senna alata as a natural treatment alternative for managing ulcers. This study provides evidence in favour of the conventional use of Senna alata for the treatment of stomach ulcers. It emphasizes the need for more research to thoroughly understand its mechanisms of action and prospective therapeutic uses.

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