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Histological Effects of Aloe Vera Ethanol Leaf Extract Gel on Ibuprofen-Induced Gastric Ulceration in Adult Male Wistar Rats

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ABSTRACT

Introduction: *Stomach ulcers are overt wound which developed on the epithelial layer of the gastric mucosa. The global burden of gastric ulcers affects millions of people annually, with a lifetime prevalence of 5% to 10%. Aloe vera gel has been found to have effects on the gastrointestinal system, this study aims to investigate the histological effects of Aloe vera on the histology of stomach ulcer-induced Wistar rats.*

Materials and Methods: *Thirty adult male Wistar rats weighing between 190 ± 10 g were employed for this study. The rats were randomly assigned to six groups (1-6) of five rats each. All rats were fed with normal rat chow for 1 week and given water ad libitum, kept and maintained under standard laboratory conditions. Animals in group 1 were the normal control, Animals in groups 2-4 were treated with a single dose of Ibuprofen 200 mg/kg. Groups 3 and 4 were treated with Aloe vera leaf gel extract at 200 and 400 mg/kg respectively for 14 days, group 5 animals were pre-treated with oral administration of Aloe vera gel extract for 14 days at a concentration of 200 mg/kg before the ulcerogenic procedures, Group 6 was treated with standard medication (Omeprazole) after inducing ulcer, while Group 2 animals were left untreated and sacrificed immediately. Gastric ulceration, was induced in 18 hours fasted rats by the oral administration of a ulcerogenic drug, Ibuprofen 200 mg/kg.. At the end of the experimental period the rats were sacrificed under 50mg/kg of ketamine anesthesia administration.*

Results: *Showed normal histoarchitectural layout of mucosa in the normal group 1, while the untreated ulcer group showed serious sloughing of the gastric epithelial lining, altered arrangements of glandular tissues in the mucosal region and eventual gastric cell death. In the low dose Aloe vera leaf gel, the section showed sloughing with mild hemorrhagic areas, with few white blood cell infiltration of the mucosal layer, the ulcer group treated with high dose aloe vera leaf gel, showed mild sloughing with areas of petechial hemorrhages. Aloe vera pre-treated group showed normal intact layers all through the mucosal epithelial lining intact submucosal layer and muscularis, while, ulcer group treated with Omeprazole, showed mild sloughing of gastric epithelium.*

Discussion: *The results from the study showed that ibuprofen actually induced gastric ulceration at 200mg/kg in the Wistar rats as evidenced by sloughing of the epithelial lining of the mucosa layer. The low dose and high dose Aloe vera gel extract offer healing effects on the injured gastric mucosa, in a similar manner with the standard ulcer drug. Additionally, the prophylactic effect of aloe vera gel extracts has more profound protective influence on the gastric epithelial mucosa of the ulcer-induced Wistar rats than the therapeutic effect.*

Conclusion: *This study concluded that ibuprofen caused gastric ulceration, the Aloe vera gel extract at varying doses including Omeprazole were able to ameliorate the ulcerogenic effect, however the prophylactic effect of Aloe vera is more efficacious than its therapeutic effect.*

Keywords
Gastric mucosa, Aloe Vera, Stomach ulcers.

Introduction

The gastric mucosa is the protective mucous membrane lining the stomach. It contains gastric glands and surface mucous cells that secrete mucus, which shields the stomach wall from acidic and enzymatic damage. This mucus is produced predominantly by the pyloric glands in the distal stomach, with smaller contributions from the body and fundus glands [1,2].

Disruption of this protective barrier by factors such as acid hypersecretion, nonsteroidal anti-inflammatory drugs, or infection predisposes the mucosa to injury [3]. The injury usually starts as Gastritis, characterized by gastric lining inflammation, which may be acute or chronic [4]. Persistent damage may progress to gastric erosion, involving shallow defects confined to the mucosa [5]. With continued insult and impaired healing, deeper lesions develop as gastric ulcer, extending beyond the mucosa into the submucosa or muscularis [6].

Severe or untreated ulcers may further advance to gastric perforation, resulting in full-thickness disruption of the gastric wall and leakage of gastric contents into the peritoneal cavity [7]. In chronic cases, especially when the pyloric region is involved, repeated injury and healing can lead to fibrosis and narrowing, culminating in gastric outlet obstruction, which significantly impairs gastric emptying [8].

Peptic ulcer disease remains a significant global health concern, affecting millions of individuals worldwide and contributing substantially to morbidity. It is characterized by ulceration in the stomach or duodenum, involving a breach in the gastrointestinal mucosa that extends into deeper layers [9].

Clinically, it presents with symptoms such as abdominal pain, bloating, nausea, weight loss, and early satiety [10]. Regardless of the duration of the disease, acute and severe complications, including bleeding, perforation or gastric outlet obstruction, can develop [11].

The most prevalent cause of gastric and duodenal ulcers is thought to be associated with infection by *Helicobacter pylori* and the widespread usage of non-steroidal anti-inflammatory drugs (NSAIDs) [3]. Moreover, more than twenty percent of peptic ulcer cases are H. pylori-negative and NSAID-negative, but are influenced by certain factors such as life-threatening trauma, psychological stress, sex, smoking, age and having chronic medical conditions are associated with a higher prevalence of peptic ulcer disease [11].

Gastric ulcer is a form of peptic ulcer, characterized by lesions that develop within the mucosal lining of the stomach, resulting in a breach in the gastric mucosa extending into the deeper layers of the stomach wall [12]. It is a multifactorial and complex disease resulting from an imbalance between defensive and aggressive factors within the gastrointestinal tract (stomach) [3,13].

The aggressive factors are endogenous factors like gastric acid, endothelins and pepsin secretion, active free radicals and oxidants, leukotrienes, and exogenous factors like ethanol or nonsteroidal anti-inflammatory drugs (NSAIDs). On the other side, gastric mucus, bicarbonate, normal blood flow, prostaglandins (PGs), nitric oxide (NO), and antioxidant enzymes like catalase (CAT) and glutathione (GSH) work as a defensive barrier, Mucosal cell death results from an increase in H+ concentration in its immediate environment due to this pH reduction [3].

Epidemiology report showed that between 1990 and 2019, the global prevalence of peptic ulcer increased from over 6 million cases to about 8 million cases, while the mortality rate declined from 278,979 to 236,139 over the same period.

The burden of the disease was observed to increase with age, with the highest prevalence, mortality, and disability-adjusted life years occurring among individuals aged 80–84 years and those aged ≥85 years. Additionally, males exhibited higher age standardized prevalence rates than females across most age groups, except among individuals younger than 25 years [11].

Huang et al. [14] also reported that 'peptic ulcer disease affects 5–10% of the world's population, with 12% of males and 10% of females reporting lifelong peptic ulcer occurrence'. In Africa, the prevalence of peptic ulcer disease has been reported to be 15.2%, with duodenal ulcers (10.2%) more common than gastric ulcers (5.2%). Prevalence varied by region, highest in West Africa, and showed a slight increase over time.

Helicobacter pylori infection was highly prevalent, particularly among PUD patients, highlighting its key etiological role [15].

Medication usage, especially nonsteroidal anti-inflammatory drugs (NSAIDs), is widely recognized as a significant factor contributing to gastric mucosal injury. However, the use of NSAIDs is directly linked with the development of gastric ulcers, with approximately 25% of chronic users experiencing gastric ulcer disease [16].

Ibuprofen is an analgesic and antipyretic drug belonging to the NSAID class and is a non-selective inhibitor of both cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2) [17,18]. Notably, ibuprofen-induced inhibition of COX activity plays a role in suppressing prostaglandin (PG) synthesis, which alleviates pain, inflammation, and fever [19].

However, NSAIDs intake causes gastrointestinal side effects that vary depending on the dose and patient population [20]. Importantly, these side effects include ulceration of the gastric mucosa, enhanced ulcerogenic response to stress, and hindered gastric ulcer healing [21]. Furthermore, it's reported that 'about 70% of patients with long-term NSAID ingestion have endoscopic

abnormalities (mucosal erosions, ulceration and subepithelial haemorrhage) despite only 10% complaining of dyspeptic symptoms' [22].

Omeprazole, a proton pump inhibitor, has been widely used in the treatment of peptic ulcer disease and has been extensively compared with other therapeutic agents in several studies, particularly H2 receptor antagonists. Most findings consistently show that omeprazole achieves a higher ulcer healing rate, shorter healing time, and better prevention of ulcer recurrence compared to H2 receptor antagonists [23]. This superiority is mainly due to its more potent and sustained inhibition of gastric acid secretion, which reduces acid-mediated irritation and damage to the gastric mucosa, thereby promoting more effective ulcer healing. In addition, studies have evaluated the use of omeprazole in combination with antibiotics for the treatment of *H.pylori* associated ulcers, where it plays a key role in improving eradication rates and enhancing overall treatment outcomes [23].

However, in certain patients, omeprazole may cause serious adverse effects, including hypersensitivity reactions, skin rashes, and impaired liver function. In addition, prolonged use of the drug has been associated with disturbances in nutrient absorption, particularly reduced uptake of vitamin B12, magnesium, and other essential trace elements [24].

Natural products have long been used for therapeutic purposes, and many modern drugs are derived from plant sources [25].

Aloe vera is one of the most effective bioactive plants within the Aloe genus, containing approximately 75 nutrients and over 200 bioactive compounds [26]. A.vera compositions include vitamins, minerals, amino acids, enzymes, and polysaccharides, which contribute to its wide-ranging therapeutic efficacy. The medicinal properties of Aloe vera are largely attributed to polysaccharides present in the inner parenchymatous tissue of the leaf [27]. It has been reported that the leaves, which consist of 3 main layers (the outer rind, the sap or latex layer, and the inner gel layer), are a source of organic acids, phenolic compounds, enzymes, vitamins, and minerals [28].

A.vera gel has been extensively utilized for wound healing due to its ability to promote tissue repair, inhibit microbial growth, and reduce inflammation [25,28]. Additionally, A.vera gel has demonstrated effectiveness in the management of burns, skin injuries, edema, and pain. Beyond dermatological applications, the gel has shown protective effects against gastric ulceration in both humans and experimental animal models, likely due to its antiinflammatory, antioxidant, cytoprotective, and mucus-enhancing properties [29].

Materials and Methods

Drugs and Chemicals

Ibuprofen and Omeprazole were gotten from MOHAS pharmaceutical Nigeria Limited, opposite Phase II, Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun-State, Nigeria.

Varying grades of ethanol, all other chemicals used were of the best analytical grades from reputable companies in the World. Distilled water was obtained from Department of Biochemistry and Molecular Biology, Obafemi Awolowo University (OAU), Ile-Ife, Osun State.

Preparation of plant extracts

Fresh Aloe vera leaves were gotten from Giwa's Garden along OAU Road 7, the leaves were averagely 45 cm long and 550 g in weight, harvested from healthy Aloe vera plants. The plant was identified by a taxonomist and a voucher number was issued and a voucher specimen was deposited at the department of Botany Obafemi Awolowo University Ile-Ife for future reference. The plants were washed with distilled water and the thick epidermis were peeled off. The gel was scooped with a spatula. The rind was selectively removed and the colorless parenchyma was grounded in a blender and centrifuged at 10,000 g to remove the fibers. The supernatant was lyophilized and stored at 4°C.

Known amount of the lyophilized powder was extracted with 95% ethanol and nearly 85% of the solvent was recovered by distillation over the boiling water bath at atmospheric pressure and the remaining under reduced pressure in rotavapor. A known amount of solvent free extract was suspended in water to obtain the desired concentration of the Aloe vera leaf gel.

Pretreatment Studies

Ethical clearance for the study was gotten from health research ethics committee (HREC), institute of public health (IPH) Obafemi Awolowo University (OAU) Ile-Ife. The animals were given humane treatment according to the guidelines of HREC, IPH, OAU.

Experimental Animals

Thirty adults male Wistar rats weighing between 190 ± 10 g were employed for this study. The animals were randomly assigned to six groups (1-6) of five rats each. All rats were fed with normal rat chow for 1 week and given water *ad libitum*, kept and maintained under standard laboratory conditions. Animals in group 1 were the normal control, Animals in groups 2-4 were treated with a single dose of Ibuprofen 200 mg/kg. Groups 3 and 4 were treated with Aloe vera leaf gel extract at 200 and 400 mg/kg respectively for 14 days, group 5 animals were pre-treated with oral administration of Aloe vera gel extract for 14 days at a concentration of 200 mg/kg before the ulcerogenic procedures, Group 6 was treated with standard medication (Omeprazole) after inducing ulcer, while Group 2 animals were left untreated and sacrificed immediately. Gastric ulceration, was induced in 18 hours fasted rats by the oral administration of a ulcerogenic drug, Ibuprofen 200 mg/kg.

At the end of the experimental period the rats were sacrificed under 50mg/kg of ketamine anesthesia administration. The abdomen was

opened by making a central abdominal incision after fastening the rats to the dissection board with board pins. The stomach was identified, accessed and removed; the stomach was then opened along the greater curvature. Ulcer index was evaluated according to severity and ulcer scores.

Experimental Model

Male Wistar rats weighing about 180-200 g were divided into six groups each comprising 5 rats each, as detailed below.

Representative photomicrographs of the Stomach lining of different groups of ibuprofen-induced ulcer in adult male Wistar rats treated with Aloe vera gel extract stained with Haematoxylin and Eosin. (Scale bar = 300µm). Control group 1 showed normal histoarchitectural layout of mucosa (Green arrow) with normal intact epithelial lining (red arrow), the glands are normally arranged longitudinally in lamina propria, submucosa beneath (light blue arrow) and serosa. Group 2 is ulcer group showing serious sloughing of the gastric epithelial lining, altered arrangements of glandular tissues in the mucosal region and eventual gastric cell death. There are also in group 2, areas of acute inflammatory cells aggregation with spots of hemorrhages and vascular congestions in the altered mucosal layer (black arrows). Group 3 is ulcer group treated with low dose of aloe vera leaf gel, the section showed severe sloughing with mild hemorrhagic areas, with few white blood cell infiltration of the mucosal layer (black arrow). Group 4 is ulcer group treated with high dose aloe vera leaf gel, the section showed mild sloughing of gastric epithelium (deep blue arrow), no necrosis was observed but there are few areas of petechial hemorrhages with intravascular congestion (black arrows), nil leucocytic inflammatory cell was seen in the mucosa. Group 5 is the Aloe vera pre-treated group with low dose Aloe vera leaf gel, the histoarchitecture of this group revealed normal intact layers all through the mucosal epithelial lining, intact submucosal layer and muscularis. Group 6 is ulcer group treated with standard medication (Omeprazole), the section showed mild sloughing of gastric epithelium, no necrosis was observed, no areas of petechial hemorrhages or intravascular congestion.

Representative photomicrographs of the Stomach lining of different groups of ibuprofen-induced ulcer in adult male Wistar rats treated with *Aloe vera* gel extract stained with Periodic Acid Schiff (Scale bar = 300µm).

Control group 1 is the normal control group which showed glycogen deposition along the mucosal layer (blue arrow), uniformly

GRP	(n)GROUPS	TREATMENTS	ROUTES	PERIOD
1	5	Control rats (1 mL/kg of distilled water)	Oral	14 days
2	5	(200mg) Ibuprofen induced-ulcer group	Oral	14 days
3	5	Ulcer + 200 mg/kg Aloe vera group	Oral	14 days
4	5	Ulcer + 400 mg/kg Aloe vera group	Oral	14 days
5	5	200 mg Aloe vera + Ulcer	Oral	14 days
6	5	Ulcer + 20mg/kg Omeprazole	Oral	14 days

Plate 1

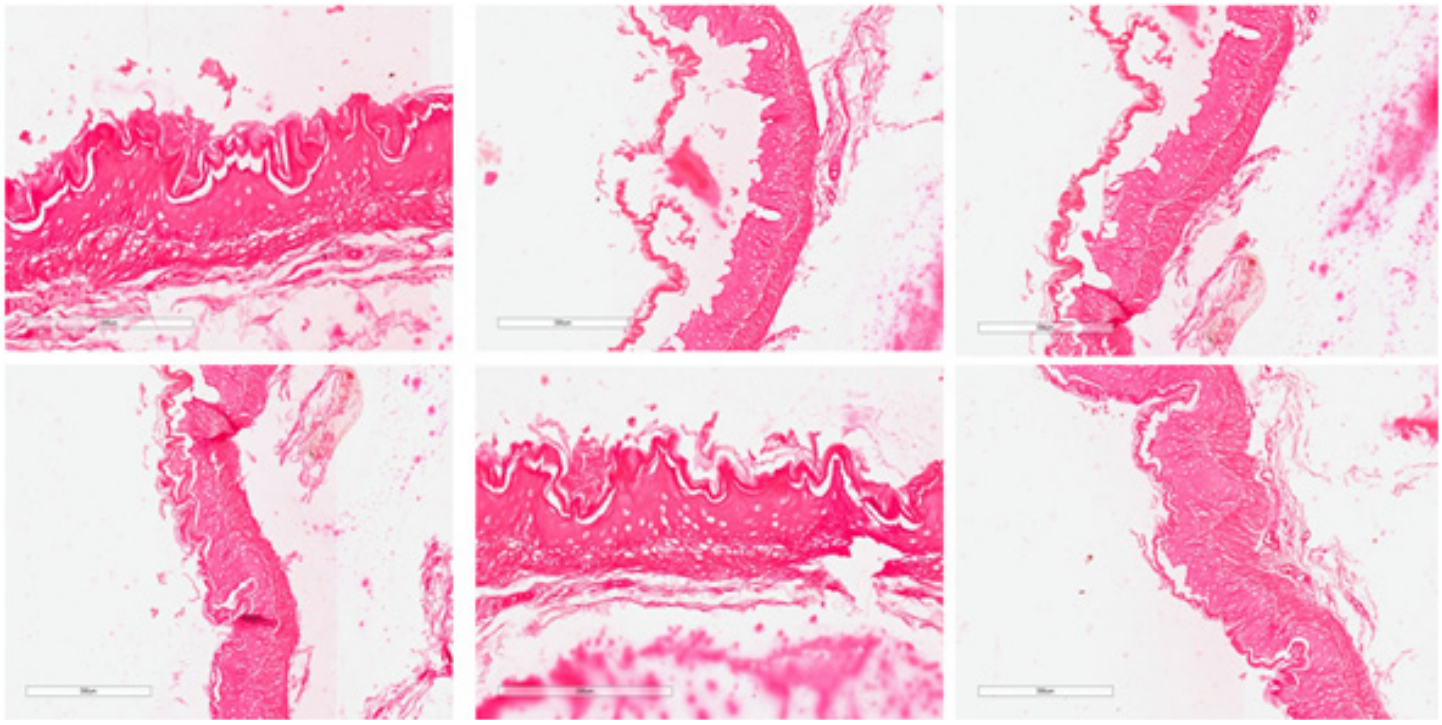


Plate 2

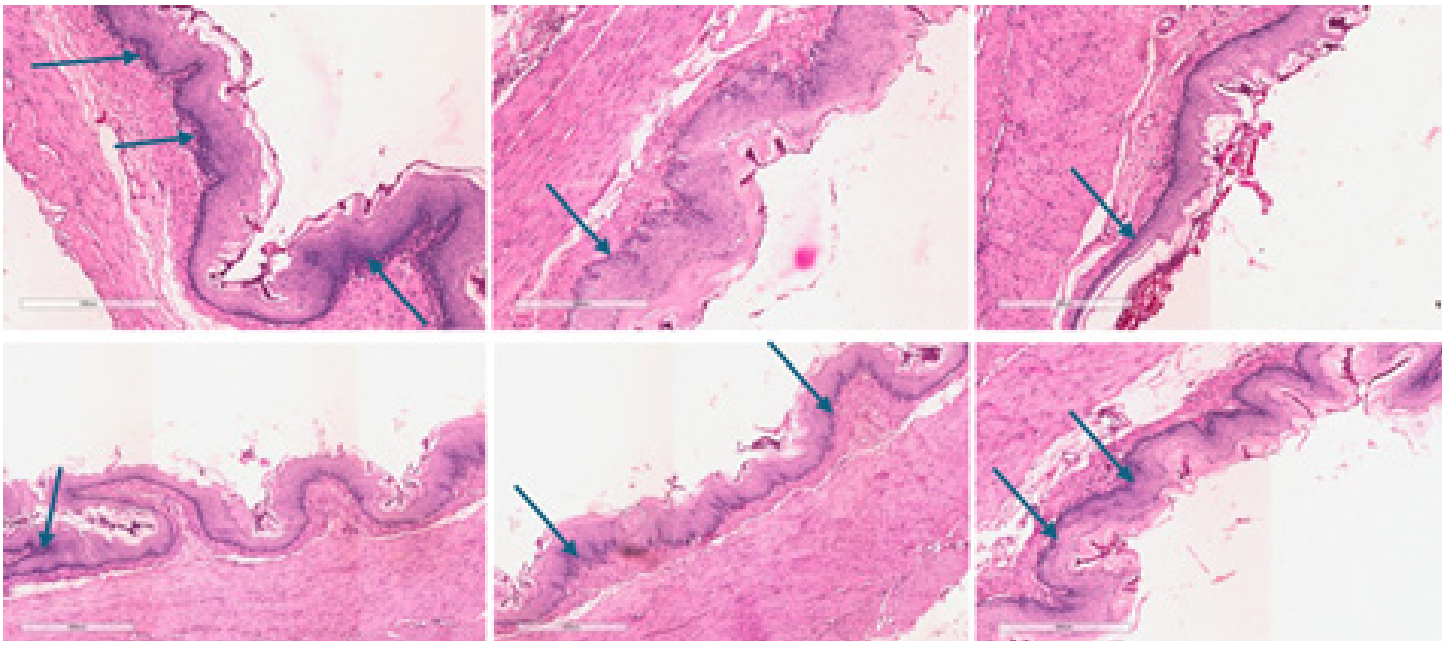
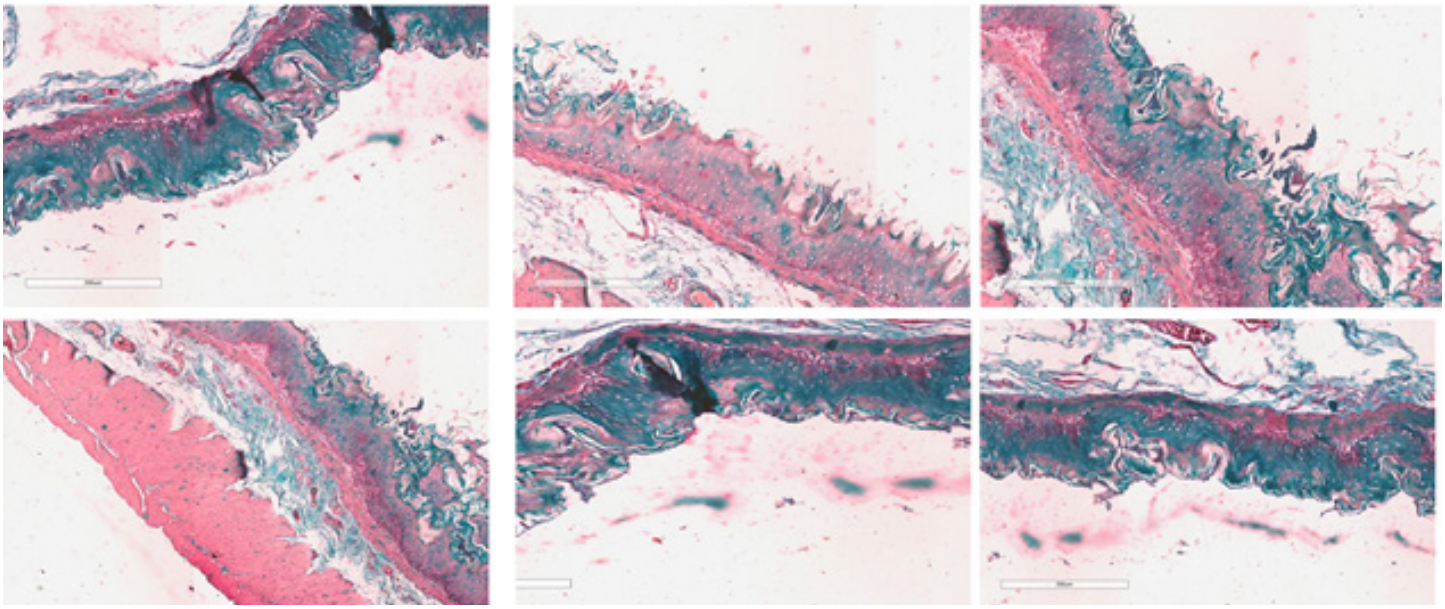


Plate 3



distributed in all the layers of the stomach (Blue arrow) with intact histoarchitectural lining. Group 2 and 3 are ulcer groups showing marked reduction in glycogen deposition of the gastric epithelial lining, which is in response to the injury caused by the ulcerogenic agent. Group 4,5 and 6 are ulcer group treated with high dose aloe vera leaf gel, the section showed reduced distribution in glycogen content as shown in blue arrows.

Representative photomicrographs of the Stomach lining of different groups of ibuprofen-induced ulcer in adult male Wistar rats treated with *Aloe vera* gel extract stained with Masson Trichrome (Scale bar = 300µm).

Control group 1 showed normal collagen fibre distribution in all the layers of the stomach (Green) with intact structural lining. Group 2 is ulcer group showing sloughing of the gastric epithelial lining and sparse collagen fibre distribution more gradually arranging at the base and edges of the damaged tissue, in response to wound repair process. Group 3 is ulcer group treated with low dose of aloe vera leaf gel, the section showed more collagen fibre distribution when compared with group 2, but very much less than the normal control. Group 4 is ulcer group treated with high dose aloe vera leaf gel, the section showed scanty collagen fibre distribution when compared to normal control Group 5 is the Aloe vera pre-treated group with low dose Aloe vera leaf gel, the histoarchitecture of this group revealed almost normal collagen fibre distribution all through the

mucosal epithelial lining, submucosal layer and muscularis. Group 6 is ulcer group treated with standard medication (Omeprazole), the collagen distribution in this group showed normal pattern which is nearly similar to the normal control group.

Discussion

The results from this study showed that Ibuprofen actually induced gastric ulceration at 200mg/kg in the Wistar rats as evidenced by sloughing of the epithelial lining of the mucosa layer, this is similar to the work of Sohail et al., [30], which stated that NSAIDs usage is associated with gastrointestinal tract side effects due the inhibition of both cyclooxygenase (COX) -1 and COX-2 enzymes leading to a decrease in gastroprotective prostaglandins (PG). The low dose and high dose Aloe vera gel extract offer healing effects on the injured gastric mucosa as evidenced by reduction to mild sloughing of the epithelial mucosa injury from very severe sloughing of non-treated group, which has similar effect as the group treated with standard drug (Omeprazole), this finding is in keeping with the work done by Mohammed et al., [31], which posited that aloe vera exerted antiulcer activities through modulation of antioxidant/oxidant status including its antisecretory properties with mitigation of pyroptosis.

Moreover, the prophylactic effect of Aloe vera leaf gel extract has more profound protective influence on the gastric epithelial mucosa of the ulcer-induced Wistar rats than the therapeutic effect, this corroborates the research carried out by Eamlamnam et al., who stated that Aloe vera gel extract reduces leukocyte adherence and TNF-α level, elevate IL-10 level and promote gastric ulcer healing, as observed in this study.

Conclusion

It can be concluded from this study that ibuprofen caused gastric ulceration in the Wistar rats, the Aloe vera gel extract at varying doses including Omeprazole were able to ameliorate the ulcerogenic effect caused by Ibuprofen, however the prophylactic effect of Aloe vera is more efficacious than its therapeutic effect.

Reference

1. Hall John E; Guyton Arthur C. Guyton and Hall text book of medical physiology (12th ed). Philadelphia, Pa: Saunders/Elsevier. 2011; 777-779.
2. Moore Keith L, Dalley Arthur F, Agur AMR. Clinically oriented anatomy (Eighth ed). Philadelphia Baltimore New York London Buenos Aires Hong Kong Sydney Tokyo: Wolters Kluwer. 2018; 454.
3. Badapanda SS, Kaur A, Jain D, et al. Epidemiology, Pathogenesis, and Induction of Peptic Ulcer: A Comprehensive review. New Emirates Medical Journal. 2024; 5.
4. Azer SA, Awosika AO, Akhondi H. Gastritis. Stat Pearls Publishing. 2024.
5. Halpert RD. Gastricerosions and ulcers. In Gastrointestinal Imaging (3rd ed). Elsevier. 2006.
6. Woolf A, Rose R. Gastriculcer. StatPearls Publishing. 2023.
7. Sigmon DF, Tuma F, Kamel BG, et al. Gastric perforation. StatPearls Publishing. 2023.
8. Jadhav S, Anwar K, Khan A, et al. Circumferential Pyloric Ulcer Inducing Gastric Outlet Obstruction: A Report of a Rare Case. Cureus. 2024; 16: e71268.
9. Almadi MA, Lu Y, Alali AA, et al. Peptic ulcer disease. The Lancet. 2024; 404: 68-81.
10. Vimala G, Shoba FG. A review on antiulcer activity of few Indian medicinal plants. Int J Microbiol. 2014; 2014: 1-14.
11. Ren J, Jin X, Li J, et al. The global burden of peptic ulcer disease in 204 countries and territories from 1990 to 2019: a systematic analysis for the Global Burden of Disease Study 2019. Int J Epidemiol. 2022; 51: 1666-1676.
12. Pujari NM, Khushtar M, Mishra A, et al. Comprehensive insights into gastric ulcer therapies: a review of current Approaches. JAPSR. 2024; 7: 4-12.
13. Proctor MJ, Deans C. Complications of peptic ulcers. Surgery (Oxford). 2014; 32: 599-607.
14. Huang C, Lai W, Sun M, et al. Prescription patterns of traditional Chinese medicine for peptic ulcer disease inTaiwan: A nationwide population-based study. J Ethnopharmacol. 2015; 176: 311-320.
15. Abdu SM, Assefa EM, Abdu H. Prevalence and patterns of peptic ulcer disease in Africa: a systematic review and meta-analysis. BMC Gastroenterol. 2025; 25: 298.
16. Scarpignato C, Hunt RH. Nonsteroidal Anti-inflammatory Drug-Related Injury to the Gastrointestinal Tract: Clinical Picture, Pathogenesis, and Prevention. Gastroenterol Clin N Am. 2010; 39: 433-464.
17. Chavez ML, DeKorte CJ. Valdecoxib: A review. Clin Ther. 2003; 25: 817-851.
18. Wahbi AA, Hassan E, Hamdy D, et al. Spectrophotometric methods for the determination of Ibuprofen in tablets. Pak J Pharm Sci. 2005; 18: 1-6.
19. Ainyanbhor IE, Edo GI, Ali AB, et al. Ibuprofen: A review on its synthesis, mechanism of action, pharmacological properties, and environmental impact. Pharmacological Research Reports. 2025; 4: 100066.
20. Varrassi G, Pergolizzi JV, Dowling P, et al. Ibuprofen Safety at the Golden Anniversary: Are all NSAIDs the Same? A Narrative Review. Adv Ther. 2019; 37: 61-82.
21. Athiyyah AF, Darma A, Sumitro KR, et al. Comparative Analysis of Gastric Mucosal damage in Wistar Rats Subjected to Varying Doses of Ibuprofen. RJPT. 2025; 3530-3536.
22. Tai FWD, McAlindon ME. Non-steroidal anti-inflammatory drugs and the gastrointestinal tract. Clin Med (Lond). 2021; 21: 131-134.
23. Zhang O, Xu Z. Research progress on omeprazole in the treatment of peptic ulcers. MEDS Clinical Medicine. 2024; 5.
24. Al Ali HS, Jabbar AS, Neamah NF, et al. Long-Term Use of Omeprazole: Effect on Haematological and Biochemical

Parameters. Acta Med Indones. 2022; 54: 585-594.

25. Keshavarzi Z, Rezapour TM, Vatanchian M, et al. The effects of aqueous extract of Aloe vera leaves on the gastric acid secretion and brain and intestinal water content following acetic acid- induced gastric ulcer in male rats. Avicenna J Phytomed. 2014; 4: 137-143.
26. Kurniawan D, Li L, Liu M, et al. Chloroplast genomes of six Aloe species: Insights into genetic variation and evolutionary patterns. Plant Gene. 2025; 43: 100530.
27. Elferjane MR, Jovanović AA, Milutinović V, et al. From Aloe vera Leaf Waste to the Extracts with Biological Potential: Optimization of the Extractions, Physicochemical Characterization, and Biological Activities. Plants (Basel). 2023; 12: 2744.
28. Sharma P, Kaur R. A Comprehensive Review of Aloe vera: Composition, Properties, Processing, and Applications. The Natural Products Journal. 2025; 16.
29. Kumar S, Kumar S, Kalita S, et al. Recent advances in therapeutic and biological activities of Aloe vera. Biocatalysis and Agricultural Biotechnology. 2024; 57: 103084.
30. Sohail R, Mattew M, Patel KK, et al. Effects of Non-Steroidal Anti-inflammatory Drugs (NSAIDS) and Gastroprotective NSAIDs on the Gastrointestinal Tract: A narrative Review. Cureus. 2023; 15: e37080.
31. Mohammed AO, Abd-Elghaffar SK, Mousa RA, et al. Aloe vera gel confers therapeutic effect by reducing pyroptosis in ethanol-induced gastric ulcer rat model: Role of NLRP3/GSDMD signaling pathway. Mol Biol Rep. 2024; 51: 401.