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Research Article

EVALUATION OF ANTIULCER AND HEPATOPROTECTIVE ACTIVITY OF *ALOE VERA* AND TURMERIC ORAL SUSPENSION IN WISTAR RATS

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ABSTRACT

Peptic ulcer disease (PUD) and drug-induced hepatotoxicity are significant global health problems associated with oxidative stress, inflammation, and cellular injury. PUD develops due to disruption of the gastrointestinal mucosal barrier, leading to ulceration mainly in the stomach and duodenum. Major causative factors include *Helicobacter pylori* infection, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol intake, and stress. The disease results from an imbalance between aggressive factors such as gastric acid, pepsin, and reactive oxygen species and protective factors including mucus secretion, bicarbonate, and mucosal blood flow. NSAIDs, particularly indomethacin, inhibit prostaglandin synthesis through COX-1 blockade, causing mucosal damage and ulcer formation. In experimental studies, indomethacin at 20 mg/kg reliably induces gastric ulcers in animal models. NSAIDs are also implicated in hepatic injury through the formation of reactive metabolites that induce oxidative stress and mitochondrial dysfunction. Drug-induced hepatotoxicity contributes significantly to acute liver failure and is characterized by elevated liver enzymes (AST, ALT, ALP, GGTP), increased bilirubin levels, and reduced protein and albumin concentrations. Histopathological changes include centrilobular necrosis, inflammatory infiltration, and hepatocellular degeneration. Natural products such as *Aloe vera* and Turmeric possess antioxidant and anti-inflammatory properties that may protect against gastric and hepatic damage. Bioactive compounds including acemannan, curcumin, and flavonoids exhibit cytoprotective and hepatoprotective effects by scavenging free radicals and stabilizing cellular membranes. This study aims to evaluate the antiulcer and hepatoprotective potential of an *Aloe vera*–Turmeric oral suspension in indomethacin-induced Wistar rat models using biochemical and histopathological parameters to determine its therapeutic efficacy.

Keywords: Antiulcer, Hepatoprotective, *Aloe vera*, Turmeric, Indomethacin, NSAIDs, Oral Suspension.

INTRODUCTION

Peptic ulcer disease (PUD) and drug-induced hepatotoxicity remain two significant health problems worldwide due to their high prevalence and common underlying mechanisms involving oxidative stress and inflammation. (Malik *et al*., 2026) Peptic ulcers are characterized by localized erosions in the mucosal lining of the stomach or duodenum, resulting from an imbalance between aggressive factors such as gastric acid, pepsin, and reactive oxygen species (ROS), and defensive factors including mucus secretion, bicarbonate production, and mucosal blood flow. Approximately 10% of the global population is affected by this disorder, with the majority of cases occurring in individuals aged 20–60 years. (Kuna *et al*., 2019). The chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as indomethacin, stress, alcohol intake, and Helicobacter pylori infection is recognized as key risk factors. NSAIDs cause gastric mucosal injury primarily by inhibiting cyclooxygenase-1 (COX-1) enzyme activity, reducing prostaglandin synthesis, impairing mucosal regeneration, and diminishing local blood flow. (Ko & Lee, 2025), (Bjamason *et al*., 2018) In laboratory models, oral administration of indomethacin at 20 mg/kg induces gastric lesions, producing an ulcer index value ranging between 30–40,

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confirming its reproducible ulcerogenic potential. (Sabmu *et al*., 2015).

Liver toxicity is another major adverse effect of NSAID therapy. The liver plays a vital role in detoxification, drug metabolism, and homeostasis, and is therefore susceptible to injury from xenobiotics. (Allison *et al*., 2023) Drug-induced hepatotoxicity accounts for nearly 20–40% of fulminant hepatic failure cases and is often associated with increased serum levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGTP), and bilirubin, accompanied by reduced albumin and total protein concentrations. (Thakur *et al*., 2024), (David & Hamilton, 2010) According to clinical standards, enzyme levels exceeding twofold above the upper limit of normal (2× ULN) are considered indicative of hepatic injury. Histopathological findings in hepatotoxic models typically reveal centrilobular necrosis, inflammatory cell infiltration, and vacuolar degeneration, confirming tissue damage. (Kalas *et al*., 2021).

To counteract these effects, natural products possessing antioxidant, anti-inflammatory, and cytoprotective properties have gained importance. (Ali *et al*., 2018) *Aloe vera* (Aloe barbadensis Miller) and Curcuma longa (turmeric) are traditional medicinal plants widely known for their antiulcer and hepatoprotective potential. *Aloe vera* contains biologically active compounds such as acemannan and anthraquinones, which enhance mucosal protection, reduce oxidative stress, and promote epithelial healing. (Sánchez *et al*., 2020) Curcuma longa contains curcumin, a polyphenolic compound that modulates inflammatory cytokines, scavenges free radicals, and stabilizes hepatocyte membranes. (Rum de Porras *et al*., 2023).

Several experimental studies have demonstrated that combination formulations of these plants significantly lower ulcer index scores, (Gupta *et al*., 2021) normalize serum enzyme levels, and restore histological integrity of gastric and hepatic tissues. (Raghavendra & Rao, 2000) The integration of biochemical markers (AST, ALT, ALP, GGTP, bilirubin, protein, albumin) with histological evaluation provides a comprehensive understanding of the therapeutic response. (Thakur *et al*., 2024). The present study aims to evaluate the antiulcer and hepatoprotective efficacy of a formulated *Aloe vera* and *Turmeric* oral suspension in Wistar rats using an indomethacin-induced ulcer and hepatotoxicity model**.** The study focuses on quantitative and histopathological parameters to determine the degree of protection and healing offered by the formulation in comparison with standard treatment.

MATERIALS AND METHODS

Preparation of Extracts and Formulation

Aloe vera gel powder and turmeric powder were extracted using methanol and ethanol solvents, respectively. (Stmhane *et al*., 2022). The oral suspension was formulated with 10 g *Aloe vera* gel powder, 5 g turmeric powder, 20 ml glycerol, sodium benzoate as preservative, and distilled water up to 200 ml, ensuring suitable pH (5.5), viscosity (180 cP), sedimentation volume (0.3), and re-dispersibility. The formulation was stable for up to 14 days under refrigeration.

Animal model and Experimental Design

Male Wistar rats (150-200 g) were divided into five groups (n=6): normal control, disease control (indomethacin only), standard control (N-acetyl cysteine 400 mg/kg), test low dose (100 mg/kg *Aloe vera* + 50 mg/kg turmeric), and test high dose (200 mg/kg *Aloe vera* + 100 mg/kg turmeric). Indomethacin was administered orally at 20 mg/kg for 5 days to induce ulcers and hepatotoxicity. (Bagheri *et al*., 2018). Treatments were administered daily by oral gavage for 21 days.

Evaluation Parameters

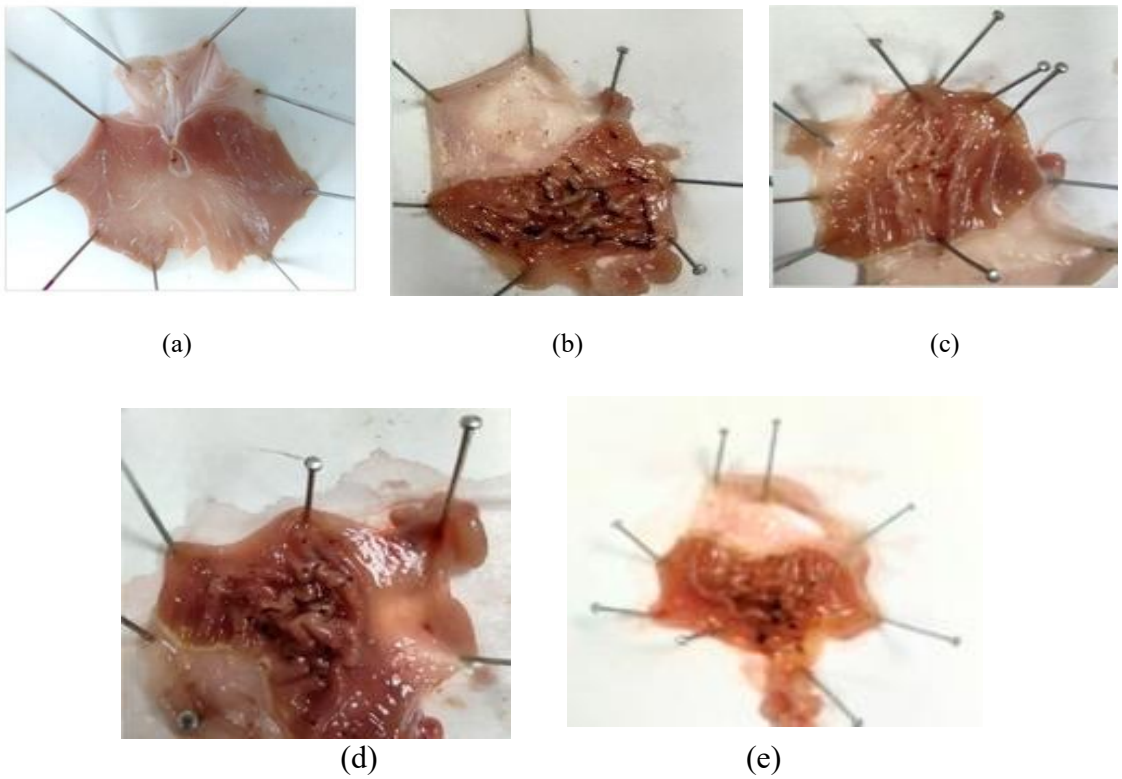
Ulcer index and ulcer protection percentage based on macroscopic gastric examination using a standardized scoring system (0-3 scale). Serum biochemical parameters: AST, ALT, ALP, GGTP, total bilirubin, albumin, and total protein measured by commercial diagnostic kits. Percentage hepatoprotection calculated relative to disease and normal controls. Histopathological examination of gastric and liver tissues stained with hematoxylin and eosin. Statistical analysis by one-way ANOVA with Tukey's post hoc test; $p < 0.01$ considered significant.

RESULTS AND DISCUSSION

Results of ulcer healing activity of *Aloe vera* and Turmeric Oral Suspension. Using experimental ulcers caused by Indomethacin (IND), this study investigated the anti-ulcer effects of N-acetyl cysteine (400 mg/kg as a reference medication) and different dosages of the prepared *Aloe vera* and turmeric oral suspension (*Aloe vera*, 100 mg/kg, turmeric 50 mg/kg low dose) (high dose *Aloe vera* 200 mg/kg, turmeric 100 mg/kg). The disease control group showed severe gastric mucosal damage with high ulcer scores and indices. Both test doses significantly reduced ulcer scores ($p < 0.01$), with the high dose achieving 87.6% protection comparable to N-acetyl cysteine (91.5%). Macroscopic and histopathological analyses confirmed mucosal restoration and reduced inflammation in treated groups. Indomethacin caused significant elevation of liver enzymes and bilirubin and reduced albumin and total protein, indicating hepatotoxicity. Both doses of the suspension reduced enzyme levels and restored serum proteins dose-dependently ($p < 0.01$), with the high dose nearing standard drug efficacy. Percentage hepatoprotection for the high dose ranged from 84.47% to 88.42% across parameters. Assessment of Liver Function Parameters Based on Serum Biochemical Markers in Different Experimental groups. All the data are presented as mean ± SEM (n=5) (one-way ANOVA followed by Tukey's post hoc test).

Figure 1. Rat gastric mucosa under macroscopic inspection in various treatment groups. (a) normal control group, (b) disease control group (c) standard control group (d) test control -1 and (e) test control-2.

Group	Ulcer Score (Mean ± SEM)	Ulcer Index	Ulcer Protection (%)
Normal Control	0	0	-
Disease Control	17.2 ± 1.15	37.1	-
Standard Control	3.2 ± 1.31	3.14	91.5
Test Low Dose	5.8 ± 1.49	8.8	76.0
Test High Dose	4.6 ± 1.02	4.58	87.6



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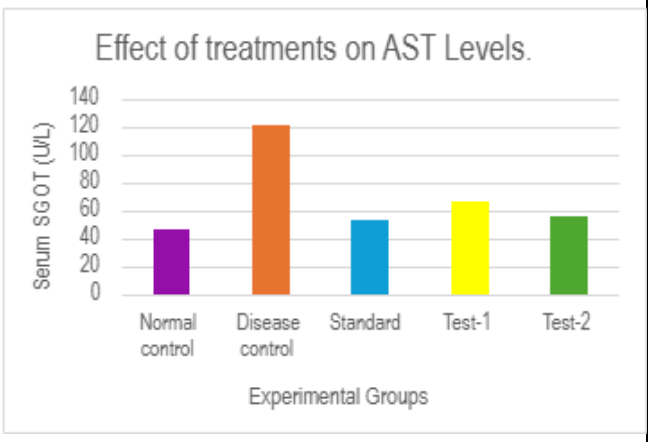


Figure 2. Effect of treatment on serum levels in Hepatotoxic Rats.

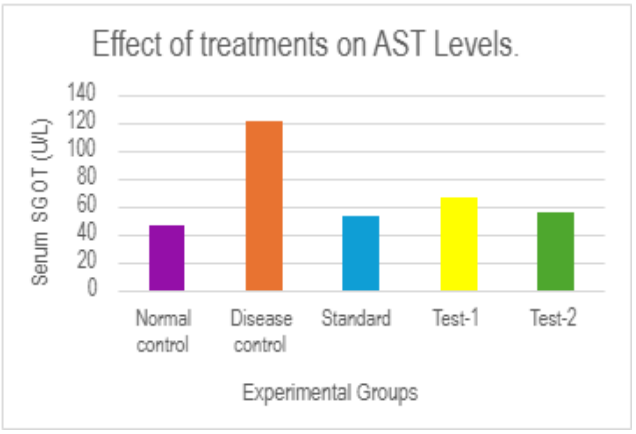


Figure 3. Effect of treatment on serum ALT AST (SGOT) (SGPT) levels in Hepatotoxic Rats

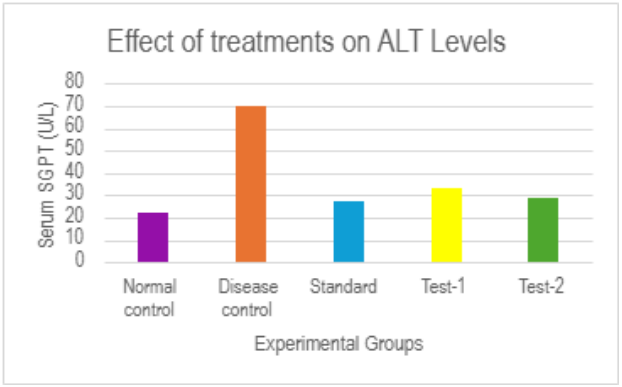


Figure 4. Effect of treatment on serum ALP Hepatotoxic Rats

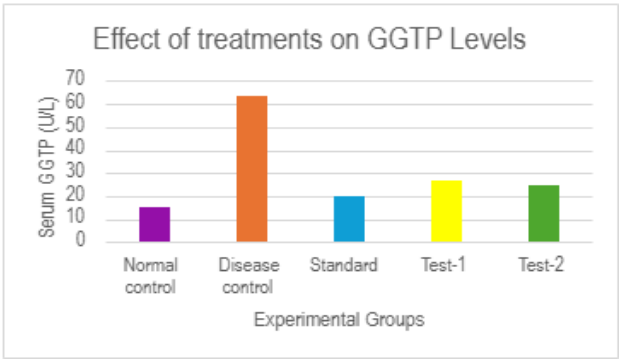


Figure 5. Effect of treatment on serum levels in GGTP levels in Hepatotoxic Rats.

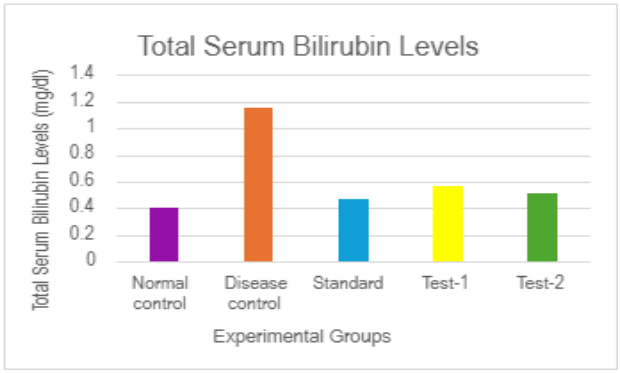


Figure 6. Effect of treatment on serum levels in Hepatotoxic Rats.

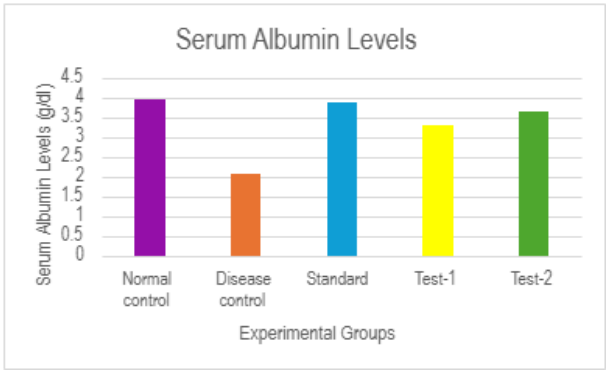


Figure 7. Effect of treatment on serum Bilirubin Albumin levels in Hepatotoxic Rats.

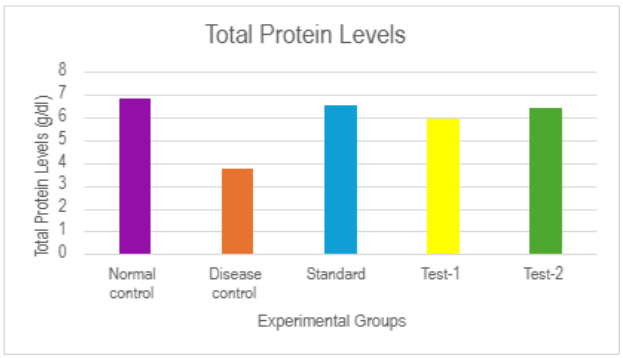


Figure 8. Effect of treatment on total protein levels in Hepatotoxic Rats.

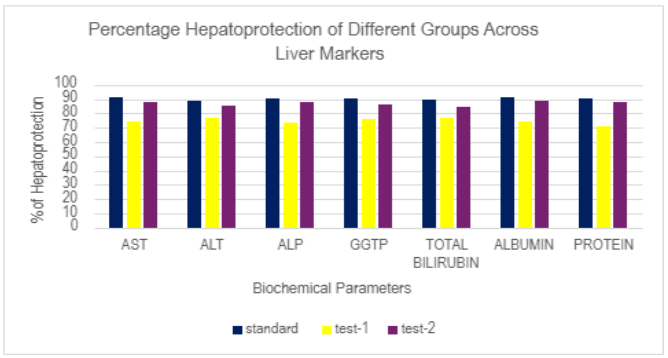
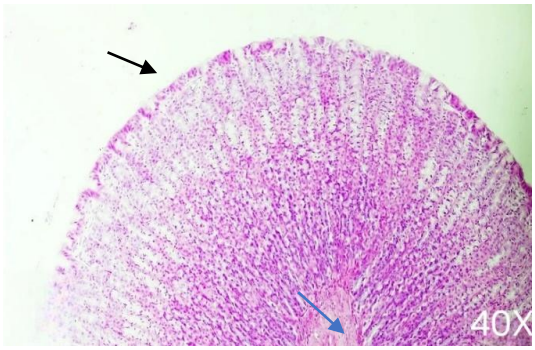


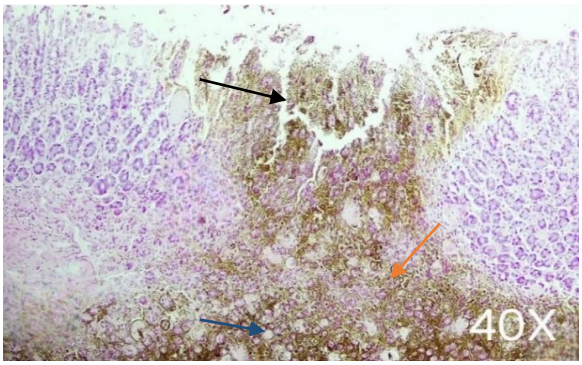
Figure 9. Percentage Hepatoprotection of standard and test groups based on biochemical markers in experimental rats.

Tissue sections of stomach and liver from the high dose group showed marked tissue integrity with reduced necrosis and inflammation, corroborating biochemical results. The damage was extensive in the disease control but was reversed significantly by test and standard treatments.



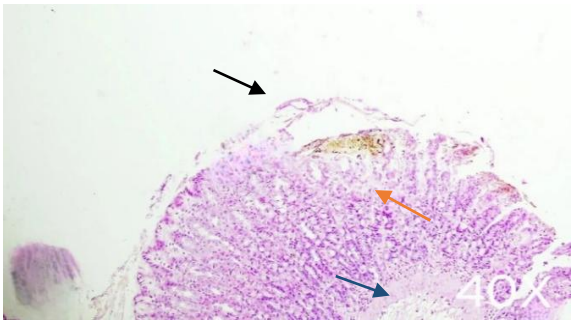
The mucosal layer (black arrow) is intact, displaying well-organized gastric glands with no signs of necrosis, inflammation, or erosion. The epithelial lining is continuous, and the muscularis mucosae (blue arrow) appear unaltered.

Figure10. Histopathology of normal control group gastric mucosa (H&E stain, 40X magnification).



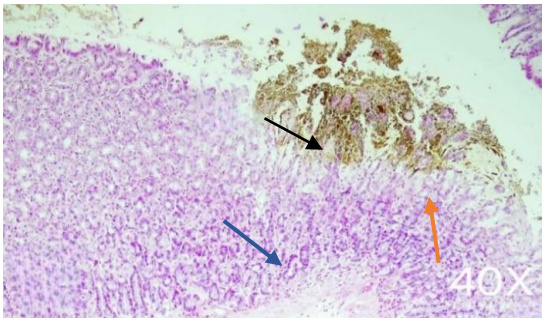
It shows extensive mucosal damage**,** including erosion of the epithelial lining (black arrow). Necrosis and infiltration of inflammatory cells are evident in the mucosal layer (orange arrow). The glandular architecture is disrupted, edema and hemorrhage in the submucosa (blue arrow).

Figure 11. Histopathology of disease control group gastric mucosa (H&E stain, 40x magnification).



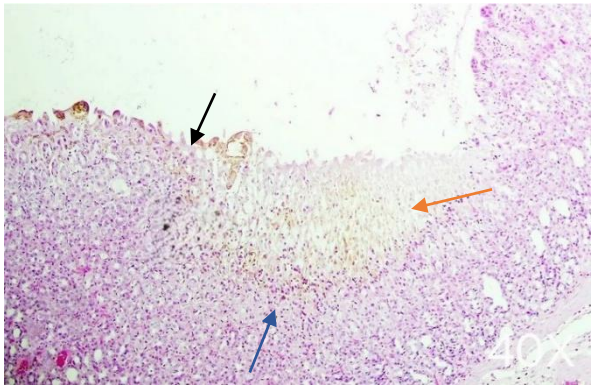
The epithelial lining (black arrow) appears more intact, indicating mucosal recovery. Minimal necrosis and inflammatory infiltration are visible (orange arrow), showing a moderate degree of healing. The architecture of the gastric glands in muscularis mucosae (blue arrow) was preserved with only mild disruption. This group shows moderate degree of ulcer inhibition and mucosal recovery compared to disease control group.

Figure 12. Histopathology of standard control group gastric mucosa (H&E stain, 40x magnification).



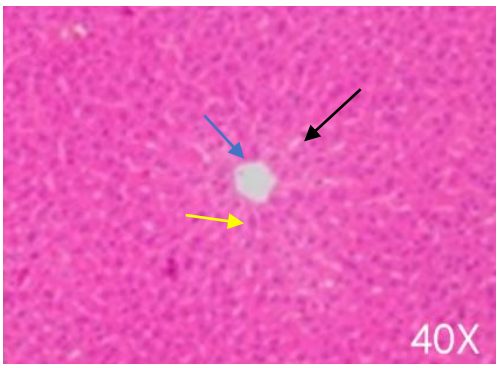
The mucosal surface showed regions of epithelial loss (black arrow), and some infiltration of inflammatory cells and presence of hemorrhagic zones in the upper mucosa (orange arrow) was reflecting localized tissue injury. The glandular structure in the lower mucosa remains largely intact (blue arrow) indicating preservation of tissue architecture. These feature shows mild antiulcer activity at a low dose.

Figure13. Histopathology of test control-1 group gastric mucosa (H&E stain, 40x magnification).



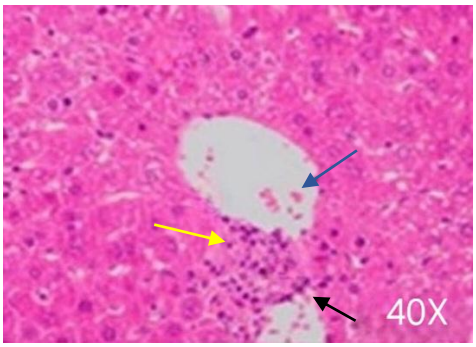
A nearly intact epithelial layer (black arrow), minimal inflammatory infiltration in the submucosa (orange arrow), and restored glandular morphology (blue arrow).as compared to the disease control group, tissue damage was markedly reduced, indicating moderate antiulcer activity at high dose and supporting a dose - dependent gastroprotective effect of the formulation.

Figure 14. Histopathology of test control-2 group gastric mucosa (H&E stain, 40x magnification).



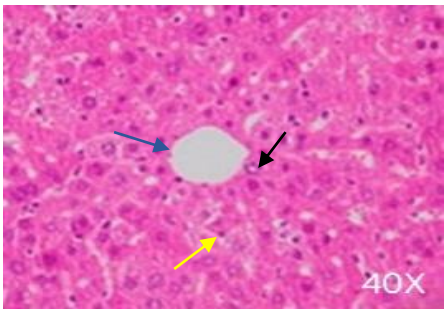
The histological architecture appears normal with well-preserved hepatic lobules, (yellow arrow) central vein, (blue arrow) and radiating hepatic cords (black arrow). Hepatocytes are polygonal with centrally located nuclei, and there is no evidence of necrosis, inflammation, or cellular degeneration.

Figure 15. Histopathology of Normal Control group Liver Tissue (H&E, 40X).



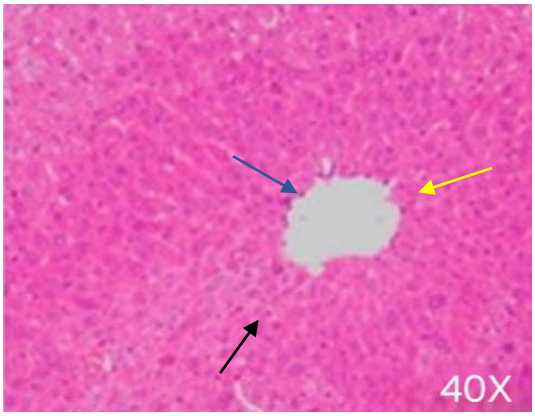
This section shows a prominent centrilobular necrosis with surrounding ballooned hepatocytes (blue arrow). There is intense inflammatory cell infiltration (yellow arrow), indicating ongoing tissue injury. The hepatic cords appear disorganized (black arrow), suggesting architecture disruption. These features indicate severe hepatotoxic damage.

Figure 16. Histopathology of Disease Control group Liver Tissue (H&E, 40X).



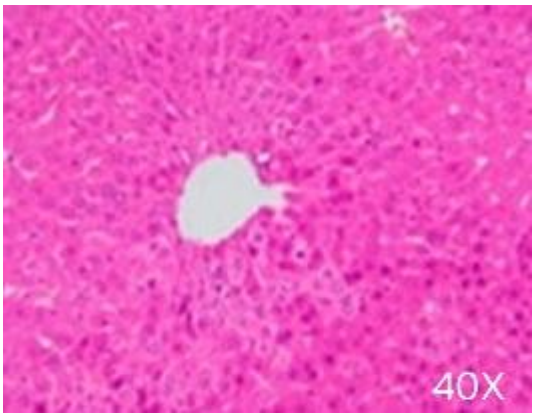
Liver histoarchitecture appears to be largely preserved. Hepatocytes show minimal vacuolar degeneration (black arrow), and the central vein is distinct (blue arrow). Mild inflammation (yellow arrow) may be present, but the overall structure indicates significant recovery from hepatic injury. This suggests the hepatoprotective potential of the standard drug (N- Acetyl Cysteine).

Figure 17. Histopathology of Standard Control group Liver Tissue (H&E, 40X).



The Hepatic tissue reveals moderate restoration of cellular architecture. The central vein (blue arrow) is visible with surrounding hepatocytes showing mild degeneration (yellow arrow) and reduced necrotic features (black arrow), compared to the disease group. The test formulation at low dose appears to exert a moderate hepatoprotective effect.

Figure18. Histopathology of Test Control-1 group Liver Tissue (H&E, 40X).



This liver tissue shows near-normal liver histology. The hepatic cords (black arrow) are well-aligned, with minimal cellular damage (yellow arrow) and clear central vein (blue arrow). The test formulation at high dose demonstrates a pronounced hepatoprotective effect, comparable to the disease control group.

Figure 19. Histopathology of Test Control-2 group Liver Tissue (H&E, 40X).

The present study demonstrated that the *Aloe vera* and Curcuma longa (turmeric) oral suspension provides significant protection against indomethacin-induced gastric ulceration and hepatotoxicity in Wistar rats. (Rahman *et al*., 2024), (Werawatganon, 2014) The observed antiulcer and hepatoprotective effects were dose-dependent and comparable to the standard drug, N-acetylcysteine, particularly at the higher dose. (Sukumaran *et al*., 2023). These findings suggest that the combination of *Aloe vera* and turmeric exerts synergistic pharmacological activity through the modulation of oxidative stress, inflammation, and tissue repair mechanisms. Indomethacin-induced gastric mucosal injury is well-established to occur via suppression of COX-1-mediated prostaglandin synthesis, increased gastric acid secretion, and generation of reactive oxygen species. (Funatsu *et al*., 2007) In the present study, the disease control group exhibited extensive mucosal erosion, haemorrhagic streaks, and high ulcer scores, indicating successful establishment of the experimental model. (Iqbal *et al*., 2025). Administration of the polyherbal suspension markedly decreased ulcer index and increased ulcer protection percentage, indicating strong mucosal defense. The higher-dose formulation afforded 87.6% ulcer inhibition, closely comparable to N-acetylcysteine (91.5%). This could be attributed to polysaccharides and anthraquinones present in *Aloe vera* which enhance mucus production, stimulate epithelial regeneration, and improve local microcirculation. (Cordiano *et al*., 2025). Simultaneously, curcumin from turmeric is known to inhibit NF-κB activation, suppress pro-inflammatory cytokines such as TNF-α and IL-6, and scavenge reactive oxygen species, thereby limiting oxidative mucosal injury. (Iweala *et al*., 2023).

The hepatoprotective findings of the study further validate the broad therapeutic potential of the formulation. Indomethacin-mediated hepatic injury was characterized by significant elevation of AST, ALT, ALP, GGTP, and bilirubin, along with decreased albumin and total protein levels. (Khan *et al*., 2019) Such alterations arise due to mitochondrial dysfunction, lipid peroxidation, and hepatocellular membrane disruption. (Ahmad *et al*., 2018) Treatment with the herbal suspension restored serum biochemical markers toward normal levels, with the high dose showing 84–88% hepatoprotection across parameters.

These improvements were supported histologically by reduced necrosis, preserved hepatic cords, and minimal inflammatory infiltration in treated groups. The hepatoprotective action could be attributed to the antioxidant capacity of curcumin and acemannan, stabilization of hepatocyte membranes, and enhancement of glutathione levels. (Khudair & Al-Gareeb, 2024).

The findings of this study are consistent with earlier reports demonstrating the protective role of *Aloe vera* gel in gastric healing and *Curcuma longa* in preventing hepatic injury. However, the present investigation is distinctive in evaluating a combined formulation in a dual organ-damage model, targeting both gastric and hepatic systems simultaneously, which mirrors real-world NSAID-induced toxicity. The synergistic interaction between *Aloe vera* and turmeric likely offers superior efficacy compared to their individual administration, as evidenced by improved biochemical and histopathological outcomes. Furthermore, the safety of the formulation was evident, as no signs of toxicity or adverse reactions were observed in the treated animals during the experimental period. This supports its potential application as a complementary or alternative therapeutic strategy for long-term NSAID users. Nevertheless, the study is limited to animal models; future investigations should focus on molecular pathway analyses, pharmacokinetics, and clinical validation in human subjects to confirm translational relevance.

CONCLUSION

The present study demonstrates that the *Aloe vera* and turmeric oral suspension exerts significant antiulcer and hepatoprotective effects against indomethacin-induced gastric and hepatic injury in Wistar rats. The formulation effectively reduced ulcer index, normalized liver function biomarkers, and restored tissue architecture in a dose-dependent manner. The higher dose exhibited therapeutic efficacy comparable to the standard drug N-acetylcysteine, indicating strong protective activity. The gastroprotective and hepatoprotective effects can be attributed to the synergistic action of phytoconstituents such as acemannan and curcumin, which possess antioxidant, anti-inflammatory, cytoprotective, and membrane-stabilizing properties. Histopathological findings further confirmed the preservation of gastric mucosa and hepatic architecture in treated groups. These findings suggest that the *Aloe vera* and turmeric suspension may serve as a promising natural alternative or adjunct therapy for managing NSAID-induced gastropathy and hepatotoxicity. However, further studies involving molecular mechanisms, long-term safety evaluation, and clinical trials are necessary to establish its therapeutic potential in humans.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

The work is approved by the IAEC with approval number CCSEA/IAEC/JLS/22/10/24/021

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AI TOOL DECLARATION

The authors declares that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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