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Research Article

Evaluating the efficacy of antibiotic, anti-inflammatory, and monoclonal antibody therapies on gut pathology in a TNBS-induced rat model of Crohn's disease

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Abstract

Crohn's disease is a chronic inflammatory bowel disorder characterized by intestinal inflammation, oxidative stress, and mucosal injury. This study evaluated the therapeutic efficacy of antibiotic, anti-inflammatory, and monoclonal antibody treatments on gut inflammation, oxidative stress, and histopathological alterations in a trinitrobenzene sulfonic acid (TNBS)-induced rat model of Crohn's disease. Fifty adult male Sprague-Dawley rats (180-200 g) were randomly divided into five groups: control, Crohn's disease model, antibiotic (ciprofloxacin), anti-inflammatory (prednisolone), and monoclonal antibody (infliximab). TNBS administration markedly reduced gut pH (to ~1.5–2.1 vs. 4.0–4.6 in controls), increased total acidity (to ~450 mEq/L vs. 175 mEq/L), and elevated malondialdehyde levels (to ~0.17 nmol/mL vs. 0.07 nmol/mL), indicating oxidative stress and mucosal injury. Mucin content declined sharply (to ~60 µg/g vs. 160 µg/g in controls). Treatment with ciprofloxacin, prednisolone, and infliximab significantly restored mucin content (by approximately 25–30%) and gut pH, while reducing total acidity and lipid peroxidation compared with untreated disease controls with infliximab showing the greatest restoration of mucin (~110 µg/g) and reduction of lipid peroxidation (~0.06 nmol/mL). Histopathological analysis confirmed reduced ulceration, hyperemia, and megacolon severity in treated groups. These findings demonstrate that targeting intestinal microbiota, inflammation, and oxidative stress can improve gut integrity and function in experimental Crohn's disease. Further studies should explore combined therapeutic strategies to optimize treatment outcomes.

Keyword: Crohn's disease, antibiotic therapy, anti-inflammatory therapy, monoclonal antibody, gut pathology

1. INTRODUCTION

Crohn's disease is a chronic, relapsing, and remitting inflammatory bowel disease characterized by persistent gastrointestinal inflammation, ulceration, and structural damage that substantially

impair quality of life (Tavakoli et al., 2021; Ranasinghe et al., 2024). The pathogenesis of Crohn's disease results from a multifactorial interaction among genetic susceptibility, environmental triggers, immune dysregulation, and gut microbiota imbalance (Boyapati et al., 2015; Petagna et al., 2020). Genome-wide association studies have identified several susceptibility genes, including *NOD2/CARD15*, *IL23R*, and *ATG16L1*, which regulate innate immune signaling and autophagy pathways essential for intestinal barrier integrity (Petagna et al., 2020). Environmental and microbial factors can exacerbate mucosal immune activation in genetically predisposed individuals, leading to chronic intestinal inflammation.

The pathophysiology of Crohn's disease involves excessive activation of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-23, impaired mucin secretion, oxidative stress–induced epithelial injury, and compromised barrier permeability (Neurath, 2014; Muro et al., 2024). These mechanisms collectively contribute to tissue destruction, ulcer formation, and the development of complications such as fibrosis and megacolon.

Although current treatments—including antibiotics, anti-inflammatory corticosteroids, and monoclonal antibodies targeting TNF-α—have improved disease management, their comparative effects on gut physiology and histopathology remain insufficiently characterized (Tamilarasan et al., 2019; Girgis et al., 2024). Most previous studies emphasize clinical remission or cytokine levels, while parameters such as mucin content, pH balance, acidity, and oxidative stress are rarely evaluated together in an experimental framework.

The trinitrobenzene sulfonic acid (TNBS)-induced rat model replicates key features of Crohn's disease, including transmural inflammation, granuloma formation, and oxidative tissue damage, making it a suitable platform for evaluating therapeutic interventions (Ferreira-Duarte et al., 2021; Motavallian-Naeini et al., 2012).

Therefore, this study aimed to evaluate the efficacy of antibiotic (ciprofloxacin), anti-inflammatory (prednisolone), and monoclonal antibody (infliximab) therapies in a TNBS-induced rat model of Crohn's disease by assessing gut pH, total acidity, mucin content, oxidative stress (malondialdehyde levels), and histopathological alterations, in order to identify their relative therapeutic potentials and inform future therapeutic strategies.

2. MATERIALS AND METHODS

2.1 Animal grouping

Fifty adult male *Sprague-Dawley* rats (180–200 g) were used for this study. The animals were housed under standard laboratory conditions (12 h light/dark cycles) and had free access to water and standard chow. They were randomly divided into five groups (n = 10 per group) as shown below.

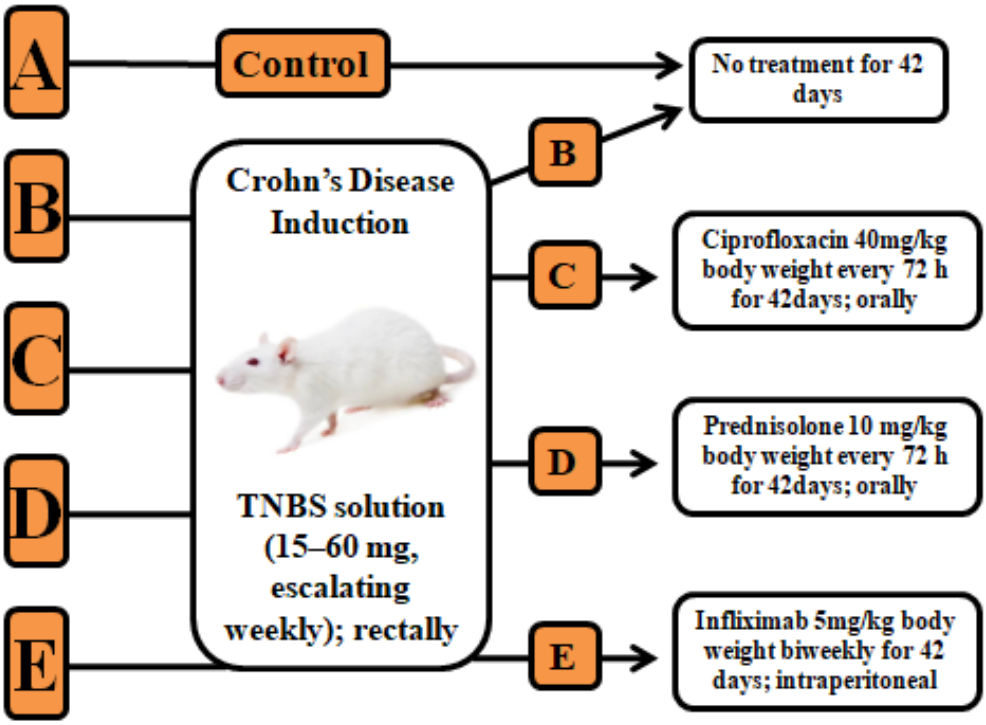


Figure 1. Animal grouping and Experimental Design

2.2 Ethical approval and animal welfare

All procedures involving animals were conducted in accordance with the *Guide for the Care and Use of Laboratory Animals* (National Research Council, 2011). Ethical approval was obtained from the Olabisi Onabanjo University Teaching Hospital Human Research Ethics Committee (OOUTH-HREC/746/2023AP).

Animal welfare was prioritized by minimizing discomfort through the use of mild anesthesia during invasive procedures, maintaining environmental enrichment, and ensuring humane endpoints. The use of rats was justified by the need to evaluate gut histology, oxidative stress, and biochemical alterations that cannot be replicated in vitro. The TNBS-induced Crohn's

disease model was chosen because it closely mimics human transmural inflammation and mucosal ulceration (Ferreira-Duarte et al., 2021).

2.3 Crohn's disease induction

Crohn's-like colitis was induced according to the method of Loeuillard et al. (2015) with modifications. Rats were fasted for 24 hours before induction but allowed free access to water. TNBS (Sigma-Aldrich, USA) was dissolved in 50% ethanol (v/v) to a total volume of 0.25 mL per rat and administered intrarectally using a flexible 3.5 Fr catheter inserted 8 cm into the colon. The dose was increased weekly (15, 30, 45, 60, 60, 60 mg) for six weeks to produce chronic transmural inflammation. After instillation, rats were maintained in a head-down position for 2–3 minutes to prevent leakage.

2.4 Administration of antibiotics, anti-inflammatory agent, and monoclonal antibody

Ciprofloxacin (40 mg/kg), prednisolone (10 mg/kg), and infliximab (5 mg/kg) were administered beginning 24 hours after the first TNBS induction. Ciprofloxacin and prednisolone were given orally every 72 hours for six weeks (Parashuram et al., 2017; Abuohashish et al., 2023). Infliximab (5 mg/kg) was administered intraperitoneally twice weekly for six weeks (Oyesola et al., 2025). The dosing schedules were selected based on published pharmacokinetic studies demonstrating effective systemic exposure and anti-inflammatory efficacy in rodent models (Norman et al., 2016; Li et al., 2023).

2.5 Assessment of hyperemia, adhesions, megacolon, and ulcerations

Rats were monitored daily for clinical signs of colitis including diarrhea, bloody stool, and body-weight loss. Macroscopic examination of the colon was performed to assess the severity of hyperemia, adhesions, megacolon, and ulceration using a validated semi-quantitative scoring system adapted from Motavallian-Naeini et al. (2012) (Table 1).

Table 1. Scoring system for macroscopic gut lesions

Feature	0	1	2	3
Hyperemia	None	Focal	Focal with bowel wall thickening	Extensive thickening of bowel wall
Adhesions	None	Mild	Moderate	Extensive
Megacolon	None	Mild	Moderate	Toxic megacolon
Ulcerations	None	Mild ulceration on one side < 1 cm	Moderate ulceration > 1 cm	Severe damage extending > 2 cm

2.6 Determination of pH, total acidity, and gastric mucus concentration

Gastric juice pH, total acidity, and mucin content were measured following Zakaria et al. (2011) and Corne et al. (1974). The stomach was isolated under mild anesthesia, and pH was determined using a calibrated digital pH meter (Hanna Instruments, HI 98127). One mL of distilled water was added to rinse the lumen, and gastric contents were centrifuged (3000 rpm, 10 min). Total acidity was determined by titrating the supernatant with 0.01 M NaOH using phenolphthalein as indicator until pH 7 was reached (Equ. 1).

$$Acidity\ (mEq/L) = \frac{\text{Volume of NaOH [mL]} \times \text{Normality of NaOH} \times 1000}{\text{Volume of gastric juice [mL]}} \quad \text{.....Equ. 1}$$

Mucin concentration was determined using 0.1% Alcian blue binding as described by Corne et al. (1974). The excised stomach was soaked for 2 hours in 0.1% Alcian blue dissolved in a buffer solution containing 0.1M sucrose and 0.05M Sodium acetate (pH adjusted to 5.8 with hydrochloric acid). The dye formed complexes with mucus after washing two times in 0.25M sucrose at 15 and 45 minutes. The mucus was eluted by immersion in 10mL aliquots of 0.5M $MgCl_2$ for 2 hours. The resulting blue solution was shaken with equal volumes of diethyl ether and centrifuged at 3000 rpm for 10 minutes. The optical density of the aqueous phase was measured at 580 nm using a spectrophotometer. Values of the absorbance measured in the different treatment groups was used to determine the corresponding concentration of alcian blue which formed complexes with mucin on the wall of the glandular portion of the stomach. Finally, the amount of mucin per gram of the net glandular tissue was calculated with a formula as shown below (Equ. 2);

$$\text{Mucin content}(\mu g/g) = \frac{\text{Alcian blue concentration } (\mu g)}{\text{Weight of glandular tissue (g)}} \quad \text{.....Equ. 2}$$

2.7 Determination of malondialdehyde (MDA) activity (lipid peroxidation)

Lipid peroxidation was quantified as malondialdehyde (MDA) using the thiobarbituric acid reactive substances (TBARS) method (Stocks & Dormandy, 1971). One mL of tissue homogenate was mixed with 2 mL of TCA-TBA-HCl reagent (15% trichloroacetic acid, 0.375% thiobarbituric acid, 0.25 N HCl), heated for 15 minutes in boiling water, and centrifuged at 3000 rpm for 10 minutes. Absorbance was read at 535 nm (Equ. 3).

$$\text{MDA}(nmol/ml) = \frac{\boldsymbol{OD} \times \varepsilon \times V}{\boldsymbol{v}} \quad \text{.....Equ. 3}$$

Where: OD = Absorbance, ε (molar extinction coefficient) = $1.56 \times 10^5\ M^{-1}\ cm^{-1}$, V = Total volume, v = Sample volume

2.8 Histological analysis

The stomach, small intestine and large intestine were harvested, trimmed of fat, and fixed in 10% neutral buffered formalin. Tissue processing involved dehydration in 70%, 90%, and 100% ethanol, clearing in xylene, and embedding in paraffin. Sections (5 μm) were stained with hematoxylin and eosin (H&E). Microscopic injury was scored (Table 2) based on mucosal architecture, crypt morphology, inflammatory cell infiltration, vascular dilatation, edema, and mast cell clustering, adapted from Ferreira-Duarte et al. (2021).

Table 2. Histological inflammation scoring system for microscopic gut injury

Histological Feature	Score 0	Score 1 (Mild)	Score 2 (Moderate)	Score 3 (Severe)
Mucosal architecture	Normal epithelium with intact villi and glands	Mild or focal distortion limited to lamina propria	Architectural distortion extending to submucosa	Diffuse destruction and loss of normal mucosal folds extending beyond submucosa
Crypt abnormalities	Normal crypt morphology	Mild crypt shortening or atrophy	Moderate atrophy and branching of crypts	Severe atrophy, branched crypts, cryptitis, or crypt abscesses
Inflammatory cell infiltration	No inflammatory infiltrate	Scattered mononuclear cells in lamina propria	Confluent inflammatory cells involving mucosa and submucosa	Massive transmural infiltration of mixed inflammatory cells
Vascular dilatation	Normal vasculature	Mild, localized vessel dilation	Moderate dilation of several vessels	Marked, generalized vascular dilation
Edema	None	Mild, limited to villi	Moderate, involving submucosa	Severe, extending through entire intestinal wall
Mast cells	Normal scattered mast cells	Small clusters (≤3) in submucosa	Multiple clusters (>3) in submucosa	Large clusters in both submucosa and serosa

Note: Each parameter was scored from 0 (normal) to 3 (severe). The total histological injury score was obtained by summing individual feature scores.

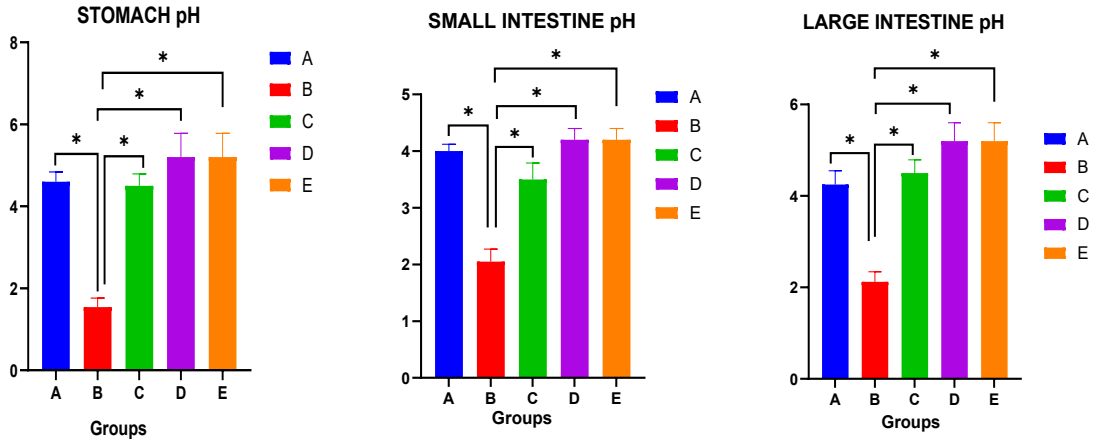
2.9 Statistical analysis

Data were expressed as mean ± standard error of mean (SEM). Statistical analyses were performed using GraphPad Prism version 8. One-way ANOVA followed by Bonferroni's post-hoc test was used to assess intergroup differences. The assumptions of normality and homogeneity of variance were verified using the Shapiro–Wilk and Levene tests, respectively. Statistical significance was considered at $P < 0.05$.

3. RESULTS

3.1 Gut pH

Induction of Crohn's-like colitis using TNBS significantly reduced gut pH levels in the stomach, small intestine, and large intestine compared with the normal control group ($p < 0.05$). Mean pH values declined from (4.6±0.24, 4.0±0.12, and 4.25±0.22) in controls to (1.54±0.22, 2.05±0.23, and 2.12±0.21), respectively, in the disease group. Treatment with ciprofloxacin (4.5±0.29, 3.5±0.24, 4.5±0.25), prednisolone (5.2±0.58, 4.2±0.20, 5.2±0.41), and infliximab (5.2±0.52, 4.3±0.24, 5.1±0.38), significantly restored gut pH toward normal levels, with infliximab-treated rats showing the highest improvement (Figure 2).

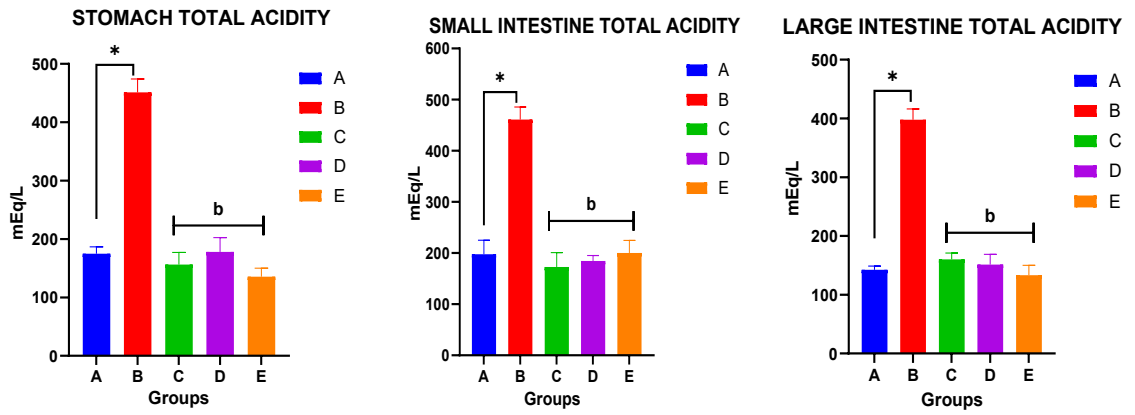


Values are mean ± SEM (n = 10).

Figure 2. Effect of antibiotic, anti-inflammatory, and monoclonal antibody therapies on gut pH in TNBS-induced Crohn's disease rats

3.2 Total acidity

Total acidity increased markedly in TNBS-treated rats, reaching (451.2±22.95, 460.4±24.22, and 397.8±18.42 mEq/L) in the stomach, small intestine, and large intestine, respectively, compared with (175±11.8, 197.7±27.5, and 142.6±6.2 mEq/L) in controls (Figure 3). Treatment with ciprofloxacin (156.4±20.7, 172.8±28.4, and 160.2±10.6 mEq/L), prednisolone (178.1±24.5, 184.6±10.6, and 151.4±17.3 mEq/L), and infliximab (135.6±14.5, 200.1±24.9, and 133.2±16.7 mEq/L), significantly reduced total acidity significantly ($p < 0.05$) most notably in the infliximab group.

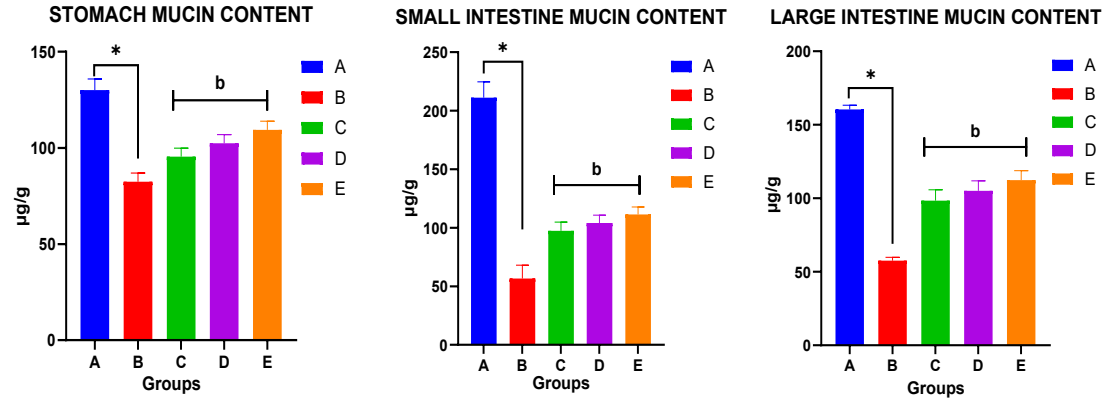


*Values are mean ± SEM (n = 10). * = P ≤ 0.05 vs. control; b = P ≤ 0.05 vs. disease group.*

Figure 3. Effect of antibiotic, anti-inflammatory, and monoclonal antibody therapies on total acidity in TNBS-induced Crohn's disease rats

3.3 Mucin content

TNBS administration significantly decreased mucin concentration in the gastric and intestinal mucosa ($p < 0.05$) (Figure 4). The control group showed mucin levels of (130.1±5.8 µg/g, 211.2±13.5 µg/g, and 160.4±2.9 µg/g) in the stomach, small intestine, and large intestine, respectively, while the disease group declined to (82.4±4.5 µg/g, 56.6±11.5 µg/g, and 57.6±2.3 µg/g). Ciprofloxacin (95.4±4.5 µg/g, 97.4±7.4 µg/g, and 98.4±7.2 µg/g), prednisolone (102.4±4.5 µg/g, 104.1±6.8 µg/g, and 105.1±6.4 µg/g), and infliximab (109.4±4.2 µg/g, 111.3±6.5 µg/g, and 112.3±5.2 µg/g) increased mucin levels in the stomach and intestine. The infliximab group showed the highest recovery ($p < 0.05$ vs. disease).

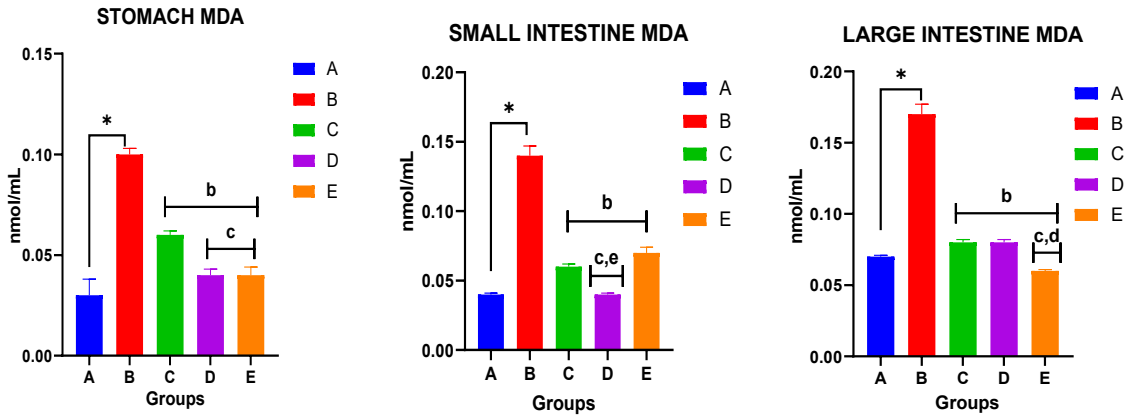


*Values are mean ± SEM (n = 10). * = P ≤ 0.05 vs. control; b = P ≤ 0.05 vs. disease group*

Figure 4. Effect of antibiotic, anti-inflammatory, and monoclonal antibody therapies on mucin content in TNBS-induced Crohn's disease rats.

3.4 Malondialdehyde (MDA) levels (lipid peroxidation)

Malondialdehyde levels, an indicator of oxidative stress, were markedly elevated in the TNBS group compared with controls (0.10±0.003, 0.14±0.007, and 0.17±0.006 nmol/mL) vs. (0.03±0.008, 0.04±0.001, and 0.07±0.002 nmol/mL), respectively; $p < 0.05$) (Figure 5). Ciprofloxacin (0.06±0.002, 0.06±0.003, and 0.08±0.002 nmol/mL), prednisolone (0.04±0.003, 0.04±0.001, and 0.08±0.002 nmol/mL), and infliximab (0.04±0.004, 0.07±0.003, and 0.06±0.001 nmol/mL) significantly reduced MDA levels, with infliximab and prednisolone demonstrating the greatest reduction ($p < 0.05$ vs. disease).



*Values are mean ± SEM (n = 10). * = P ≤ 0.05 vs. control; b = P ≤ 0.05 vs. disease group; c = P ≤ 0.05 vs. ciproflaxacin group; d = P ≤ 0.05 vs. prednisolone group*

Figure 5. Effect of antibiotic, anti-inflammatory, and monoclonal antibody therapies on malondialdehyde (MDA) levels in TNBS-induced Crohn's disease rats

Table 3. Effect of antibiotic, anti-inflammatory, and monoclonal antibody therapies on macroscopic gut lesions in TNBS-induced Crohn's disease rats.

Group	Ulceration	Hyperemia	Adhesion	Megacolon
A	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00
B	3.60±0.24[a]	3.00±0.00[a]	2.80±0.20[a]	2.80±0.20[a]
C	2.80±0.20[a]	2.40±0.24[a]	2.40±0.24[a]	1.40±0.24[b]
D	2.40±0.24[b]	1.80±0.20[b]	1.00±0.32[b]	1.20±0.37[b]
E	1.00±0.32[b]	1.00±0.32[b]	0.80±0.20[b]	0.40±0.24[b]

Values are mean ± SEM (n = 10). a = P ≤ 0.05 vs. control; b = P ≤ 0.05 vs. disease group.

3.5 Macroscopic gut pathology: ulceration, hyperemia, adhesion, and megacolon

TNBS induction produced severe ulceration, hyperemia, adhesions, and megacolon, with mean lesion scores of 2.8–3.6 compared to 0.0 in controls (Table 3). Treatment groups demonstrated

marked reduction of these lesions, particularly in the infliximab group, which showed near-normal scores (ulceration 1.0±0.3; megacolon 0.4±0.2).

3.6 Photomicrograph of small intestine histology

Photomicrographs of small intestine showed (A) control group with intact villi, goblet cells (thin black arrow), muscularis mucosa (thick black arrow), and Brunner's glands (red circle); (B) TNBS group showing degenerated villi and thickened muscularis layer; (C) ciprofloxacin-treated group showing regenerated villi; (D) prednisolone-treated showing partial mucosal restoration; and (E) infliximab-treated showing complete mucosal recovery (Figure 6).

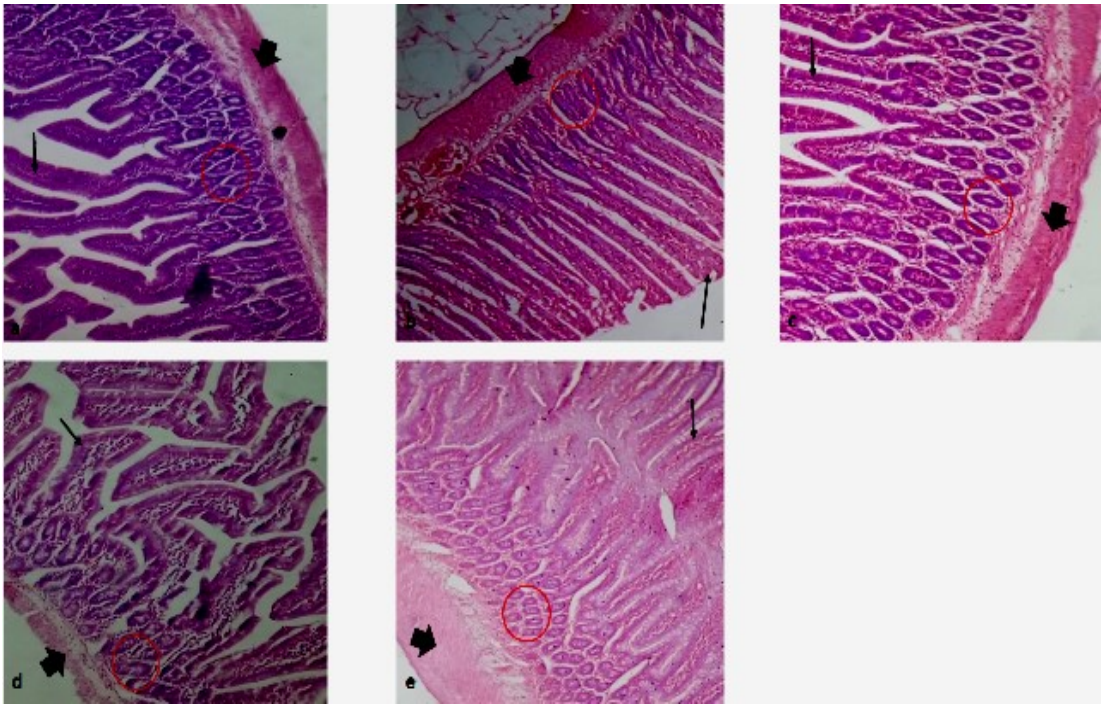


Figure 6. Photomicrograph of small intestine histology

3.7 Photomicrograph of Large Intestine

Photomicrographs of large intestine showed (A) control group with intact crypts of Lieberkühn (red circle), goblet cells (blue thin arrow), and muscularis layer (black thick arrow); (B) TNBS group showing severe villus atrophy and thickened muscularis layer; (C–E) treated groups showing progressive recovery of mucosal integrity, with infliximab exhibiting the most complete restoration (Figure 7).

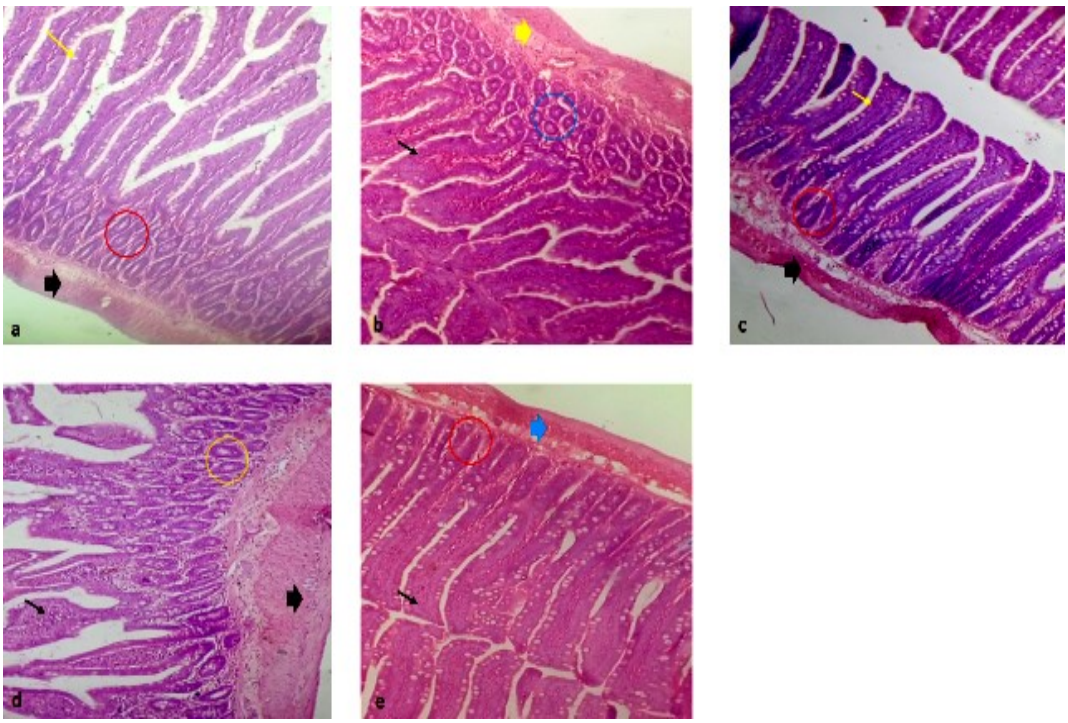


Figure 7. Photomicrograph of Large Intestine shows

4. DISCUSSION

The present study evaluated the therapeutic efficacy of antibiotic, anti-inflammatory, and monoclonal antibody therapies on gut inflammation, oxidative stress, and histopathology in a TNBS-induced rat model of Crohn's-like disease. The key findings showed that TNBS administration markedly decreased gut pH and mucin content while increasing total acidity and malondialdehyde (MDA) levels, indicating severe oxidative and mucosal injury. These alterations were accompanied by macroscopic ulceration, hyperemia, adhesions, and megacolon, as well as microscopic architectural distortion and inflammatory cell infiltration. Treatment with ciprofloxacin, prednisolone, and infliximab significantly ameliorated these changes, with infliximab producing the most complete restoration of gut structure and physiology.

The observed reduction in mucin and pH with elevated oxidative stress parallels findings in other TNBS-induced colitis studies where mucosal injury was associated with epithelial barrier dysfunction and lipid peroxidation (Motavallian-Naeini et al., 2012; Ferreira-Duarte et al., 2021). Similar to our results, Zheng et al. (2022) reported that an acidic intestinal environment promotes dysbiosis and inflammation in Crohn's disease. Restoration of mucin content by prednisolone

and infliximab agrees with Xu et al. (2021), who showed corticosteroids enhance epithelial barrier integrity in organoid models of Crohn's disease.

The marked reduction in MDA levels following therapy confirms the antioxidant benefits of anti-inflammatory and anti-TNF-α treatments reported by Li et al. (2023) and Muro et al. (2024). Our findings are also consistent with previous reports that ciprofloxacin attenuates oxidative stress and bacterial overgrowth, thereby improving intestinal barrier function (Norman et al., 2016; Aden & Reindl, 2019).

The protective effect of ciprofloxacin likely reflects its modulation of the gut microbiota and suppression of pathogenic bacteria metabolites that drive inflammation. Prednisolone's benefit stems from inhibition of NF-κB signaling and pro-inflammatory cytokines (TNF-α, IL-1β, IL-6), resulting in decreased leukocyte infiltration and restored mucin production (Xu et al., 2021). Infliximab's superior performance can be attributed to its neutralization of TNF-α, leading to reduced oxidative stress, down-regulation of adhesion molecules, and promotion of epithelial healing (Fatima et al., 2024). Collectively, these mechanisms converge to stabilize epithelial tight junctions, normalize luminal acidity, and enhance mucosal regeneration.

Histological analysis corroborated the biochemical data. TNBS-induced rats exhibited extensive villus blunting, crypt atrophy, and transmural inflammatory infiltration—hallmarks of Crohn's pathology (Neurath, 2014). In treated groups, particularly infliximab, mucosal restoration was evident through re-established villi, intact crypts of Lieberkühn, and minimal edema or vascular dilatation. These results align with studies demonstrating that anti-TNF therapy reduces mucosal ulceration and promotes tissue repair (Girgis et al., 2024).

The concurrent improvement in gut pH, mucin content, and MDA reduction across treatments underscores the interdependence between microbial balance, inflammation, and oxidative damage in Crohn's disease (Aden & Reindl, 2019). The findings suggest that therapeutic strategies targeting both the microbiome and inflammatory cytokine pathways may yield synergistic benefits. While antibiotics address bacterial triggers, monoclonal antibodies mitigate immune-mediated tissue injury.

This study was limited by its short treatment duration and absence of cytokine profiling or microbiota sequencing, which could have provided deeper mechanistic insights. Future work should investigate combination regimens and evaluate molecular markers such as TNF-α, IL-10, and tight-junction proteins to further elucidate how these therapies restore gut homeostasis.

Overall, the findings demonstrate that reducing oxidative stress and inflammation through antibiotic, corticosteroid, or monoclonal antibody therapy significantly improves intestinal physiology and architecture in experimental Crohn's disease, with infliximab showing the highest efficacy.

5. CONCLUSION AND RECOMMENDATIONS

This study demonstrates that therapeutic intervention with ciprofloxacin, prednisolone, and infliximab effectively mitigates TNBS-induced intestinal injury by restoring gut biochemical and structural integrity. All treatments improved key physiological parameters—including normalization of gut pH, reduction of total acidity, restoration of mucin content, and suppression of oxidative stress (malondialdehyde levels). Histological examination confirmed marked recovery of mucosal architecture, decreased inflammatory infiltration, and reduced vascular congestion, particularly in infliximab-treated rats.

These findings highlight the interrelated roles of microbial modulation, anti-inflammatory action, and oxidative stress control in maintaining intestinal homeostasis. Among the therapeutic options tested, infliximab exhibited the greatest efficacy in reversing both biochemical and morphological alterations, underscoring the central importance of TNF-α inhibition in Crohn's disease therapy.

Future research should investigate longer treatment durations, explore potential combination regimens, and incorporate molecular and microbiota analyses to better define mechanisms of gut healing and therapeutic synergy.

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Conflict of Interest

The author declares no conflict of interest.

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