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Ameliorative Effect of *Convolvulus pluricaulis* in a Rat Model of Ulcerative Colitis

Kinjal Patel*, Shivedra Verma, Rahul Trivedi, Sweta Besh and Rajesh A. Maheshwari

Department of Pharmacy, Sumandeep Vidyapeeth Deemed to be University, Piparia, Vadodara - 391760, Gujarat, India; kinjalpatel54@gmail.com

Abstract

Background: Ulcerative colitis (UC) is a chronic inflammatory disease characterized by recurrent inflammation and ulceration of the colonic mucosa. Its pathogenesis involves immune dysfunction, inflammatory cytokine production, and oxidative stress. Although conventional drugs provide symptomatic relief, prolonged therapy may cause undesirable side effects. *Convolvulus pluricaulis* possesses anti-inflammatory, antioxidant, and gastroprotective properties; however, its protective effects in experimental UC remain insufficiently investigated. **Objective:** The present research focused on evaluating the therapeutic potential of the methanol extract of *C. pluricaulis* (CPE) in a chemically induced model of ulcerative colitis in rats using dextran sulfate sodium. **Methods:** Ulcerative colitis was induced in Wistar rats by administering 5% DSS in drinking water for 14 days. Animals were divided into six groups: normal control, disease control, three CPE-treated groups (250, 500, and 1000 mg/kg, orally), and a sulfasalazine-treated group (500 mg/kg). Treatments were administered from day 14 to day 56. Evaluation parameters included the Disease Activity Index (DAI), colon length and weight, superoxide dismutase (SOD) and catalase activities, TNF-α, IL-6, and CRP levels, and colonic histopathological changes. **Results:** The disease control group exhibited increased DAI, colon weight, inflammatory biomarkers, and histological damage, with decreased antioxidant enzyme activities. CPE at 500 and 1000 mg/kg reduced DAI scores, preserved colon length, decreased colon weight, significantly suppressed CRP, TNF-α, and IL-6 levels, and restored SOD and catalase activities. Histological examination revealed substantial protection of colonic tissue architecture at these doses. The 250 mg/kg dose produced minor, statistically insignificant improvements in most parameters. The effects of higher CPE doses were comparable to those of sulfasalazine. **Conclusion:** *Convolvulus pluricaulis* extract exhibited dose-dependent anti-inflammatory and antioxidant effects in DSS-induced colitis in rats. Doses of 500 and 1000 mg/kg effectively alleviated biochemical and histopathological indicators of colonic damage, suggesting its potential as a therapeutic candidate for ulcerative colitis.

Major Findings: *C. pluricaulis* methanol extract demonstrated significant dose-dependent amelioration of DSS-induced colitis, particularly at 500 and 1000 mg/kg, by reducing disease activity, inflammatory mediators (TNF-α, IL-6, and CRP), and colonic damage while restoring SOD and catalase activities. Histopathological evaluation corroborated the biochemical findings, indicating preservation of colonic mucosal architecture and attenuation of inflammation.

Keywords: *Convolvulus pluricaulis,* Colitis, Colon Weight, DAI, Dextran Sulfate Sodium, Inflammatory Biomarkers

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1. Introduction

Ulcerative colitis (UC) is a persistent and recurrent form of Inflammatory Bowel Disease (IBD) that mainly targets the mucosal lining of the colon. It is clinically marked by symptoms such as abdominal discomfort, frequent diarrhoea, rectal bleeding, and unintended weight loss. Although substantial headway has been achieved in elucidating the disease mechanisms, the primary cause of UC remains largely unknown[1,2]. Current therapeutic options—including corticosteroids, aminosalicylates and immunomodulators—often come with significant side effects and limited long-term effectiveness[3]. This has led to increased interest in alternative treatment

*Author for correspondence

strategies, especially those derived from medicinal plants, because of their capacity to offer better safety and lasting treatment benefits[4].

Shankhpushpi (*Convolvulus pluricaulis*) is a well-recognised medicinal herb frequently utilised in *Ayurveda*, historically valued for its cognitive-enhancing, anti-inflammatory, and antioxidant effects[5-7]. Emerging pharmacological research indicates that it may help regulate oxidative stress and inflammation—two key contributors to UC pathogenesis. However, there is a lack of comprehensive studies evaluating its therapeutic role in experimental models of inflammatory bowel disease.

This work employs a well-established preclinical model of ulcerative colitis (DSS-induced in rats) to investigate the protective efficacy of *C. pluricaulis* against colonic inflammation. Through analysing clinical symptoms, tissue-level changes, and biochemical indicators of inflammation, this research aims to determine the herb's therapeutic potential of natural origin for UC and to reinforce the relevance of traditional botanical remedies in contemporary treatment approaches for inflammatory disorders.

2. Materials and Methods

2.1 Drugs and Chemicals

DSS was sourced from Tokyo Chemical Industries, India, based in Chennai. The methanol extract of *C. pluricaulis* was supplied by Amsar Private Limited, Indore. Sulfasalazine (SSZ) tablets along with ketamine and xylazine were obtained from a licensed local pharmacy. Biochemical analysis kits were provided by Endocrine and Allergy Laboratory Pvt. Ltd., Ahmedabad.

2.2 Experimental Animals

The study was conducted in compliance with the protocol approved by the Institutional Animal Ethics Committee of SVDU (Approval No.: SVU/DP/IAEC/2022/11/60). Adult Wistar rats of either sex, weighing 200–250 g, were utilised for the experiments. The animals were housed under controlled laboratory conditions, including a 12-hour light/dark cycle, ambient temperature of 24 °C, and relative humidity maintained between 35% and 60%. A standard rodent diet was provided, and purified water was available ad libitum.

2.3 Experimental Design

Six groups, each comprising six rats, were randomly assigned and treated according to the experimental protocol presented in Table 1.

Upon study completion, anaesthesia was achieved by intraperitoneal administration of ketamine (10 mg/kg) along with xylazine (1 mg/kg). Specimens of blood were drawn from the retro-orbital plexus and left to clot. Following centrifugation at 5000 rpm for 20 minutes, serum was extracted and preserved at –20°C for future biochemical testing. Colons were dissected, rinsed with normal saline, and assessed macroscopically for length and weight. Tissue samples were immersed in 10% formalin for subsequent microscopic evaluation.

2.4 Determination of the Disease Activity Index for Colitis

The extent of colitis was evaluated every day using the Disease Activity Index (DAI), using scoring criteria from a previously established method[10]. The DAI was

Table 1. Experimental groups and treatment regimen in the DSS-induced ulcerative colitis model

Group	Treatment Description	DSS Administration	Treatment (Day 14–56)	Dose
I	Normal control	No	Distilled water	-
II	Disease control	5% DSS provided in the drinking water over 14 days[8,9]	None	-
III	Treatment I – *C. pluricaulis* (Low dose)	5% DSS provided in the drinking water over 14 days	*C. pluricaulis* methanol extract	250 mg/kg via oral route
IV	Treatment II – *C. pluricaulis* (Medium dose)	5% DSS provided in the drinking water over 14 days	*C. pluricaulis methanol* extract	500 mg/kg via oral route
V	Treatment III – *C. pluricaulis* (High dose)	5% DSS provided in the drinking water over 14 days	*C. pluricaulis* methanol extract[9]	1000 mg/kg via oral route
VI	Standard treatment	5% DSS provided in the drinking water over 14 days	Sulfasalazine	500 mg/kg via oral route

determined by averaging the individual scores for reduction in body weight, faecal consistency, and blood in stool, as outlined in Table 2.

2.5 Assessment of Oxidative Stress Markers

The evaluation of catalase enzyme activity, along with the analysis of key endogenous antioxidant enzymes, among which Superoxide Dismutase (SOD), was performed in accordance with well-established and validated methodologies described in prior studies[11,12].

2.6 Measurement of Pro-inflammatory Markers TNF-α, IL-6, and CRP

Concentrations of TNF-α, IL-6, and CRP in serum samples underwent quantitative analysis using ELISA kits, strictly following the maker'sguidelines.

2.7 Histopathology

Colon tissue samples were swiftly excised, rinsed with saline, and immersed in 10% buffered formalin for fixation. Subsequent to fixation, the tissues were cast in paraffin; sections of tissue, each sliced at a thickness of 5 µm and stained with hematoxylin and eosin for histopathological evaluation. Observations were made using a bright-field microscope (Olympus BX10, Japan), and photomicrographs were captured using an Olympus DP12 camera (Japan). To ensure unbiased evaluation, the histopathological examination was carried out by a pathologist who was unaware of the treatment group allocations.

2.8 Statistical Evaluation

All values are reported as mean ± SEM. Statistical comparisons among groups were assessed using one-way ANOVA with Bonferroni's post hoc test where necessary, analysed via GraphPad Prism. A p-value less than 0.05 was taken as statistically significant.

3. Results

3.1 Impact of CPE on DAI, Colon Length and Weight in DSS-induced Colitis

The DAI score was significantly higher in the disease control rats than in the normal control animals, indicating the severity of the condition. CPE treatment at 500 mg/kg and 1000 mg/kg caused a remarkable reduction in DAI scores compared with the untreated rats. In contrast, the 250 mg/kg dose of CPE did not result in a statistically significant change in DAI score (Figure 1).

3.2 Impact of CPE on Colon Length and Colon Weight in DSS-persuaded Colitis

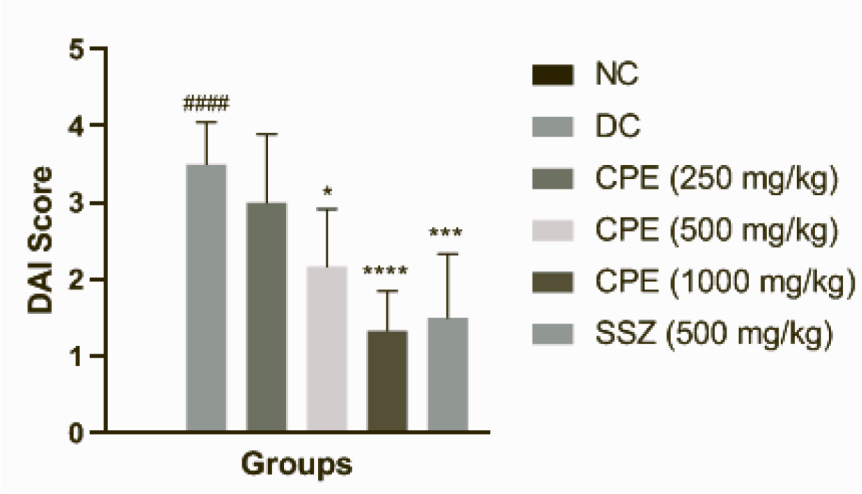
The colon length was significantly reduced in the disease control animals compared with the normal control rats, indicating inflammation-associated tissue shortening. Treatment with CPE at doses of 500 mg/kg and 1000 mg/kg expressively preserved colon length as opposed to the disease control group. But the 250 mg/kg dose of CPE was unable to produce a statistically substantial alteration in colon length. Colon weight was considerably elevated in the disease control group as opposed to the healthy control group, suggesting inflammation-related oedema or tissue thickening. Administration of CPE at 500 mg/kg and 1000 mg/kg expressed a statistically significant reduction in colon weight in relation to the disease control group. In contrast, the 250 mg/kg dose did not cause a statistically significant change in colon weight (Figure 2).

3.3 Impact of CPE on CRP, TNF-α and IL-6 in DSS-induced Colitis

Levels of inflammatory markers—CRP, TNF-α, and IL-6—were significantly higher in the untreated animals relative to the control rats, indicating heightened systemic inflammation. Oral administration of CPE

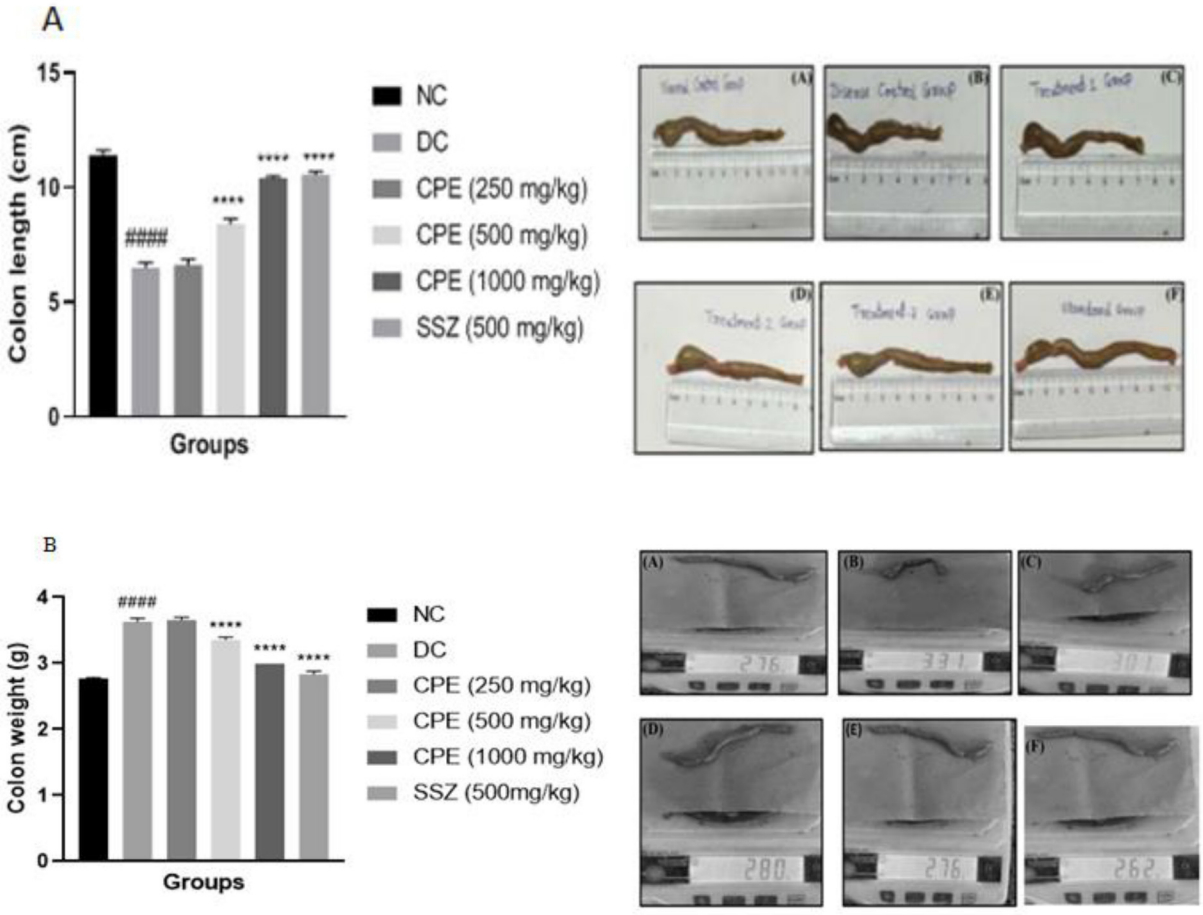
Table 2. Scoring criteria for DAI based on reduction in body weight, faecal consistency, and blood in stool

DAI Score	Fecal Consistency	Blood in Stool	Reduction in Body Weight	Description
0	Normal	Negative	0%	Healthy condition
1	Normal	Weakly positive	1–5%	Mild symptoms
2	Soft	Positive	6–9%	Moderate symptoms
3	Soft	Strongly positive	10–15%	Severe symptoms
4	Diarrhea	Gross bleeding (visible blood)	>15%	Very severe condition



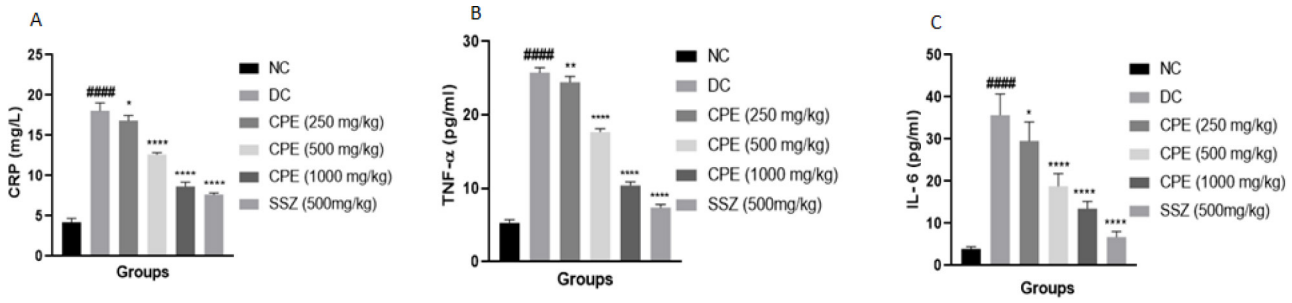
The findings are reported as mean ± SEM (n = 6). ####P<0.001 indicates a significant difference against the normal control group; *P<0.05, ***P<0.001, and ****P<0.0001 denote significant differences verses the disease control group.

Figure 1. Effect of CPE on disease activity index in DSS-induced colitis.



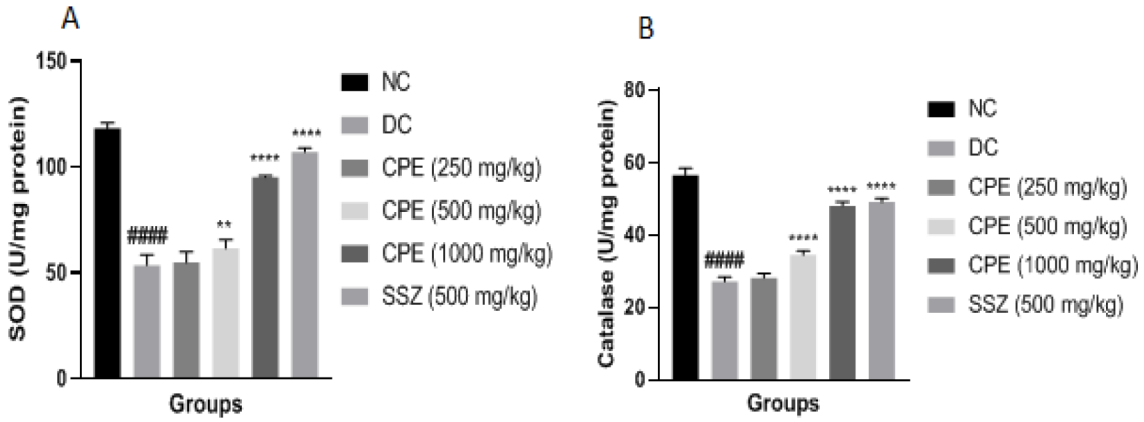
The findings are reported as mean ± SEM (n = 6). ####P<0.001 indicates a significant difference against the normal control group; ****P<0.0001 denote significant differences verses the disease control group.

Figure 2. Effect of CPE on colon length **(A)** and colon weight **(B)** in DSS-induced colitis.



The findings are reported as mean ± SEM (n = 6). ####P<0.001 indicates a significant difference against the normal control group; *P<0.05, **P<0.01 ****P<0.0001 denote significant differences verses the disease control group.

Figure 3. Effect of CPE on CRP **(A)**, TNF-α **(B)** and IL-6 **(C)** in DSS-induced colitis.



The findings are reported as mean ± SEM (n = 6). ####P<0.001 indicates a significant difference against the normal control group; **P<0.01 ****P<0.0001 denote significant differences verses the disease control group.

Figure 4. Effect of CPE on SOD **(A)** and catalase **(B)** in DSS-induced colitis.

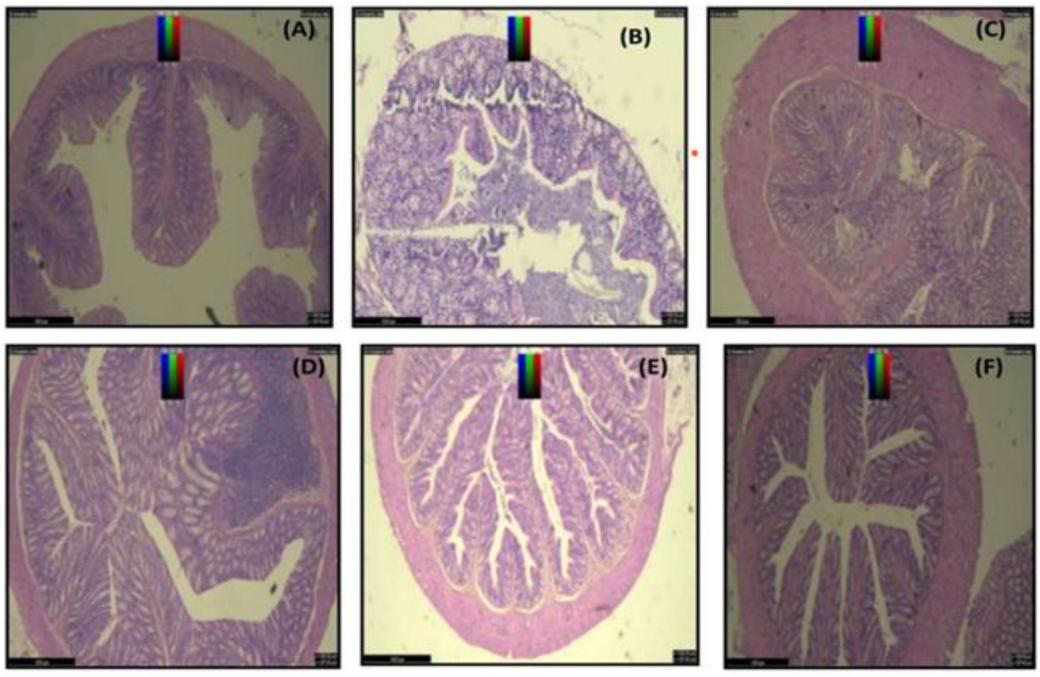


Figure 5. Effect of CPE on histology of colon: **(A)** NC **(B)** DC **(C)** CPE 250 mg/kg **(D)** CPE 500 mg/kg **(E)** CPE 1000 mg/kg in DSS-induced colitis.

at doses of 250 mg/kg, 500 mg/kg, and 1000 mg/kg contributed to a measurable decrease in CRP levels, TNF-α, and IL-6 versus the disease control group, suggesting dose-dependent inflammation-lowering effects of the extract (Figure 3).

3.4 Impact of CPE on SOD and Catalase in DSS-induced Colitis

A significant suppression of SOD and catalase levels was seen in the disease control group versus normal controls, suggesting heightened oxidative stress. Treatment with CPE at doses of 500 mg/kg and 1000 mg/kg effectively reversed these changes, significantly enhancing enzyme activities. However, the 250 mg/kg dose of CPE did not induce any notable improvement in either enzyme (Figure 4).

3.5 Impact of CPE on Histology of Colon in DSS-induced Colitis

Histopathological analysis of colon tissues corroborated the biochemical results. The normal control group displayed well-preserved mucosal and glandular structure. In contrast, the disease control group exhibited severe histological abnormalities, including mucosal erosion, crypt loss, depletion of goblet cells, and marked inflammatory cell infiltration. Administration of CPE at 500 mg/kg and 1000 mg/kg meaningfully attenuated these pathological changes, as evidenced by the preservation of crypt architecture and a reduction in inflammatory infiltration. Similarly, SSZ-treated rats demonstrated comparable protective effects. However, CPE at a dose of 250 mg/kg resulted in only mild to moderate histological improvement (Figure 5).

4. Discussion

This research aimed to determine the therapeutic value of CPE in a rat model of colitis, revealing dose-dependent anti-inflammatory, antioxidant, and tissue-protective effects. The DAI—which integrates clinical signs such as weight loss, stool consistency, and bleeding—was significantly higher in the disease control group, confirming severe colonic inflammation. These results align with previous research that validates DAI as a robust marker of colitis severity[13]. Administration of CPE at 500 mg/kg and 1000 mg/kg significantly lowered DAI scores, indicating improvement in disease manifestations, whereas the 250 mg/kg dose showed no statistically meaningful impact. This dose-dependent effect is consistent with earlier studies where higher doses of plant-derived extracts yielded significant therapeutic benefits in colitis models[14,15].

Colonic shortening, a characteristic indicator of inflammation-induced tissue damage, was observed in the disease control animals but was notably reversed by CPE treatment at 500 and 1000 mg/kg. This observation concurs with prior findings that associate colon length reduction with mucosal injury severity and inflammatory swelling. Additionally, the reduced colon weight in CPE-treated groups further supports the extract's capacity to alleviate inflammation-related oedema and leukocyte infiltration, paralleling the effects of other polyphenol- and flavonoid-rich natural compounds, which modulate inflammatory pathways by suppressing mediator synthesis[13,16].

Markers of systemic inflammation—CRP, TNF-α, and IL-6—were meaningfully elevated in the disease control group, consistent with active inflammatory processes in colitis. Treatment with CPE at all tested doses effectively decreased these cytokines and acute-phase proteins in a dose-dependent manner, underscoring the extract's systemic anti-inflammatory potential. These findings are in agreement with reports demonstrating the anti-inflammatory capacity of phytochemical-rich agents like alkaloids, flavonoids and phenolic compounds in inflammatory bowel disease models[17-20].

Oxidative stress, a key contributor to colitis pathogenesis, was evidenced by the marked depletion of endogenous antioxidant enzymes SOD and catalase in diseased rats. Such enzyme reductions are well documented in IBD and correlate with elevated reactive oxygen species and mucosal injury[21]. CPE treatment at 500 and 1000 mg/kg significantly restored these antioxidant defences, highlighting its free radical scavenging and enzymatic protective effects, consistent with other studies on plant-derived antioxidants in colitis[22-25].

Histological evaluation reinforced the biochemical and clinical findings, showing severe mucosal disruption, crypt damage, goblet cell loss, and inflammatory infiltration in the disease control group—classical features of colitis[26]. Conversely, rats receiving 500 and 1000 mg/kg of CPE displayed

marked preservation of mucosal architecture and reduced inflammatory cell infiltration, effects comparable to those observed with sulfasalazine treatment. The 250 mg/kg dose produced only modest histological improvements, further supporting the dose-dependent nature of CPE's protective properties. Similar histopathological improvements have been documented with other bioactive phytochemicals such as quercetin and β-sitosterol, reinforcing the therapeutic promise of plant-based interventions in intestinal inflammation[27,28].

5. Conclusion

In conclusion, the present study provides compelling evidence that CPE confers significant dose-dependent protective effects in the management of colitis. Administration of higher doses of CPE (500 mg/kg and 1000 mg/kg) markedly diminished disease-associated clinical symptoms by significantly reducing inflammatory responses, oxidative stress levels, and histopathological damage. These doses effectively preserved critical parameters such as colon length and tissue integrity, highlighting the extract's ability to maintain colonic structure and function under inflammatory conditions. The observed decrease in pro-inflammatory cytokines and restoration of antioxidant enzyme activities further underscore the multifaceted mechanisms through which CPE mediates its beneficial effects. Conversely, the lower dose of 250 mg/kg demonstrated limited efficacy, suggesting a threshold dosage is necessary to achieve therapeutic outcomes. Taken together, these findings indicate that CPE possesses potent anti-inflammatory, antioxidant, and mucosal protective properties that could be harnessed for the development of novel treatments for colitis. Future research should focus on isolating the active compounds within CPE and elucidating the molecular pathways involved to fully exploit its therapeutic potential and ensure safety and efficacy in long-term applications.

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Author Contribution

Kinjal Patel: Conceptualisation, literature review, drafting, and revision; Sivendra Verma: Data collection, references, tables, figures, and editing; Sweta Besh: Scientific review and editing; Rahul Trivedi: Literature analysis and critical revision; Rajesh Maheshwari: Supervision, review, and final approval. All authors have read and agreed to the published version of the manuscript.