



RESEARCH ARTICLE

Protective role of curcumin against colon injury through the regulation of oxidative stress, inflammation and maintenance of colon tissue architecture

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Abstract

The study evaluated the protective effect of curcumin (Cr) and the underlying mechanisms involved in colon injury induced by azoxymethane (AOM). The anti-inflammatory and antioxidant potential of Cr was evaluated through measurement of proinflammatory markers and antioxidant enzymes levels using enzyme linked immunosorbent assay (ELISA). The colon tissue architecture and fibrosis were examined through haematoxylin and eosin (H & E) and Masson trichrome and Sirius red staining. Further, the expression pattern of interleukin - 6 (IL - 6) protein was analysed using immunohistochemistry (IHC) staining. The results indicate that Cr was effective in reducing colon lesions induced by AOM by decreasing the levels of tumour necrosis factor - alpha (TNF - α), C - reactive protein (CRP) and IL - 6 ($p < 0.05$). Curcumin treatment regulates oxidative stress in the colon tissues by reducing lipid peroxidation and increasing the levels of antioxidant enzymes level ($p < 0.05$). Histopathological analysis showed that AOM treatment caused colon injury, including congestion, inflammation and fibrosis. Curcumin treatment maintained colon tissue architecture by reducing congestion, fibrosis and inflammation. Interleukin - 6 expression was significantly increased in the colon tissues induced by AOM. However, Cr treatment decreased IL - 6 protein expression compared with the AOM treated group. These results suggest that Cr consumption may be a promising natural remedy for preventing colon pathogenesis. Given its capacity to reduce inflammatory markers and oxidative stress, Cr might play a vital role in maintaining colon health.

Keywords: colon lesion; curcumin; immunohistochemistry; inflammation; oxidative stress

Introduction

Inflammatory bowel diseases (IBD), including Crohn's disease (CD) and ulcerative colitis (UC), are characterised by chronic inflammation of the intestinal mucosa and are among the most prevalent gastrointestinal disorders (1). The incidence and prevalence of IBD have continued to increase worldwide, with a growing number of patients presenting with IBD related symptoms (2). The etiology of IBD is not yet fully understood; however, it is recognised as a complex and heterogeneous group of gastrointestinal disorders involving interactions among genetic susceptibility, intestinal microbiota, immunological dysregulation and environmental factors in genetically predisposed individuals (3). The interplay among these factors disrupts intestinal homeostasis, resulting in dysregulated gut inflammatory responses. Moreover, an imbalance between pro-inflammatory and anti-inflammatory mediators is considered a major contributor to intestinal inflammation in IBD (4). Because

inflammation is closely associated with the generation of reactive intermediates, including reactive oxygen species (ROS) and reactive nitrogen species (RNS), oxidative stress has been proposed as a key mechanism involved in the pathophysiology of IBD (5). Furthermore, oxidative stress is closely related with inflammatory responses and it has been concerned in the propagation and exacerbation of IBD (6). Azoxymethane (AOM) is commonly used as a compound tumour inducer in animal models to cause colon pathogenesis, especially in studies to stimulate colorectal cancer development (7, 8). Azoxymethane induced morphological alterations were associated with aberrant crypt foci (ACF) development as well as DNA damage in proliferated cells (9, 10). The AOM induced morphological alterations, involving ACF formation, are a sign of DNA damage in epithelial cells rapidly and are associated with early stages in colon cancer (11, 12). The current pharmacological treatments for colonic lesions, including colorectal cancer and IBD, are effective but often

cause side effects such as increased risk of infections, gastrointestinal disturbances and immunosuppression (13, 14). So, there is an increasing demand for more efficient and safer therapeutic options. Natural compounds and their bioactive compounds demonstrate significant potential in the management of colon lesions due to antioxidant and chemopreventive effects and anti-inflammatory (15, 16). Curcuma longa (turmeric) contains the primary natural polyphenol in its rhizome. Curcumin has shown great potential for disease prevention by modulating inflammatory pathways and neutralising free radicals due to its strong antioxidant and anti-inflammatory properties (17, 18). Additionally, Cr has been found to reduce the activity of xanthine oxidase that plays a role in the synthesis of ROS in phorbol-12-myristate-13-acetate (PMA)-treated NIH3T3 cells, thereby inhibiting PMA induced tumour promotion and decreasing ROS production (19, 20). The aim of the current study is to explore the protective effects of Cr against colon injury with particular focus on its antioxidant, anti-inflammatory and preserving tissue architecture. The role of Cr in reducing fibrosis, as assessed by Sirius staining and Masson trichrome. Furthermore, the expression patterns of inflammatory protein (interleukin - 6 (IL - 6)) were determined by immunohistochemistry (IHC) in different experimental groups.

Materials and Methods

Grouping of animals and treatment plans

A total number of 32 male albino rats of 5–6 weeks old with weights between 175–200 grams were acquired from King Saud University, Saudi Arabia. Following one week of acclimation, the animals were divided into 4 experimental groups. Each group consisted of 8 rats that were treated for a period of 5 weeks consecutively. Group I served as the control group, receiving normal saline solution via oral gavage. Group II, the disease control group, was administered AOM orally at a dosage of 15 mg / kg body weight (21) twice a week for the same duration. Group III received a co-treatment of AOM and Cr, with Cr administered at a dosage of 50 mg / kg body weight (22, 23). Group IV was treated exclusively with Cr (Table 1). After 5 weeks, animals were sacrificed and samples (colon tissue and blood) were collected for the assessment of antioxidant enzyme activity, inflammatory marker levels, lipid peroxidation, as well as for histopathological and immunohistochemical analyses. Ethical approval was obtained with number QU-J-UG-2-2025-54308.

Measurement of oxidative stress markers

After 5 weeks of treatment, all rats were euthanised using chloroform and colon tissues were taken and rinsed with phosphate buffered saline (PBS). The tissues were homogenised and the homogenate was centrifuged for 15 min. The blood was collected and serum was separated for biochemical assessment. The supernatant and serum samples were then stored at -20 °C for subsequent investigation of antioxidant enzymes from Abcam in Cambridge, UK. The results were then analysed accordingly.

Measurement of inflammatory markers level

Enzyme linked immunosorbent assay (ELISA) was made to quantitatively evaluate the inflammatory markers tumour necrosis factor - alpha (TNF - α), C - reactive protein (CRP) and IL - 6 in colon tissue homogenates / serum samples by ELISA kits, conferring to the manufacturer's instructions. The absorbance was measured by a microplate reader at the suggested wavelength. The concentrations of inflammatory markers were designed from the standard calibration curve and expressed as pg / mL.

Evaluation of lipid peroxidation levels

Lipid peroxidation was calculated by measuring malondialdehyde (MDA) levels using a lipid peroxidation assay kit from Abcam, UK. The absorbance was measured using a microplate reader at the recommended wavelength. Raised MDA levels were considered as increased membrane lipid peroxidation.

Histopathological analysis

The rats were sacrificed, washed in normal saline and the colon tissue samples were fixed in 10 % buffered formalin. An automated tissue processor was used to process the tissues (Leica TP 1020) and paraffin blocks were made. After that, a rotary microtome (Leica RM 2125RT) was used to cut sections at a thickness of 5 μ m of paraffin embedded blocks. Histopathological examination was performed on colon tissue samples obtained from all experimental groups using hematoxylin and eosin (H & E) staining to evaluate morphological and pathological alterations. After staining, the sections were mounted by distyrene plasticizer xylene (DPX) mounting medium and examined under a light microscope (Leica DMD108). Representative photomicrographs were captured and examined for histopathological changes. Moreover, Sirius red and Masson trichrome staining (Connective Tissue Stain, Abcam) was used to evaluate the fibrosis in different experimental groups and results were interpreted accordingly.

Expressional evaluation of interleukin - 6 protein through immunohistochemical staining

The expression pattern of IL - 6 protein (Abcam, U.K) in the colon tissue was evaluated as described earlier (23). Thin colon tissue sections (5 μ m) were cut and sections were put on polylysine coated slides and dewaxed, hydrated and rinsed in buffered phosphate saline. Endogenous peroxidase activity was done through 0.3 % hydrogen peroxide for 15 min to block the endogenous peroxidase activity. Moreover, antigen retrieval via microwave for 22 min was performed in citrate buffer (pH 6.0), followed by washing with PBS three times for 3 min each. Then, a blocking agent was added to a tissue to block the unwanted site. Primary monoclonal antibody IL - 6 protein was applied at a 1 : 250 dilution overnight at 4 °C in a humid chamber. Thereafter, slides were incubated with a secondary antibody for 1 hr, followed by streptavidin - biotin enzyme complex incubated at room temperature for 40 min. Lastly, slides were counterstained with hematoxylin and diaminobenzidine (DAB) as a chromogen. Independent pathologists evaluated the IL - 6 protein expression scores. Cytoplasmic positive cells were counted in 4

Table 1. Animals were randomly divided into four groups, having eight animals per group

Group name	Short name	Treatment plan
Normal control	NC	A standard saline solution was given to the rats
Disease control (negative control)	AOM	Azoxymethane administered orally
Curcumin treatment (treatment group)	AOM + Cr	Co-treatment with AOM and Cr
Curcumin treatment only	Cr	Curcumin only

selected fields from each tissue section. Interleukin - 6 expression was considered negative when it was less than 5 %. The percentage positivity was graded as follows: 5–20 % were considered as 1, 20–30 % as 2, 30–65 % as 3 and more than 65 % as 4. The results were analysed and photographs were captured. Cytoplasmic expression of IL - 6 protein was considered as positive and bluish colour considered as negative.

Statistical analysis

All data are presented as mean $\pm$ standard error of the mean (SEM). Comparisons among multiple groups were carried out using one way analysis of variance (ANOVA) with a statistical package (SPSS) followed by Tukey's post hoc test. Differences were considered statistically significant at $p < 0.05$.

Results

Effect of curcumin on oxidative stress

In this investigation, the levels of antioxidant enzymes were measured. It was observed that all the antioxidant enzyme levels were within the

normal range in the control group. However, in the disease control group, antioxidant enzyme concentrations were significantly lower ($p < 0.05$) than in the control group (Fig. 1). Moreover, in the Cr + AOM treated group, antioxidant enzyme levels significantly ($p < 0.05$) as compared to the disease control group.

Effect of curcumin on lipid peroxidation

High levels of ROS cause the peroxidation of membrane lipids, causing MDA in the generation of the by-product MDA. The increased membrane lipid peroxidation caused by AOM induced a rise in MDA generation. Malondialdehyde levels significantly increased in AOM induced group as compared to the control group ($p < 0.05$) (Fig. 2). However, the increased MDA levels were significantly reduced after Cr treatment as compared with the AOM treated group ($p < 0.05$).

Effect of curcumin on inflammatory markers

In the current study, the levels of anti-inflammatory markers (TNF - α , CRP and IL - 6) were assessed in different experimental groups of rats. In the disease control group, inflammatory marker levels were significantly high ($p < 0.05$) in contrast to the control

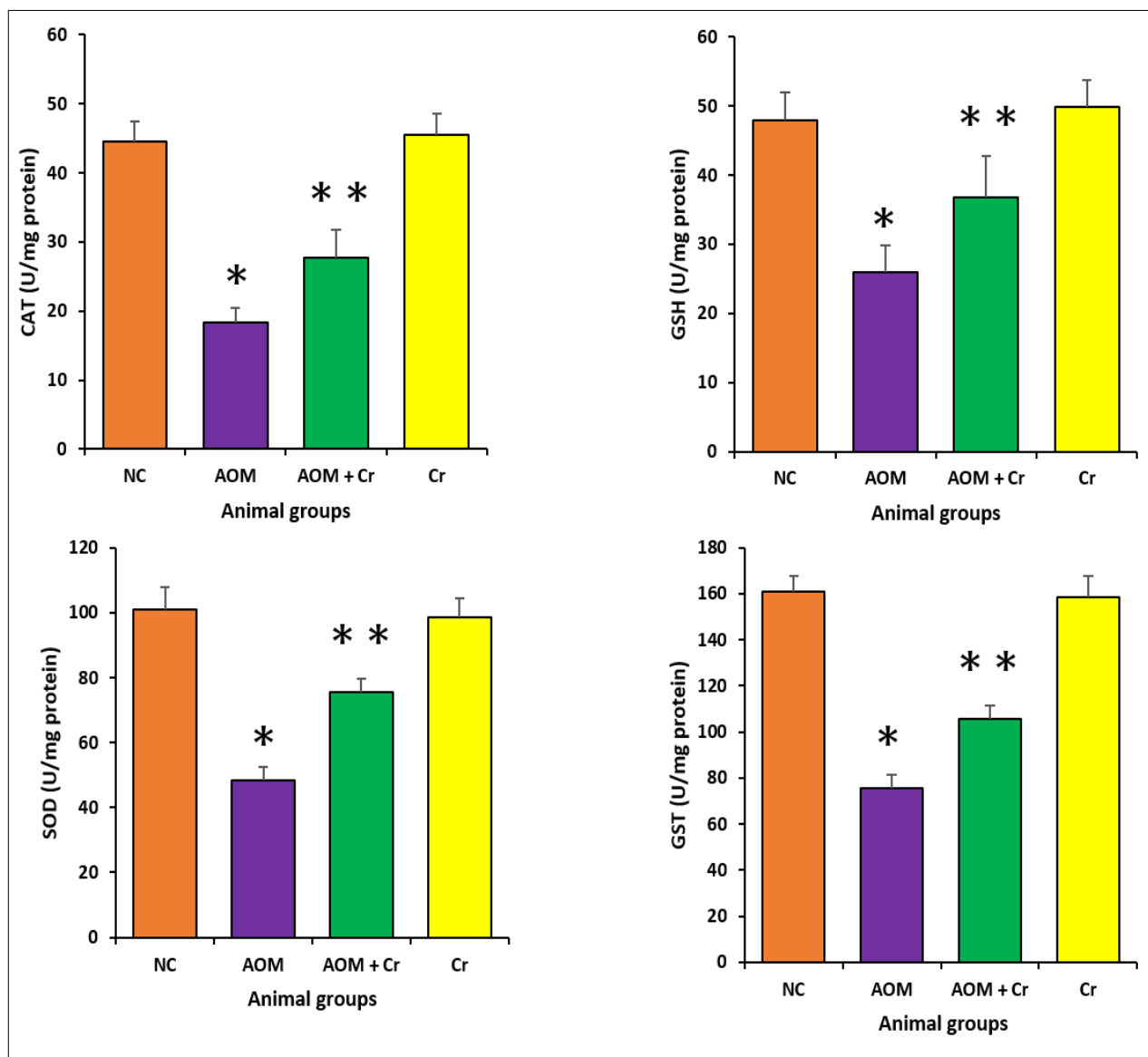


Fig. 1. The antioxidant enzymes levels in different experimental groups examined. The values signify the standard error of mean ($\pm$ SEM), with 8 rats per group. Differences were considered statistically significant at $p < 0.05$. In the figures, * indicates a significant difference compared with the normal control (NC) group, while ** indicates a significant difference compared with the AOM-treated group.

CAT: catalase; GSH: glutathione; SOD: superoxide dismutase; GST: glutathione S-transferase; NC: normal control; AOM: azoxymethane administered orally; AOM + Cr: co-treatment with azoxymethane administered orally and curcumin; Cr: curcumin.

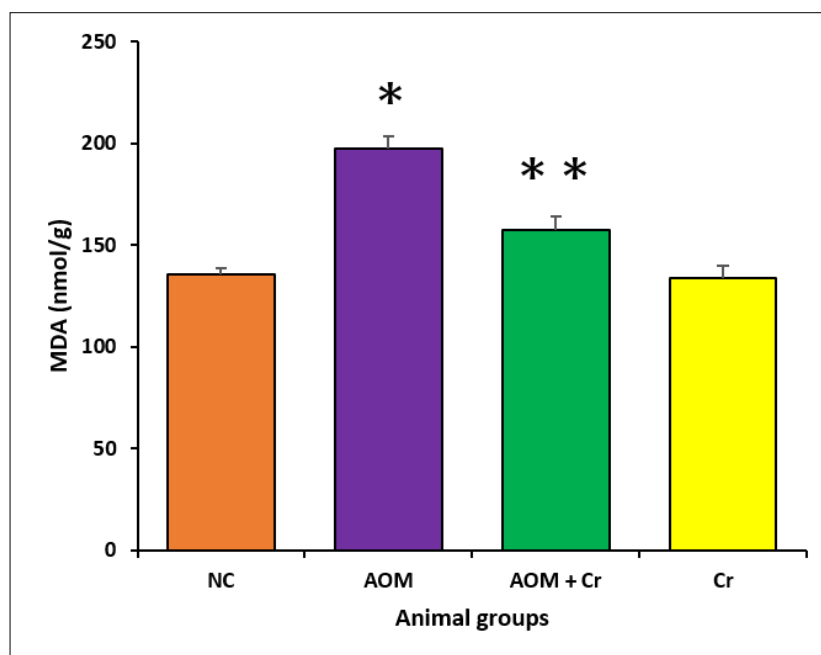


Fig. 2. The antioxidant enzymes levels in different experimental groups examined. The values signify the standard error of mean ($\pm$ SEM), with 8 rats per group. The treatment with AOM only led to increased levels of MDA as compared to NC animals. The level of MDA in Cr + AOM animal group was decreased ($p < 0.05$) as compared to AOM treated only. The statistical differences are represented with an asterisk (*) representing significance at ($p < 0.05$) as compared to the normal control group, while a double asterisk (**) indicates statistical significance when compared to the AOM-treated group alone ($p < 0.05$).

MDA: malondialdehyde; NC: normal control; AOM: azoxymethane administered orally; AOM + Cr: co-treatment with azoxymethane administered orally and curcumin; Cr: curcumin.

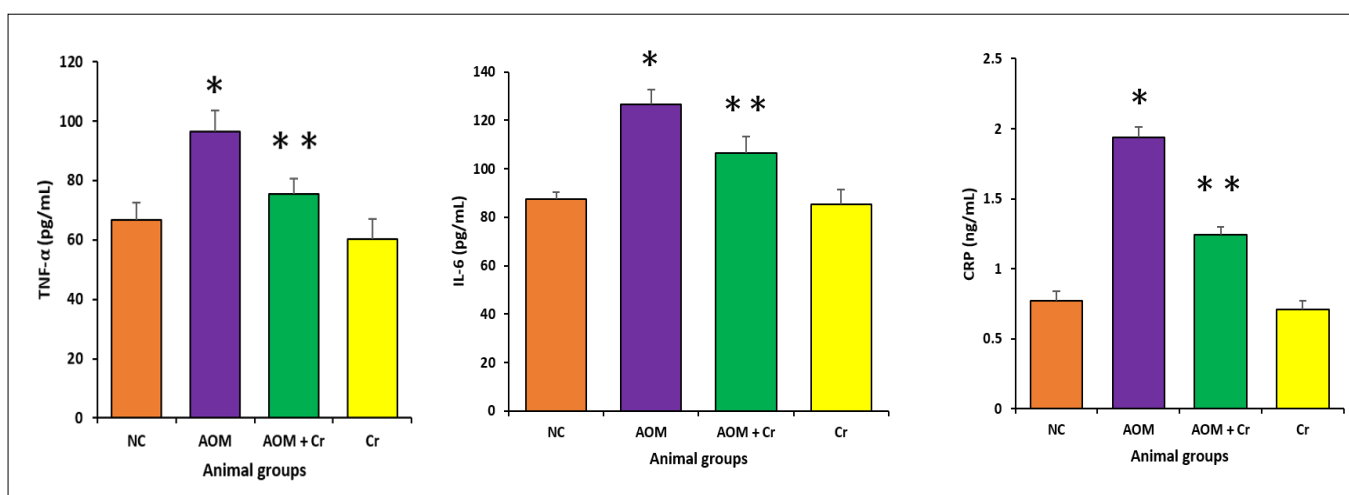


Fig. 3. Inflammatory marker levels were assessed in several experimental groups, each with eight rats. For each of the following groups, the data are shown as mean $\pm$ standard error of the mean (SEM). When compared to NC animals, the AOM-treatment only increased TNF- α , IL-6 and CRP levels. The increased level of these inflammatory markers in the Cr + AOM animal group was considerably decreased by the Cr therapy ($p < 0.05$). The statistical variations are shown with an asterisk (*) reported significance at ($p < 0.05$) in contrast to the typical control group and double asterisk (**) represents when compared with the AOM-treated group only ($p < 0.05$).

TNF - α : tumour necrosis factor - alpha; IL - 6: interleukin - 6; CRP: C - reactive protein; NC: normal control; AOM: azoxymethane administered orally; AOM + Cr: co-treatment with azoxymethane administered orally and curcumin; Cr: curcumin.

group (Fig. 3). Moreover, in the Cr + AOM treated group, inflammatory markers levels were significantly lower compared to AOM treated groups.

Effects of curcumin on colon tissue architecture

The potential effect of Cr on colon tissue architecture was measured through H & E staining in different experimental groups and the results were compared. The extent of inflammation, congestion and fibrosis was considered as no changes: 0; mild: 1; moderate: 2; and severe: 3. The animals in the control group revealed normal colon architecture. However, in the AOM treated group, it was revealed

that AOM causes various changes, including infiltration of inflammatory cells, congestion, hemorrhages and fibrosis, which are indicative of colon injury (Fig. 4). In the group that received both Cr and AOM, these alterations were found to be significantly less than in the AOM treated group. These findings, however, indicate that Cr has a colon protective effect by preserving colon tissue architecture.

Effects of curcumin on fibrosis

Masson trichrome and Sirius red staining were performed to assess the fibrosis in different experimental groups. Azoxymethane treated rats showed increased collagen deposition as noticed by blue color

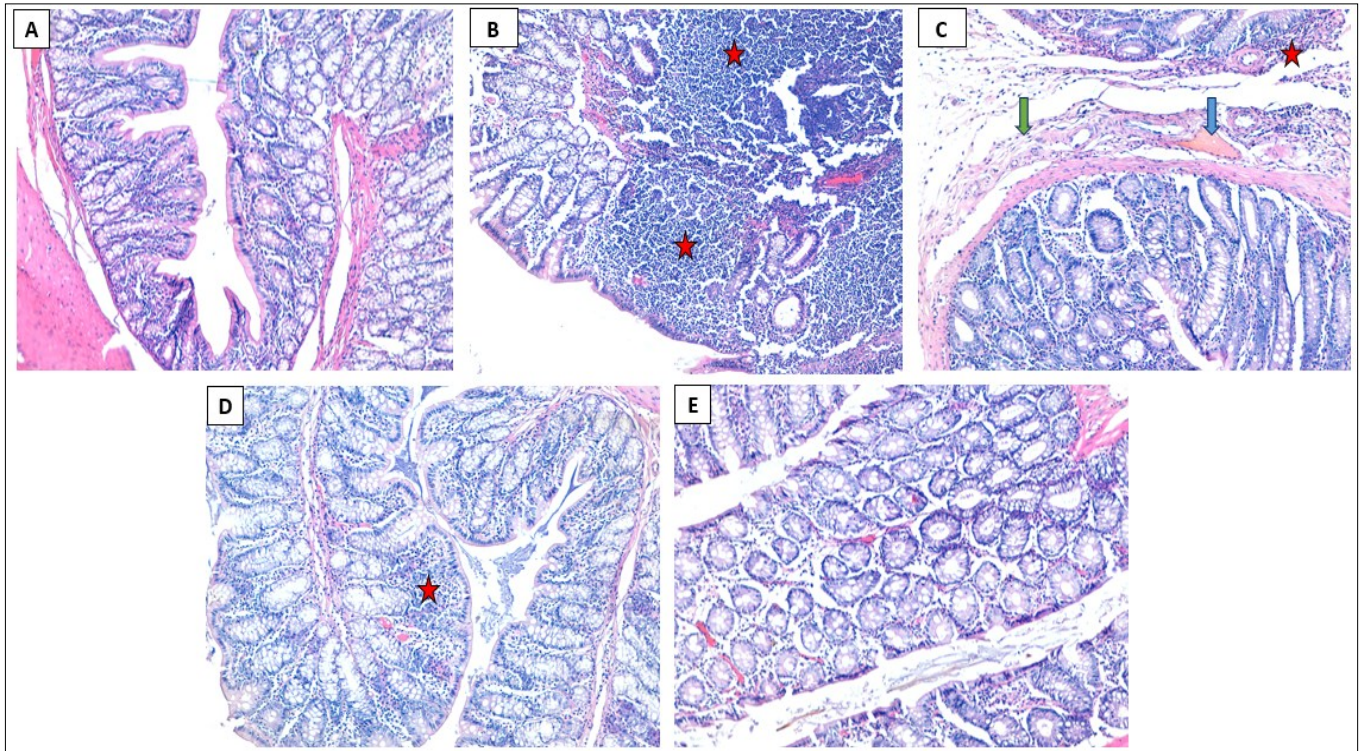


Fig. 4. Effect of curcumin (Cr) on colon tissues was evaluated using haematoxylin and eosin (H & E) staining. A: the colon's typical normal architecture was noticed in the control group controls; (B and C) the animal treated with azoxymethane administered orally (AOM) only exhibited inflammatory cell infiltration, dilated blood vessels and fibrosis; (D) administration of co-treatment with azoxymethane administered orally and curcumin (AOM + Cr) noticeably mitigated the tissue changes; (E) curcumin - only treatment group showed normal tissue architecture (scale bar = 100 μ m). Inflammatory cells are indicated by the red star, edema by the blue arrow and fibrosis by the green arrow.

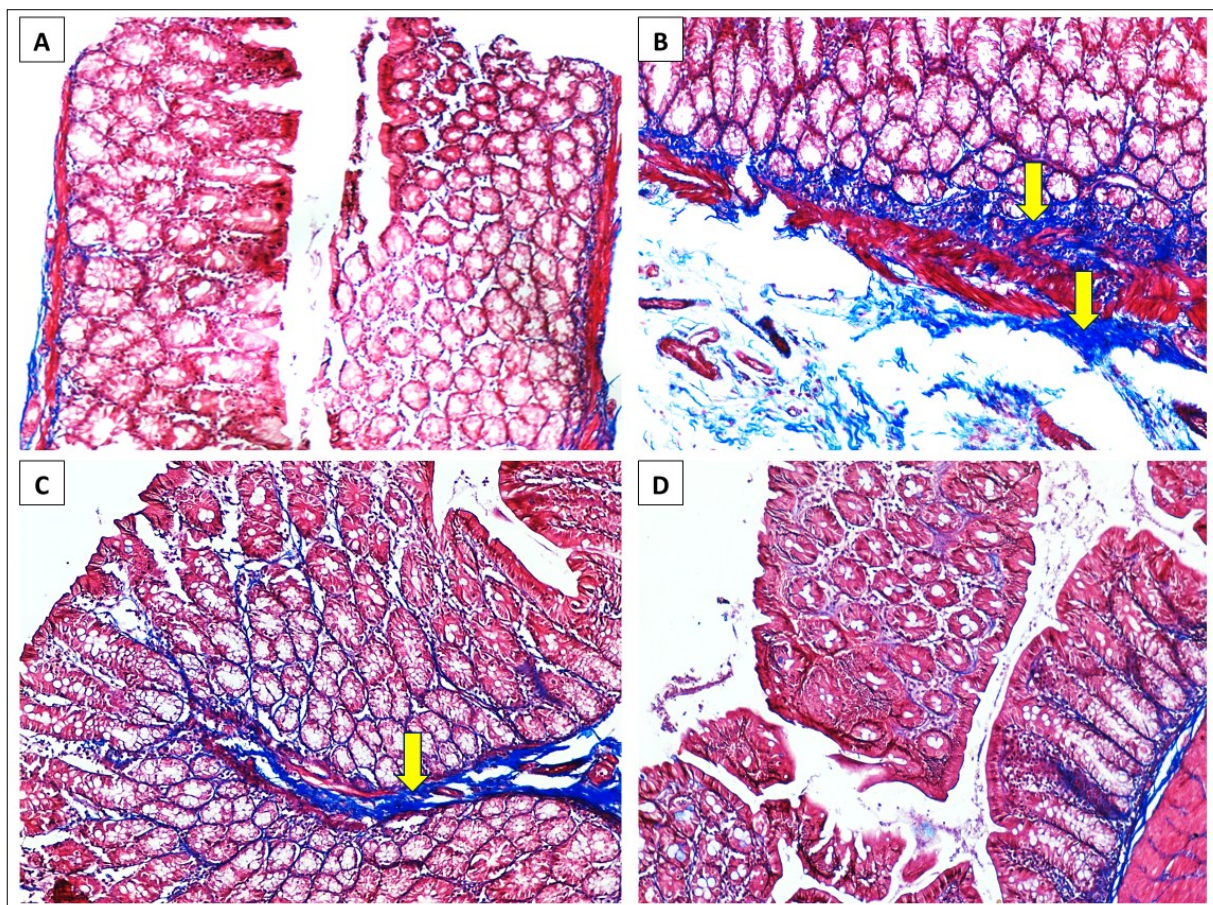


Fig. 5. Effect of curcumin (Cr) on colon tissues to evaluate the fibrosis using Masson trichrome staining. A: typical architecture of the colon in control of animals as the normal distribution of fibre; B: azoxymethane administered orally (AOM) only treated colon tissues showed deposition of fibrosis; C: treatment group (AOM rats treated with Cr) noticeably decreased the fibrosis; D: the animals in the Cr only group had normal fibre distribution (scale bar = 100 μ m). The yellow arrow shows collagen fibre.

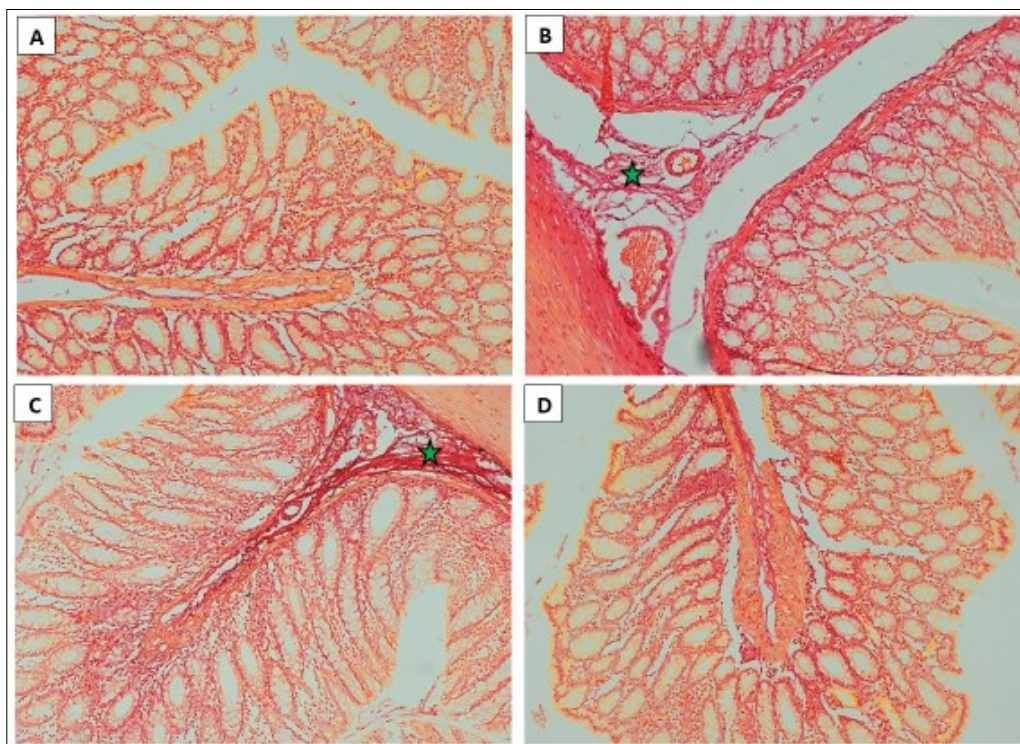


Fig. 6. Effect of curcumin (Cr) on colon tissues to evaluate the fibrosis using Sirius red staining. A: typical architecture of colon in control animals as normal distribution of fibre; B: azoxymethane administered orally (AOM) only treated colon tissues showed significant fibrosis; C: treatment group (AOM rats treated with Cr) noticeably mitigated the fibrosis as low fibrosis; D: only the Cr-treated animals had no or normal fibre dispersion (scale bar = 100 μm). The green star shows collagen fibre.

for Masson trichrome and red color for Sirius red staining. However, the AOM plus Cr group showed considerably lower fibre depositions (Fig. 5 and 6). There was no collagen deposition observed in the control group as well as the Cr only treated group.

Curcumin effect on the expression of interleukin-6 protein

The expression of IL - 6 protein was assessed in each experimental group using IHC staining and the findings were analysed accordingly.

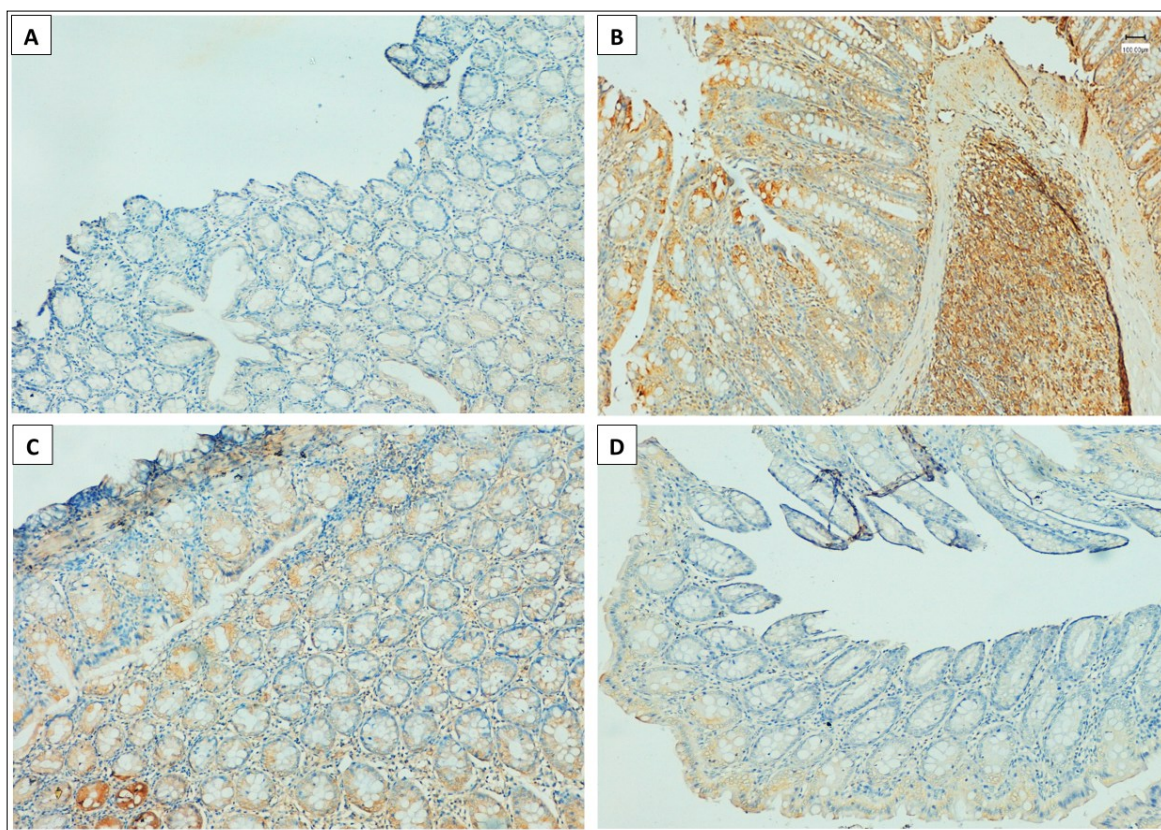


Fig. 7. Curcumin (Cr) effect on colon tissues to evaluate the interleukin - 6 (IL - 6) expression using immunohistochemistry (IHC) staining. A: in control animals, displays no expression; B: only azoxymethane administered orally (AOM)-treated colon tissues showed high levels of cytoplasmic IL - 6 protein expression; C: treatment group (AOM rats treated with Cr) decreased the expression; D: animal group treated with Cr only showed no expression (scale bar = 100 μm). The blue arrow shows cytoplasmic expression of IL-6 protein.

High IL - 6 expression was observed in the AOM treated group (Fig. 7), while no expression was detected in the control and Cr only groups. However, when Cr was administered along with AOM, the protein expression was lower in comparison to the AOM group (Fig. 7).

Discussion

Colonic diseases, such as colorectal malignancy as well as IBD, remain a major cause of mortality and morbidity, thus increasing the burden of this pathogenesis globally (24, 25). They are caused by a variety of factors, including inflammation, oxidative damage and alterations in cellular signalling pathways. Additionally, inflammation has been identified as a critical factor in the development of several pathologies, including neoplasia, which involves a variety of cellular and molecular mechanisms (26–28). Oxidative stress refers to an imbalance between the production and buildup of ROS in cells and tissues and the biological systems' ability to eliminate these reactive products (29). This condition has been recognised as a major factor contributing to the onset of pathogenesis. This study aims to determine the protective effects of Cr on colon injury by examining its role in reducing oxidative stress, inflammation and preserving the morphology of colon tissues. Curcumin is an essential part of many traditional herbal medicine systems, including Ayurvedic medicine, used to treat various illnesses (30). Antioxidant enzymes are components of an antioxidant defence system, which detoxifies ROS and thereby inhibits their harmful effects to prevent diseases. In this study, a substantial decrease in the activity of antioxidant enzymes and an increase in MDA level were observed in the animal group treated with AOM. However, rats treated with Cr in addition to AOM showed a significant increase in the levels of antioxidant enzymes and a decrease in the MDA level. A study based on a mouse model of IBD was performed to examine the role of Cr transdermal gel on oxidative stress and intestinal inflammation in interleukin - 10 knockout mice. Based on findings, it was reported that Cr transdermal gel showed a protective role against oxidative stress (31). Furthermore, another study based on lung injury reported that the treatment of Cr (50 mg / kg b.w.) in rats administered benzopyrene significantly increased the levels of antioxidant enzymes (23). Further, a study based on colitis reported that Cr treatment was related to improvement of macroscopic and microscopic colitis scores, reduced myeloperoxidase (MPO) activity and reduced MDA levels in acetic acid induced colitis (32). Inflammation plays a crucial role in various diseases, including colon pathogenesis. To examine the specific mechanism through which Cr reduces colonic inflammation, this study developed a rat model of colon injury to investigate its anti-inflammatory effects. In this study, the levels of inflammatory markers in AOM induced colon lesions were found to be substantially higher. Besides, Cr significantly reduced the levels of inflammatory markers. In this regard, previous studies have tested whether the addition of Cr or rutin to the diet would improve colonic inflammation in *mdr1a*^{-/-} mice. It was reported that Cr, but not rutin, meaningfully decreased histological signs of colonic inflammation in *mdr1a*^{-/-} mice (33). The anti-inflammatory potential of Cr was reported as colonic IL - 6 and TNF - α levels were significantly raised in the dextran sodium sulphate (DSS) group as compared to controls. Curcumin administration prevented substantial rises in IL - 6 and TNF - α expression (34). It was reported that Cr is a powerful targeted receptor-interacting protein 3 (RIP3) inhibitor with

anti-inflammatory and anti-necroptotic effects, maintains intestinal barrier function and efficiently improves colitis injury. Inflammatory cell infiltration, hemorrhages, congestion, fibrosis, blood vessel dilatation and edema are all signs of AOM induced colon damage, according to the current study. This change was dramatically reduced by the Cr plus AOM induced colon injury group. It was reported that Cr administration prevented DSS induced colon shortening, reduced loss of mucosal epithelium and goblet cells, improved inflammatory infiltration and overall decreased the histopathological score (35). Further, it was exhibited that histological examination of the distal colon tissue revealed that the DSS treatment caused the destruction of colonic architecture and strong infiltration of the inflammatory cells as compared to the normal morphology of colon tissue. While colon tissues from the Cr treated mice revealed mainly intact colon histology and reduced numbers of infiltrating cells (34). Based on the results, it is argued that Cr has a preventive effect against colon injury. It can also reduce the histological changes associated with AOM induced damage. In this study, it was found that treatment with AOM resulted in significant collagen deposition; however, this effect was reduced with Cr treatment. Sirius red staining indicated that fibrosis levels were higher in the colon of rats treated with AOM, while administration of Cr led to a reduction in fibrosis formation. Another study based on lung injury reported that rats in the control group displayed a normal lung architecture, in contrast to those with benzopyrene, which exhibited increased collagen deposition. Additionally, pretreatment with Cr significantly reduced collagen accumulation, thereby helping to maintain a nearly normal lung architecture. Cytokines are important pathophysiological aspects that control the initiation, progression and, in some cases, resolution of IBD inflammation (36). An immunohistochemical investigation indicated that the AOM group showed high IL - 6 protein expression, whereas the normal control group observed no protein expression. Furthermore, Cr treated groups showed reduced IL - 6 protein expression and this study suggests that Cr works as an anti-inflammatory drug by lowering IL - 6 protein expression. These findings suggested that Cr might reduce inflammation by inhibiting IL - 6 from being expressed in colon lesions caused by AOM.

Conclusion

This study demonstrates that curcumin (Cr) effectively protects against azoxymethane (AOM) induced colon injury by significantly reducing oxidative stress, suppressing inflammatory markers (tumour necrosis factor - α (TNF - α), C - reactive protein (CRP) and interleukin - 6 (IL - 6)) and preserving colon tissue architecture. These findings highlight the potential of Cr as a natural therapeutic agent for the prevention and management of colon related pathogenesis. Importantly, the study provides mechanistic insight into the protective effects of Cr through modulation of oxidative stress and inflammatory pathways. Future studies should focus on validating these findings in clinical settings, investigating the molecular signalling pathways underlying Cr action in greater detail and evaluating long term efficacy, optimal dosing strategies and bioavailability improvements. Additionally, comparative studies with existing therapeutic agents are warranted to better define its translational potential.

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Authors' contributions

AYB and AHR contributed to the methodology and conception. SAA, HOA, MAA and SAE conducted data analysis and investigation. WMA and FFAA prepared the original draft. AHM reviewed and edited the content and AYB provided supervision. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: Ethical approval was obtained with number QU-J-UG-2-2025-54308.

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