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A histological study to investigate the effects of astaxanthin on aspirin-induced gastric ulceration in rats

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Abstract

Objective

Gastric ulcer disease remains a significant global health concern, affecting millions worldwide, particularly due to common risk factors such as chronic nonsteroidal anti-inflammatory drug (NSAID) use. Aspirin, a widely used NSAID, compromises gastric mucosal integrity by inhibiting prostaglandin synthesis and increasing oxidative stress and inflammation, leading to ulcer formation. Astaxanthin, a naturally occurring carotenoid with potent antioxidant, anti-inflammatory, and anti-apoptotic properties, has gained attention for its potential gastrointestinal protective effects. This study aimed to evaluate the therapeutic potential of astaxanthin in the treatment and recovery of aspirin-induced gastric ulcers. By assessing histological parameters in an experimental rat model, we investigated whether astaxanthin alone or in combination with omeprazole offers superior mucosal protection and healing compared to conventional treatment.

Materials and Methods

Female albino rats were housed under controlled conditions (22 ± 2°C, 12-hour light/dark cycle) with free access to food and water. Gastric ulcers were induced in all groups, except for the negative control, via oral administration of aspirin (100 mg/kg). The ulcerated animals were then divided into seven groups (n = 10): Control (C), receiving distilled water and standard feed; Positive Control (T1), with aspirin-induced ulcers and no treatment; T2, treated with omeprazole (20 mg/kg); T3, treated with astaxanthin (50 mg/kg); T4, treated with astaxanthin (75 mg/kg); T5, treated with omeprazole (20 mg/kg) + astaxanthin (50 mg/kg); and T6, treated with omeprazole (20 mg/kg) + astaxanthin (75 mg/kg).

Results

Histological analysis revealed that rats treated with astaxanthin (50 mg/kg or 75 mg/kg) exhibited well-preserved epithelial layers, intact gastric glands, and normal mucous neck and parietal cells, indicating substantial mucosal recovery. In contrast, the omeprazole-treated group displayed mild pathological alterations, suggesting incomplete healing. Combination therapy with astaxanthin and omeprazole further enhanced mucosal protection, showing fewer histopathological changes compared to omeprazole alone. These findings suggest that astaxanthin promotes superior gastric mucosal healing following aspirin-induced ulceration, likely due to its potent antioxidant and anti-inflammatory properties.

Conclusion

This study demonstrates that astaxanthin significantly protects against and enhances gastric mucosal healing in aspirin-induced gastric ulcers. Compared to omeprazole, astaxanthin exhibited superior histological preservation, highlighting its potential as an effective therapeutic agent. The observed antiulcer effects may be attributed to astaxanthin's antioxidant, anti-inflammatory, and anti-apoptotic properties. These findings support astaxanthin as a promising candidate for gastric ulcer management, either alone or in combination with conventional treatments. Further research is warranted to elucidate its precise mechanisms and clinical applicability.

Keywords: albino rats, aspirin, astaxanthin, gastric ulcer, omeprazole

Paper Type: Research Paper.

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Introduction

Peptic ulcer disease (PUD) is a chronic condition affecting approximately 10% of the global population. It develops due to an imbalance between the aggressive factors of gastric acid

secretion and the protective mechanisms of the mucosal barrier (Narayanan et al., 2018). One of the primary causes of peptic ulcers is the excessive use of non-steroidal anti-inflammatory drugs (NSAIDs), including aspirin, which is widely used for its anti-inflammatory properties and to prevent blood clot formation. NSAID use increases gastric acid secretion while reducing mucosal blood flow, leading to mucosal damage and predisposing individuals to *Helicobacter pylori* (*H. pylori*) infection (Lanas & Chan, 2017; Ayoub et al., 2018). *H. pylori* infection is a major risk factor for gastric tissue damage, particularly affecting the mucous membrane (Lanas et al., 2015; Sadoud et al., 2023). Current treatments for peptic ulcers, such as proton pump inhibitors (PPIs) and histamine H2 receptor antagonists, have been associated with adverse effects, high recurrence rates, and drug interactions (Akahoshi et al., 2005; Chiou et al., 2005). As a result, there is growing interest in natural alternatives, particularly phytobiotics and medicinal plants, which have demonstrated antimicrobial, antioxidant, and therapeutic properties. The use of these natural compounds in animal feed has shown multiple benefits, including improved zootechnical performance, disease suppression (Amirteymoori et al., 2021; Mohammadabadi et al., 2023), antimicrobial and antioxidant activities (Hajalizadeh et al., 2019; Jafari Ahmadabadi et al., 2023), hypocholesterolemic effects, enhanced digestive enzyme activity, and improved liver function (Safaei et al., 2022; Shokri et al., 2023; Mohammadabadi et al., 2024). Additionally, studies indicate that dietary supplementation with these bioactive compounds enhances feed consumption, improves feed conversion ratios, and increases carcass yield, while also providing cytoprotective effects against various diseases, including peptic ulcer disease (Vahabzadeh et al., 2020; Shokri et al., 2023). Effective medical management of peptic ulcers involves reducing gastric acidity by inhibiting or neutralizing acid secretion, eliminating environmental risk factors such as NSAID use and smoking, and promoting mucosal protection through prostaglandin analogs and barrier-forming agents (Ahluwalia et al., 2019). However, conventional drugs often cause adverse effects, including shortness of breath, irregular heartbeat, dry mouth, and headaches, leading to a shift toward safer, natural alternatives with fewer side effects (Seo et al., 2012). Astaxanthin, a naturally occurring red carotenoid pigment, is primarily found in marine organisms such as microalgae, fish, and shrimp. Since humans cannot synthesize carotenoids, they must obtain them through dietary sources (Higuera-Ciapara et al., 2006). Astaxanthin belongs to the xanthophyll class of oxygenated carotenoids, which are derived from lycopene in plants. In aquatic ecosystems, microalgae synthesize astaxanthin, which is then consumed by zooplankton, insects, and crustaceans, ultimately transferring the pigment to fish and other marine organisms, contributing to their pigmentation (Pashkow et al., 2008).

Astaxanthin is recognized as a potent antioxidant due to its unique chemical structure, which enables it to neutralize reactive oxygen species (ROS). Studies have demonstrated that

astaxanthin's antioxidant capacity is ten times greater than that of other carotenoids such as lutein, zeaxanthin, and beta-carotene, and 100 times stronger than alpha-tocopherol (Kidd, 2011). Moreover, research has highlighted its anti-inflammatory, anti-apoptotic, and anti-cancer properties, as well as its potential as a nutritional supplement in food, pharmaceuticals, and animal feed (Yamashita et al., 2013). However, to our knowledge, no comprehensive study has investigated the therapeutic potential of astaxanthin in the treatment of gastric ulcers. In this study, we aimed to evaluate the efficacy of astaxanthin in the treatment, healing, and recovery of aspirin-induced gastric ulcers. By assessing its effects on histological parameters in an experimental rat model, we sought to determine whether astaxanthin alone or in combination with omeprazole provides superior protection and recovery compared to conventional treatment. The findings of this study may contribute to the development of novel, more effective therapeutic strategies for managing peptic ulcer disease.

Material and Methods

Preparation of laboratory animals: This study was conducted in the animal facility affiliated with the Department of Life Sciences at the College of Education, University of Al-Qadisiyah. A total of 70 female Albino rats, weighing between 180 and 250 g, were obtained from Saddat Al-Hindiyah, Babylon Governorate. The animals were housed in plastic cages covered with tightly meshed metal lids and lined with clean wood shavings. The cages were cleaned and sterilized two to three times per week. The animals were maintained under controlled laboratory conditions, including a light/dark cycle of 11 hours of light and 13 hours of darkness, adequate ventilation, and a temperature range of 22–25°C. The rats were acclimatized for two weeks before the experiment. They were provided with standard commercial feed and water ad libitum throughout the study.

Preparation of experimental solutions-aspirin solution: Aspirin powder was used to prepare a solution with a final concentration of 100 mg/kg body weight. This was achieved by dissolving 1 g of aspirin powder in 100 mL of 1% carboxymethyl cellulose. Each rat received 1 mL per 100 g of body weight via oral administration (Voelker & Hammer, 2012).

Omeprazole solution: Omeprazole was used as a treatment for aspirin-induced gastric ulcers. A solution of 20 mg/kg was prepared by dissolving 2.0 g of omeprazole in 100 mL of distilled water, yielding a concentration of 2 mg/mL. Each rat received 1 mL per 100 g of body weight (Weng et al., 2024).

Astaxanthin solution: Astaxanthin powder was obtained from Tic.San.Sis Akvaryum Kimya. Two concentrations, 50 mg/kg and 75 mg/kg body weight, were prepared by dissolving

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the powder in distilled water. Each rat received a daily oral dose of 1 mL using a specialized dosing instrument (Erbaş et al., 2024).

Experimental design-induction of gastric ulcers: Gastric ulcers were induced in 70 female Albino rats by administering aspirin at a dose of 100 mg/kg for seven consecutive days following a 14-hour fasting period. The occurrence of gastric ulcers was confirmed by measuring the pH of gastric juice and comparing it with that of the control group.

Animal grouping and treatment: The rats were randomly divided into seven groups, each consisting of 10 animals:

1. Negative control group (C): Received only distilled water and standard feed throughout the 21-day experiment.
2. Positive control group (T1): Gastric ulcers were induced with aspirin but left untreated for 21 days.
3. Omeprazole treatment group (T2): Gastric ulcers were induced, and the rats were treated with omeprazole (20 mg/kg) for two weeks following ulcer induction.
4. Astaxanthin (50 mg/kg) treatment group (T3): Gastric ulcers were induced, and the rats were treated with astaxanthin (50 mg/kg) for two weeks following ulcer induction.
5. Astaxanthin (75 mg/kg) treatment group (T4): Gastric ulcers were induced, and the rats were treated with astaxanthin (75 mg/kg) for two weeks following ulcer induction.
6. Combined Omeprazole (20 mg/kg) + Astaxanthin (50 mg/kg) treatment group (T5): Gastric ulcers were induced, and the rats were treated with both omeprazole (20 mg/kg) and astaxanthin (50 mg/kg) simultaneously for two weeks following ulcer induction.
7. Combined Omeprazole (20 mg/kg) + Astaxanthin (75 mg/kg) treatment group (T6): Gastric ulcers were induced, and the rats were treated with both omeprazole (20 mg/kg) and astaxanthin (75 mg/kg) simultaneously for two weeks following ulcer induction.

Animal sacrifice and sample collection: At the end of the experiment, the rats were sacrificed by chloroform anesthesia. The abdominal cavity was opened, and the stomach was excised and dissected along the greater curvature. The pH of the gastric juice was measured using a pH meter, after which the contents were emptied and rinsed with normal saline. The stomachs were examined for ulcer formation using a microscope, and the severity of the ulcers was recorded.

Histological sample preparation and analysis: Histological evaluation was performed using light microscopy. Stomach tissue samples were fixed in 10% buffered formalin, dehydrated in graded ethanol, and embedded in paraffin. Sections were cut perpendicular to the stomach wall and stained with hematoxylin and eosin (H&E). Images were captured at 10X and 40X magnification using a Leica DM3000 LED microscope (Chesnick et al., 2010).

Results and discussion

Macroscopic examination of stomach tissue: Gross examination of gastric ulcers in the positive control group (T1) during the first and second weeks confirmed the successful induction of gastric ulcers by aspirin. This was evidenced by the presence of multiple ulcers with bleeding bands dispersed across the stomach lining, along with signs of inflammation. In contrast, the negative control group (C) exhibited an intact gastric mucosa, indicating the absence of ulcers. Additionally, omeprazole and astaxanthin (administered individually or in combination) demonstrated efficacy in treating gastric ulcers, as evidenced by a reduction in bleeding and inflammation, particularly after the second week of treatment, as shown in Figure 1.

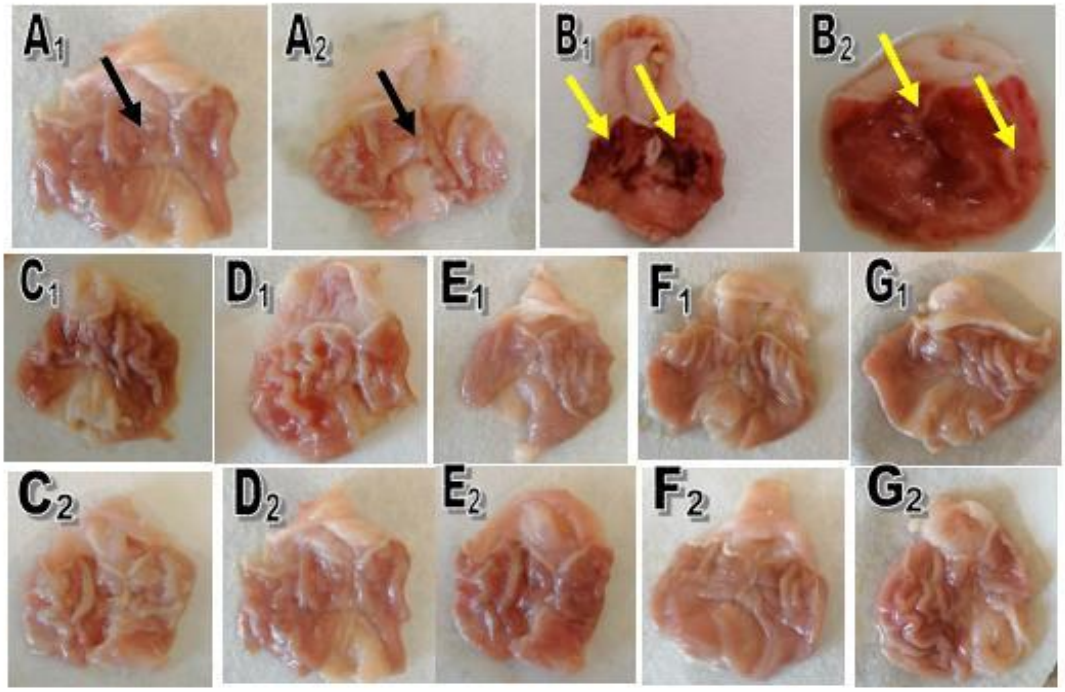

Figure 1. Macroscopic examination of gastric tissue at the end of the first and second weeks of the experiment. Sections A1 and A2 represent group C (black arrow), showing normal gastric tissue in both weeks. Sections B1 and B2 represent group T1 (yellow arrow), indicating multiple ulcers and bleeding bands visible to the naked eye, particularly during the first and second weeks. Sections C1 and C2 correspond to group T2, D1 and D2 to group T3, E1 and E2 to group T4, F1 and F2 to group T5, and G1 and G2 to group T6, all of which exhibited no gastric ulcers in the first and second weeks of the experiment

Microscopic examination of stomach tissues: Microscopic examination of the gastric tissue in the negative control group (C) revealed normal stomach structures, including the

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muscularis mucosae, gastric glands, gastric pits, and columnar epithelium. In contrast, histopathological analysis of tissue sections from the positive control group (T1) at the end of the first week of treatment showed pathological changes characterized by the presence of the muscularis mucosae, numerous mucosal glands, congestion, and destruction of the columnar epithelium. By the end of the second week, the pathological changes in group T1 had worsened, with increased inflammatory cell infiltration, hyperplasia in the muscularis mucosae, and further destruction of the columnar epithelial tissue, as shown in Figure 2 (A, B, C, D). In the esomeprazole-treated group (T2, 20 mg/kg), microscopic examination at the end of the first week revealed mild histopathological changes in the epithelial and muscularis mucosae layers, along with slight necrosis in the muscularis mucosae. However, by the end of the second week, histological analysis showed significant recovery, with normal epithelial tissue, the presence of gastric pits, and reconstruction of gastric gland cells within the muscularis mucosae. Additionally, the presence of chief and parietal cells was observed, as shown in Figure 3 (A, B, C, D). Similarly, microscopic examination of tissue sections from the astaxanthin-treated group (T3, 50 mg/kg) at the end of the first week revealed minor pathological changes in the epithelial layer, along with evidence of gastric gland cell repair in the muscularis mucosae. However, some gastric gland destruction and slight necrosis in the muscularis mucosae were also observed. By the end of the second week, the tissue sections showed a substantial restoration of normal histological structure, including the epithelial layer, gastric glands, mucosal neck cells, parietal cells, and chief cells. However, slight necrosis in the muscularis mucosae persisted, as shown in Figure 4 (A, B, C, D).

Microscopic examination of stomach tissue sections from the astaxanthin-treated group (T4, 75 mg/kg) at the end of the first week revealed normal gastric architecture, with restored epithelial layer integrity, well-formed gastric glands in the mucosal layer, and normal mucosal neck cells and parietal cells. Chief cells within the gastric glands were also observed. By the end of the second week, significant improvement was evident, with the epithelial layer and gastric glands appearing normal in the mucosal layer, along with a clearly defined muscularis mucosae, as shown in Figure 5 (A, B, C, D). Histological examination of stomach tissue from the combination treatment group (T5, astaxanthin 50 mg/kg + omeprazole 20 mg/kg) at the end of the first week demonstrated reformation of epithelial cells and repair of gastric glands in the muscularis mucosae, with an overall normal tissue appearance. Parietal and chief cells within the gastric glands were also evident. By the second week, the stomach tissue exhibited a normal histological structure, with a well-defined epithelial layer, properly formed gastric glands in the mucosal and muscularis mucosae, and a notable improvement in parietal and chief cells, as shown in Figure 6 (A, B, C, D).

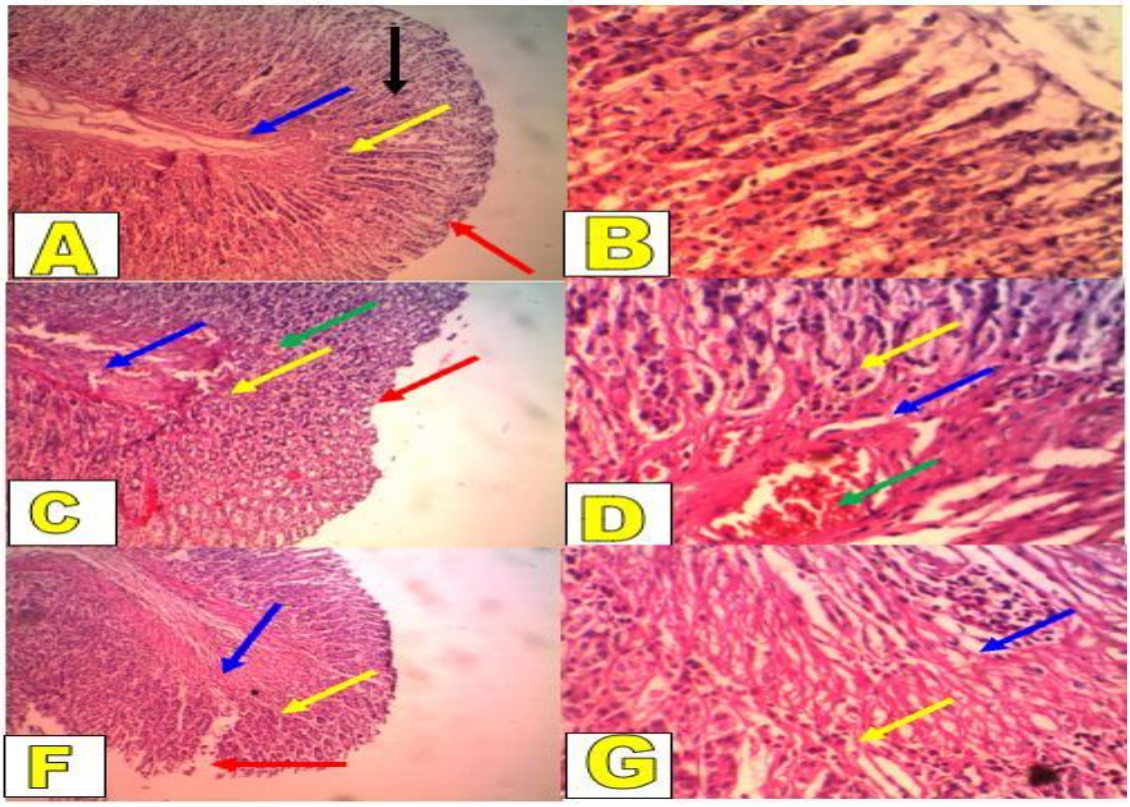

Figure 2. Histological sections of stomach tissue in control groups. Sections A (10X) and B (40X) show stomach tissue from the negative control group at the end of the experiment, displaying normal gastric architecture. The blue arrow indicates the muscularis mucosae, the yellow arrow marks the gastric glands, the black arrow highlights the gastric pits, and the red arrow denotes the columnar epithelium. Sections C (10X) and D (40X) depict stomach tissue from the positive control group (T1) at the end of the first week, revealing histopathological changes, including alterations in the muscularis mucosae, gastric glands, blood congestion, and destruction of the columnar epithelium. Sections F (10X) and G (40X) illustrate stomach tissue from the positive control group (T1) at the end of the second week, showing more severe histopathological changes, including inflammatory cell infiltration, hyperplasia of the muscularis mucosae, and further destruction of the columnar epithelium.

Similarly, histological examination of stomach tissue from the combination treatment group (T6, astaxanthin 75 mg/kg + omeprazole 20 mg/kg) at the end of the first week showed significant tissue improvement, with epithelial layer restoration and gastric gland repair in the mucosal muscularis layer and mucosal neck cells. Normal tissue structure was observed in the parietal cell layer and chief cells within the gastric glands. By the end of the second week, the histological examination confirmed normal gastric architecture, including a well-preserved epithelial cell

layer, gastric glands, and mucosal muscular cells located beneath the gastric glands. Additionally, the parietal cell layer and chief cells exhibited normal morphology, as shown in Figure 7 (A, B, C, D).

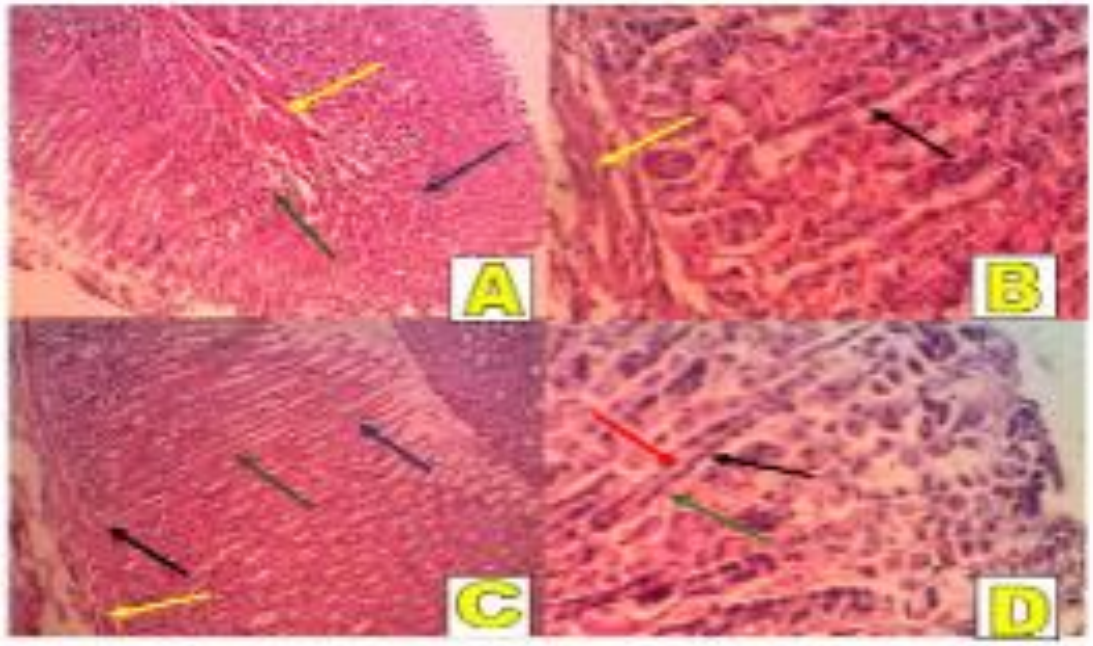

Figure 3. Histological sections of stomach tissue from the omeprazole-treated group (T2, 30 mg/kg). Sections A (10X) and B (40X) show histopathological changes at the end of the first week, with the blue arrow indicating inflammatory cell infiltration, the yellow arrow marking hyperplasia of the muscularis mucosae, and the red arrow highlighting destruction of the columnar epithelium. Sections C (10X) and D (40X) depict stomach tissue at the end of the second week, showing normal gastric architecture with only mild necrosis

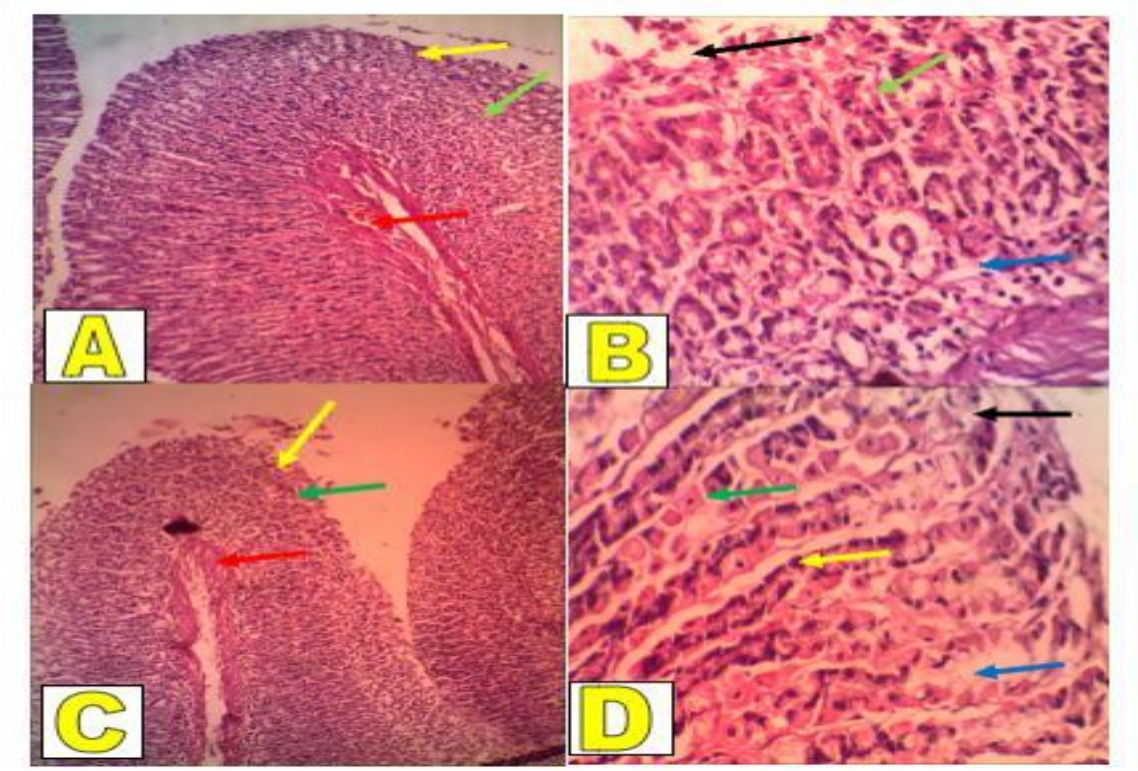

Figure 4. Histological sections of stomach tissue from the astaxanthin-treated group (T3, 50 mg/kg). Sections A (10X) and B (40X) show mild histopathological changes in the first week, with the yellow arrow indicating the epithelial layer, the green arrow marking the gastric gland, the red arrow denoting the muscularis mucosae, the black arrow highlighting the destruction of some gastric glands, and the blue arrow indicating mild necrosis. Sections C (10X) and D (40X) show stomach tissue from the second week, demonstrating restored normal gastric architecture

Despite the availability of various therapeutic options—including proton pump inhibitors, H_2 receptor antagonists, antacids, and mucosal protectants—gastric ulcers remain among the most prevalent gastrointestinal conditions, affecting approximately 5–10% of the global population (Yibirin et al., 2021). The pathogenesis of gastric ulcers is multifactorial, primarily driven by an imbalance between aggressive factors—such as excessive gastric acid secretion, pepsin activity, and reactive oxygen species (ROS)—and gastroprotective mechanisms, including the mucosal barrier, endogenous antioxidants, bicarbonate secretion, and cellular regeneration (Alves Araujo de Lima et al., 2022). If left untreated, gastric ulcers can lead to severe complications, including gastrointestinal bleeding, perforation, and an increased risk of gastric cancer. The most common risk factors for gastric ulcers include *Helicobacter pylori* infection and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) (Xie et al., 2022).

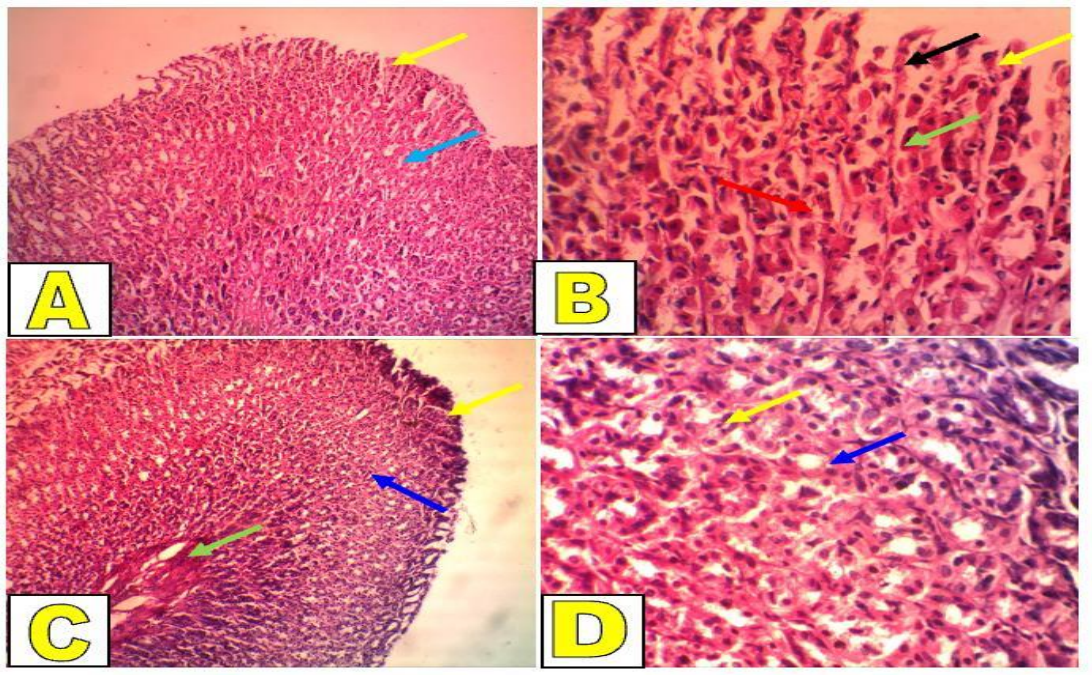

Figure 5. Histological sections of stomach tissue from the astaxanthin-treated group (T4, 75 mg/kg). Sections A (10X) and B (40X) at the end of the first week show normal gastric tissue, with the yellow arrow indicating the epithelial layer, the blue arrow marking the gastric glands, the black arrow highlighting the mucous neck cells, the green arrow denoting the parietal cells, and the red arrow indicating the chief cells within the gastric glands. Sections C (10X) and D (40X) at the end of the second week also show normal stomach tissue

Aspirin, a widely used NSAID, has been shown to induce gastric injury through prostaglandin-dependent mechanisms, which involve neutrophil infiltration and increased ROS production, ultimately leading to gastric mucosal damage and apoptosis (Musumba et al., 2009). Consistent with previous studies (Musumba et al., 2009; Seo et al., 2012; Ahluwalia et al., 2019), our findings demonstrate that aspirin-induced gastric damage is characterized by significant pathological alterations, including the destruction of columnar epithelial tissue, inflammatory cell infiltration, and hyperplasia of the mucosal muscular layer (Figure 1). Astaxanthin, a carotenoid with potent antioxidant properties, has gained recognition as a nutraceutical and has been approved by the U.S. Food and Drug Administration (FDA) as a dietary supplement (Aly et al., 2024). Studies have demonstrated its strong anti-inflammatory, anti-apoptotic, and antioxidative effects, which surpass those of vitamin E and beta-carotene (Fakhri et al., 2018). Our histological analysis revealed that treatment with astaxanthin (75 mg/kg) in combination with omeprazole (20 mg/kg) (T6) resulted in significant improvement in gastric tissue as early as the first week of treatment. Restoration of the epithelial layer, repair of gastric glands, and regeneration of mucosal

neck and mucosal muscular cells were observed. By the second week, histological examination confirmed the presence of normal gastric tissue, indicating complete recovery. Our study suggests that astaxanthin exerts a gastroprotective effect against aspirin-induced ulcers, either comparable to or superior to omeprazole alone. Furthermore, the combination of astaxanthin and omeprazole demonstrated an accelerated and enhanced therapeutic effect (Figures 2–6). These findings align with previous studies reporting that astaxanthin protects the gastric mucosa and reduces deep ulcerations, with effects comparable to those of omeprazole pre-treatment (Fakhri et al., 2018).

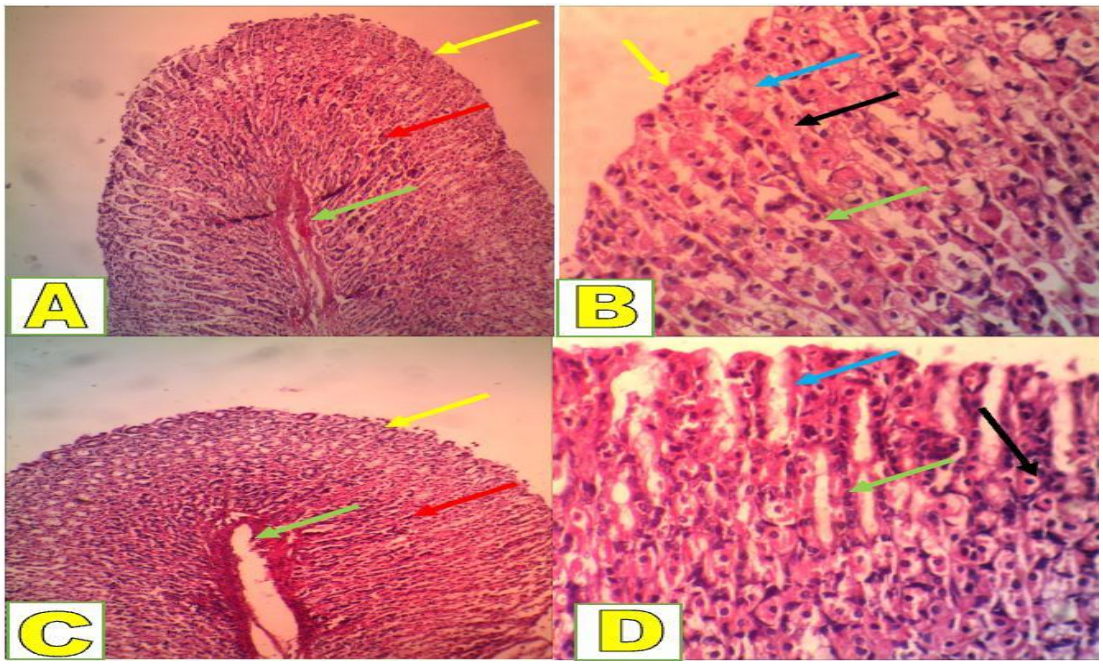

Figure 6. Histological sections of stomach tissue from the group treated with astaxanthin (50 mg/kg) and omeprazole (20 mg/kg) (T5). Sections A (10X) and B (40X) at the end of the first week, as well as sections C (10X) and D (40X) at the end of the second week, show normal stomach tissue. The yellow arrow indicates the epithelial layer, the red arrow marks the gastric glands, and the green arrow highlights the muscularis mucosae

The therapeutic efficacy of astaxanthin may be attributed to its broad spectrum of antioxidant activities, including singlet oxygen quenching, free radical scavenging, and inhibition of lipid peroxidation, which collectively help maintain cellular membrane integrity (Fakhri et al., 2018). Additionally, astaxanthin has been reported to enhance endogenous antioxidant defense mechanisms by increasing intracellular protective molecules that mitigate oxidative cell damage (Yin et al., 2021).

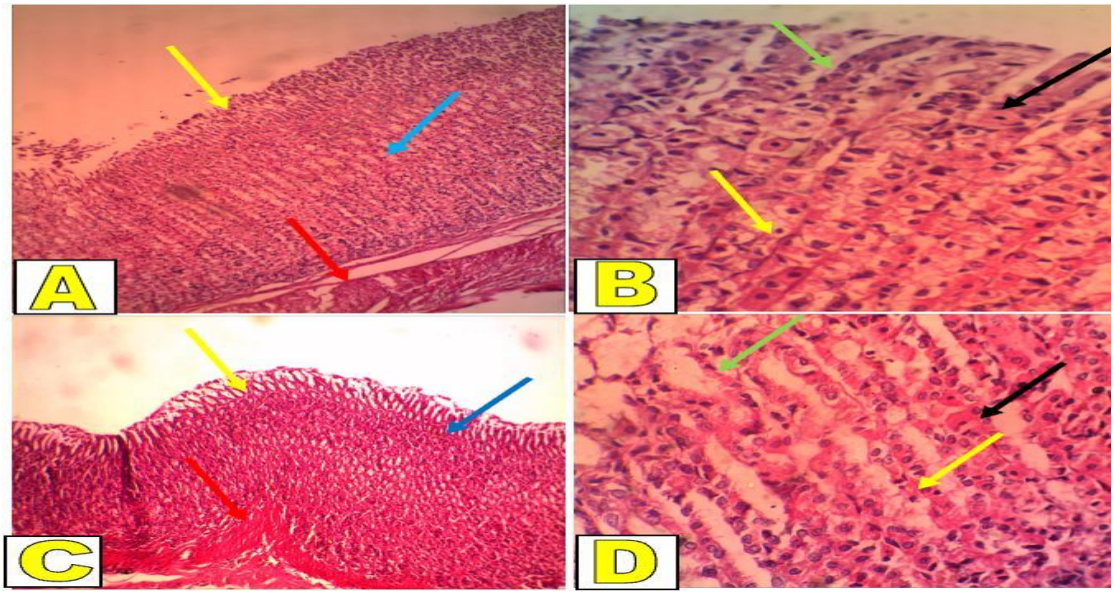

Figure 7. Histological sections of stomach tissue from the group treated with astaxanthin (75 mg/kg) and omeprazole (20 mg/kg) (T6). Sections A (10X) and B (40X) at the end of the first week, as well as sections C (10X) and D (40X) at the end of the second week, show normal gastric tissue. The yellow arrow indicates the epithelial layer, the blue arrow marks the gastric glands, and the red arrow highlights the muscularis mucosae. Additionally, the green arrow indicates the mucous neck cells, the black arrow marks the parietal cells, and the yellow arrow also denotes the chief cells within the gastric glands

Beyond its antioxidant potential, astaxanthin exhibits broad immunomodulatory effects across various disease models (Nishida et al., 2021). Notably, it has been shown to inhibit the activation of nuclear factor kappa B (NF-κB), a key transcription factor that regulates the expression of multiple pro-inflammatory mediators. In agreement with previous studies, our findings suggest that astaxanthin treatment significantly suppressed NF-κB signaling in gastric tissue, further supporting its gastroprotective effects through anti-inflammatory mechanisms (Lawrence, 2009; Aly et al., 2024).

Conclusion: Our study demonstrates that aspirin significantly increases the risk of gastric ulcers in mice, while astaxanthin exhibits potent anti-ulcerogenic properties. The combination of astaxanthin and omeprazole provided an enhanced and faster therapeutic effect, promoting gastric tissue repair and recovery. However, further mechanistic studies are warranted to identify the key molecular pathways underlying these protective effects. Specifically, a comprehensive analysis of inflammatory and apoptotic mediators may provide deeper insights into the molecular mechanisms by which astaxanthin mitigates gastric ulceration and facilitates mucosal healing.

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Conflict of Interest: There is no conflict of interest

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مطالعه بافت شناسی برای بررسی اثرات آستاگزانتین بر زخم معده ناشی از آسپرین در موش صحرایی

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چکیده

هدف: بیماری زخم معده همچنان یک نگرانی مهم بهداشتی جهان است و میلیون‌ها نفر را در سراسر جهان، به ویژه به دلیل عوامل خطر رایج مانند استفاده از داروهای ضد التهابی غیراستروئیدی مزمن (NSAID) تحت تاثیر قرار می‌دهد. آسپرین، یک NSAID است که به طور گسترده مورد استفاده قرار می گیرد و با مهار سنتز پروستاگلاندین و افزایش استرس اکسیداتیو و التهاب، یکپارچگی مخاط معده را به خطر می اندازد و منجر به تشکیل زخم می‌شود. آستاگزانتین، یک کاروتنوئید طبیعی با خواص آنتی اکسیدانی، ضد التهابی و ضد آپوپتوز قوی است که به دلیل اثرات بالقوه محافظتی دستگاه گوارش مورد توجه قرار گرفته است. این مطالعه با هدف ارزیابی پتانسیل درمانی آستاگزانتین در درمان و بهبود زخم معده ناشی از آسپرین انجام شد. با ارزیابی پارامترهای بافت شناسی در یک مدل موش آزمایشگاهی، بررسی شد که آیا آستاگزانتین به تنهایی یا در ترکیب با امپرازول در مقایسه با درمان معمولی محافظت و بهبودی بهتری از مخاط ارائه می‌دهد.

مواد و روش‌ها: موش‌های صحرایی ماده تحت شرایط کنترل شده (۲±۲۲ درجه سانتی گراد، چرخه نور/تاریکی ۱۲ ساعت) با دسترسی آزاد به غذا و آب قرار گرفتند. زخم معده در همه گروه‌ها به جز گروه کنترل منفی از طریق تجویز خوراکی آسپرین (mg/kg ۱۰۰) ایجاد شد. حیوانات سپس به هفت گروه (۱۰ تایی) تقسیم شدند: کنترل (C)، دریافت آب مقطر و خوراک استاندارد؛ کنترل مثبت (T1)، با زخم‌های ناشی از آسپرین و بدون درمان؛ T2، تحت درمان با امپرازول (mg/kg ۲۰)؛ T3، تحت درمان با آستاگزانتین

(mg/kg ۵۰)؛ T4، تحت درمان با آستاگزانتین (mg/kg ۷۵)؛ T5، تحت درمان با امپرازول (mg/kg ۲۰) + آستاگزانتین (mg/kg ۵۰)؛ و T6، تحت درمان با امپرازول (mg/kg ۲۰) + آستاگزانتین (mg/kg ۷۵).

نتایج: تجزیه و تحلیل بافت‌شناسی نشان داد که موش‌های تحت درمان با آستاگزانتین (mg/kg ۵۰ یا mg/kg ۷۵) لایه‌های اپیتلیال، غدد معده دست نخورده و سلول‌های مخاطی طبیعی گردن و جداری را نشان دادند که نشان‌دهنده بهبود قابل‌توجه مخاطی است. در مقابل، گروه تحت درمان با امپرازول تغییرات پاتولوژیک خفیفی را نشان دادند که نشان دهنده بهبودی ناقص بود. درمان ترکیبی با آستاگزانتین و امپرازول محافظت از مخاط را بیشتر کرد و تغییرات هیستوپاتولوژیک کمتری را در مقایسه با امپرازول به تنهایی نشان داد. این یافته‌ها نشان می‌دهد که آستاگزانتین به دلیل خواص آنتی‌اکسیدانی و ضدالتهابی قوی آن، به دنبال زخم‌های ناشی از آسپرین، باعث بهبودی بهتر مخاط معده می‌شود.

نتیجه‌گیری: یافته‌های این مطالعه نشان می‌دهد که آستاگزانتین به طور قابل توجهی از بهبود مخاط معده در زخم معده ناشی از آسپرین محافظت می‌کند و آن را تقویت می‌کند. در مقایسه با امپرازول، آستاگزانتین حفظ بافت شناسی برتری را نشان داد و پتانسیل آن را به عنوان یک عامل درمانی موثر برجسته کرد. اثرات ضد زخم مشاهده شده ممکن است به خواص آنتی اکسیدانی، ضد التهابی و ضد آپوپتوز آستاگزانتین نسبت داده شود. این یافته‌ها از آستاگزانتین به عنوان یک کاندید امیدوارکننده برای مدیریت زخم معده، چه به تنهایی یا در ترکیب با درمان‌های مرسوم، پشتیبانی می‌کنند. تحقیقات بیشتر برای روشن شدن مکانیسم‌های دقیق و کاربرد بالینی آن ضروری است.

واژه‌های کلیدی: آسپرین، آستاگزانتین، امپرازول، زخم معده ، موش صحرایی آلبینو

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