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Dr. Mohammed Abdulrazzaq

a) Assistant Professor,
Department of Anesthesia
Techniques Techniques, College
of Health and Medical
Techniques/ Baghdad, Middle
Technical University, Iraq

b) Department of Health
Administration Techniques,
Techniques, Institute of Medical
Technology Al-Mansour, Middle
Technical University, Iraq

Corresponding Author:

Dr. Mohammed Abdulrazzaq

a) Assistant Professor,
Department of Anesthesia
Techniques Techniques, College
of Health and Medical
Techniques/ Baghdad, Middle
Technical University, Iraq

b) Department of Health
Administration Techniques,
Techniques, Institute of Medical
Technology Al-Mansour, Middle
Technical University, Iraq

Gastroprotective effect of *Acacia nilotica* pod extract against ethanol-induced gastritis in Wistar rats

Mohammed Abdulrazzaq

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Abstract

Objectives: This study was designed in order to investigate the gastroprotective effect of ethanolic extract of *Acacia nilotica* pods (ANE) in rats with ethanol induced gastritis.

Materials and Methods: Thirty Wistar rats were grouped into five groups (A-E) comprising of 6 rats each. Group A is the control group; group B is the ethanol group; group C is the omeprazole group (20 mg/kg bw); groups D and E are the ANE groups with 250 mg/kg bw and 500 mg/kg bw being administered. Groups C-E received treatments for 7 days, followed by administration of absolute ethanol (5 mL/Kg) to groups B-E for induction of gastric ulceration. The rats were euthanized and the stomach was examined grossly and microscopically. Evaluation of gastric pH, mucus content, ulcer area and antioxidant parameters such as glutathione, catalase, superoxide dismutase and malondialdehyde was done.

Results: The gastric pH, the level of mucus and the level of antioxidant enzymes of the rats that were exposed to absolute ethanol but not pretreated were significantly reduced. Nonetheless, the pretreated groups revealed that rats pretreated with ANE had an impressive increase in pH, mucus content, less ulcer area (89.6) and a regained antioxidant enzyme level compared to the control group. The histopathological analysis revealed that ANE pretreatment prevents the gastric mucosa when compared to omeprazole.

Conclusion: This study showed that ANE exhibited a gastroprotective effect against ethanol induced gastric injury in rats. This effect can be linked to its antioxidant and mucoprotective properties.

Keywords: *Acacia nilotica*, gastroprotection, ethanol-induced gastritis, antioxidant enzymes, rats

Introduction

Gastric mucosa inflammation also known as gastritis is a universal issue among people. The condition is caused by a myriad of reasons that include uncontrolled alcohol use, non-steroidal anti-inflammatory drugs (NSAIDs) abuse, and infection by the flesh-eating bacteria; *Helicobacter pylori* (Sung *et al.*, 2009) ^[17]. Ethanol use in experimental gastritis induction is not very new. The given model is the product of the direct cytotoxic effect of ethanol leading to the disruption of the protective layer of the gastric mucosa and the subsequent upsurge in the generation of reactive oxygen products (Szabo *et al.*, 1985) ^[18].

The existing treatment alternatives of gastritis comprise non-prescription proton pump inhibitors and H₂-receptor agonists. Nevertheless, the long-term use of this medication changes acid-base balance leading to the deficiency of nutrients, probability of microbial infection and probable drug interactions (Sheen and Triadafilopoulos). It is due to this reason that alternatives to plant-based and safer drugs are sought.

The *Acacia nilotica* (L.) is a medicinal plant belonging to the family Fabaceae and it is extensively spread across the African continent and the Indian subcontinent. Various elements of this plant are traditionally used in the treatment of diverse diseases including cough, diarrhea, dysentery, dental diseases, and skin disorders. These parts include stem bark, leaves, gums, and pods of this plant (Singh *et al.*, 2009) ^[16]. The plant is particularly famous for the high phytochemical content of the pods. These compounds, including phenolic compounds, flavonoids, gallic acid and tannins have been reported to have good antioxidant properties and anti-inflammatory effects (Ali *et al.*, 2012) ^[2].

Antimicrobial, antioxidant, and antiulcerogenic effects are some of the biological effects of *A. nilotica* that have been frequently reported (Saini *et al.*, 2017) ^[14]. The gastroprotective properties of its stem bark, leaves have been reported, however, a study on the action of the ethanol extract of its pods on gastritis induced by ethanol has not been well studied. At this point we made a hypothesis that the ethanolic pod extract of *A. nilotica* will soothe the gastric mucosal ethanol-induced damage in rats through its anti-oxidant and anti-inflammatory effect. This paper consequently examines the gastroprotective behavior of the ethanolic extract of the pods of *A. nilotica* (ANE) on an ethanol-induced gastritis model in Wistar rats.

Materials and Methods

Preparation of the extract: The pods of *A. nilotica* collected were washed using tap water to loosen dust and debris after which they were washed using distilled water. The shade-drying was done at room temperature over a period of two weeks and then a mechanical grinder was used to powder them into a rough one. Approximately 500 g of the powder material was Soxhlet extracted in 1.5 L of 70% ethanol as solvent over 24-36 repetitions (Trease & Evans, 2009) ^[19]. The extract, which was obtained was filtered and concentrated at low pressure at 40 °C using a rotary evaporator (Heidolph, Germany). The product was a semi-solid mass (ANE) that was stored in an airtight container at 4 °C until it was used again. (Mbegu *et al.*, 2015) ^[10]. The percentage yield was calculated by using the following formula:

$$\text{Yield (\%)} = \text{Weight of extract} / \text{Weight of dried powder} \times 100$$

Experimental animal: A total of 30 healthy adult Wistar rats of either sex, weighing 150-200 g, were procured from the Central Animal House of Middle Technical University. The animals were housed in polypropylene cages under standard laboratory conditions (temperature 25 ± 2 °C, 12/12 h light/dark cycle) and provided with a standard pellet diet (Amrut Lab, India) and water *ad libitum*. The study protocol was approved by the Institutional Animal Ethics Committee (IAEC) of Middle Technical University (Reference No: MTU/IACUC/00065/2024) and conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA).

Experimental design for gastritis study

After one week of acclimatization, the rats were divided randomly into five groups ($n = 6$).

- **Group I, normal control:** Received vehicle, 1% w/v Carboxymethyl cellulose, CMC, p.o. for 7 days. On the 7th day, 1 hour after the last dose, they received distilled water, 10 ml/kg, p.o. instead of ethanol.
- **Group II, disease control:** Received vehicle (1% CMC) orally for 7 days. On the 7th day, 1 hour after the last dose, absolute ethanol (5 ml/kg, p.o.) was administered to induce gastric lesions (Mizui & Doteuchi, 1983) ^[11].
- **Group III (Standard drug):** treated with Omeprazole (20 mg/kg, p.o.) suspended in 1% CMC for 7 days. On the 7th day, after 1 hour of the last dose, absolute ethanol (5 ml/kg, p.o.) was administered.
- **Group IV:** Low Dose ANE received ANE at a dose of 250 mg/kg, p.o., suspended in 1% CMC, for 7 days. On the 7th day, absolute ethanol 5 ml/kg, p.o. was given 1 hour after the last dose.

- **Group V:** High Dose ANE - received ANE (500 mg/kg, p.o., suspended in 1% CMC) for 7 days. On the 7th day, 1 hour after the last dose of ANE, absolute ethanol (5 ml/kg, p.o.) was administered.

Induction of gastritis and sacrifice

In all groups, except the Normal Control group, a single oral dose of absolute ethanol at 5 ml/kg was administered for the induction of gastritis as described by Mizui and Doteuchi (1983) ^[11] and Albaayit *et al.* (2016) ^[1]. All animals were killed under deep anesthesia one hour after ethanol administration.

Macroscopic and biochemical analysis

Macroscopic evaluation: Each rat's stomach was excised, opened along the greater curvature, and gently rinsed with ice-cold normal saline in order to study the gastric mucosa. The lesions were scored visually. The ulcer index was calculated based on the severity of the lesions according to Guth *et al.* (1979) ^[6].

Biochemical analysis: Gastric tissue was homogenized in ice-cold phosphate buffer solution. After centrifugation, the supernatant was used for the calculation of some markers of oxidative stress such as Malondialdehyde content as per Ohkawa *et al.* (1979) ^[13], reduced Glutathione as per Ellman (1959) ^[4], and the activity of Superoxide Dismutase enzyme and catalase as per Kakkar *et al.* (1984) ^[8].

Histopathological examination

Samples of the gastric tissue from the glandular portion of the stomach were fixed in 10% neutral buffered formalin, processed, embedded in paraffin wax, sliced into 5 µm thick sections, and stained with Hematoxylin & Eosin (H&E). Light microscopic examinations were conducted for histopathological changes such as edema, leukocyte infiltration, or mucosa injury (Bancroft & Gamble, 2008) ^[3]. Samples were further stained with Periodic Acid-Schiff stain for the estimation of mucus secretion.

Statistical analysis

Results were presented as Mean $\pm$ Standard Error of Mean (SEM). Results were analyzed with one-way analysis of variance (ANOVA), alongside Tukey's test for comparison of multiple groups. A value of < 0.05 was considered statistically significant. Statistical analysis was carried out with GraphPad Software (GraphPad Prism version 8.0, USA).

Results

Macroscopic and biochemical findings

Ethanol administration resulted in extensive hemorrhagic lesions and mucosal necrosis in the gastric lining of untreated rats. The ulcer index and ulcer area were markedly increased (443.8 ± 45.1 mm²) in the ethanol control group. There were also significant decreases in gastric pH (3.3 ± 0.2) and mucus content (0.7 ± 0.2 mg/g tissue) in the same group. However, pretreatment with omeprazole or ANE resulted in drastic reduction in the extent of gastric lesions. The ulcer area was significantly lower in ANE 250 mg/kg (46.2 ± 19.1 mm²) and 500 mg/kg (65.2 ± 21.2 mm²) groups, corresponding to 89.6% and 85.3% inhibition, respectively (Table 1). Both treatments of ANE significantly increased gastric pH (6.0-6.2) and mucus content in comparison with the ethanol control ($p < 0.05$).

Table 1: Effect of *Acacia nilotica* Pod extract treatment on pH, mucus content, ulcer area, and inhibition in rats

Pretreatment	pH of gastric content	Mucus content	Ulcer area (mm ²)	Inhibition (%)
1% CMC v/v (normal control)	7.2 ± 0.2	1.2 ± 0.5	0	NA
Ethanol (5 mL/kg Bw) (untreated control)	3.3 ± 0.2	0.7 ± 0.2	443.8 ± 45.1	NA
Omeprazole in 1% CMC (20 mg/kg Bw)	7.4* ± 0.2	0.9* ± 0.2	69.2* ± 17.3	84.4
ANE in 1% CMC (250 mg/kg Bw)	6.2* ± 0.1	0.7* ± 0.2	46.2* ± 19.1	89.6
ANE in 1% CMC (500 mg/kg Bw)	6.0* ± 0.3	0.7* ± 0.3	65.2* ± 21.2	85.3

*Significant differences at $p > 0.05$ compared with untreated control. Bw: body weight; ANE: *Acacia nilotica* Pod extract; NA: not applicable.

Antioxidant and oxidative stress parameters

Ethanol-treated rats showed significantly increased MDA levels (indicative of lipid peroxidation) and decreased GSH, SOD, and CAT activities compared with normal controls. Pretreatment with ANE significantly restored these

antioxidant markers in a dose-dependent manner. The high-dose ANE group (500 mg/kg bw) showed comparable results with the omeprazole group. This indicates ANEs strong antioxidant defense against ethanol-induced oxidative stress (Figure 1).

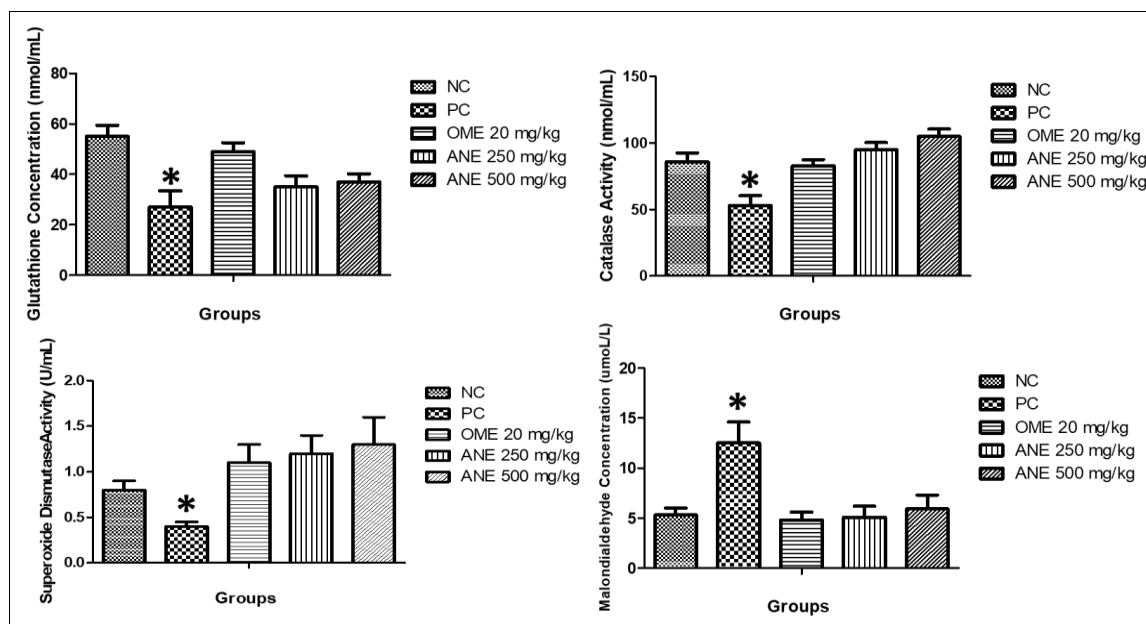


Fig 1: Concentrations of glutathione, catalase, superoxide dismutase and malondialdehyde in gastric tissues of rats in the different treatment groups.

Histopathological observations

Histological sections from the ethanol control group revealed extensive epithelial cell necrosis, submucosal edema, vascular congestion, and inflammatory cell infiltration (Figure 1B). Pretreatment with omeprazole (Figure 2C) and ANE (Figures

2D, E) preserved the integrity of the mucosal epithelium, with minimal edema and no visible ulceration. PAS-stained sections demonstrated intense magenta coloration in ANE-treated groups, confirming enhanced mucin production (Figure 3D-E).

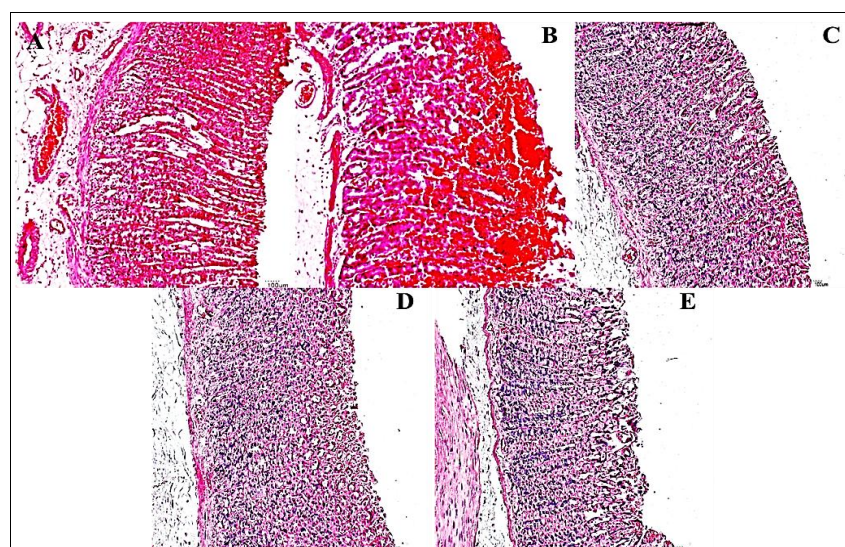


Fig 2: Photomicrograph of gastric mucosa showing (A) normal gastric mucosa (negative control) (B) gastric mucosa with area of ulceration characterized by hemorrhage (positive control) (C) pre-treatment with 20 mg/kg bw omeprazole (D) pre-treatment with 250 mg/kg bw ANE (E) pre-treatment with 500 mg/kg bw ANE showing minimal or absence of ulceration (Hematoxylin and eosin x 200)

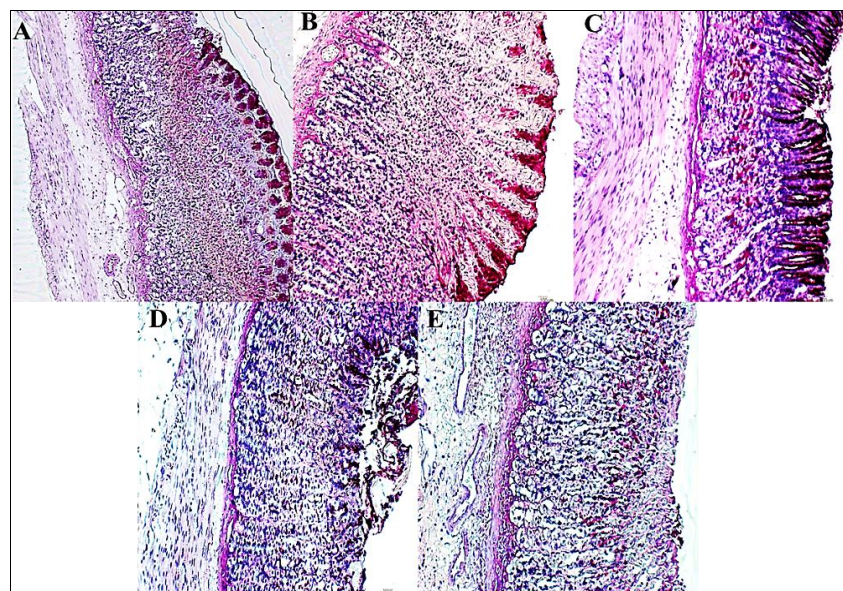


Fig 3: Photomicrograph of gastric mucosa showing (A) normal gastric mucosa (negative control) (B) gastric mucosa with area of ulceration (positive control) (C) pre-treatment with 20 mg/kg bw omeprazole (D) pre-treatment with 250 mg/kg bw (E) pre-treatment with 500 mg/kg bw ANE showing minimal or absence of ulceration. Increased purplish coloration signifies increased mucus production (periodic acid-Schiff stain, $\times 200$).

Discussion

The present study demonstrates that pre-treatment with the ethanolic pod extract of *Acacia nilotica* (ANE) markedly attenuates ethanol-induced gastric mucosal injury in Wistar rats, as evidenced by significantly reduced ulcer area, increased gastric pH and mucus content, preservation of histological architecture, and restoration of antioxidant defenses. These results are in corroboration with earlier preclinical reports indicating anti-ulcer and gastroprotective activity of *A. nilotica* extracts, where polyphenol-rich fractions produced protection in multiple ulcer models, such as pylorus ligation, stress- and NSAID-induced models, and the activity was attributed largely to phenolic constituents and tannins (Bansal & Goel, 2012; Saini *et al.*, 2017) [14, 20].

Mechanistically, ethanol exerts direct cytotoxicity on the gastric epithelium, promotes free-radical formation, depletes cellular glutathione (GSH), and impairs the mucous barrier events that ultimately result in hemorrhagic erosions and inflammation (Szabo *et al.*, 1985) [18]. That ANE restored GSH and up-regulated enzymatic antioxidants (SOD, CAT) and reduced MDA suggests a potential free-radical-scavenging and membrane-stabilizing effect. These antioxidant effects are plausibly mediated by the high content of polyphenols, tannins, and flavonoids reported in *A. nilotica* pods, including gallic acid derivatives and kaempferol glycosides, which have been shown to neutralize reactive oxygen species and inhibit lipid peroxidation in related models (Ali *et al.*, 2012; Singh *et al.*, 2009; Hafez *et al.*, 2024) [2, 16, 7].

Mucosal defense is another key determinant of ulcer susceptibility, and this was clearly enhanced by ANE. Increased PAS staining in the treated animals and the measured increase in mucus content indicated that ANE either stimulated mucin secretion or preserved the mucosal glycoproteins from ethanol-mediated degradation. Plant polyphenols and tannins can form protective complexes with mucosal proteins and stimulate prostaglandin mediated mucous secretion; such actions would provide an explanation for the cytoprotective profile observed here and are consistent with earlier reports indicating mucoprotective mechanisms for *Acacia* species (Bansal & Goel, 2012; Muhammad *et al.*, 2023) [12, 20].

Besides antioxidant and mucoprotective effects, anti-inflammatory activity is very likely to be an important contributor to gastroprotection. In fact, several recent studies report the anti-inflammatory activity of *A. nilotica* extracts in both *in vitro* and *in vivo* ways by reducing pro-inflammatory cytokines (TNF- α , IL-6, and IL-1 β) and suppressing neutrophil infiltration (Hafez *et al.*, 2024; Manzo *et al.*, 2019) [7, 9]. These effects are correlated with accelerated tissue healing in models of dermal and mucosal injuries. Our histological observation of decreased inflammatory infiltrates in rats treated with ANE lends support to this multimodal mechanism.

The therapeutic promise of ANE is also supported by its comparative efficacy versus the standard therapy, omeprazole. Both doses of ANE produced an ulcer inhibition comparable to omeprazole in terms of reduction of ulcer area, preservation of mucosa, and restoration of antioxidants. Though omeprazole acts mainly by suppression of gastric acid secretion (Sheen & Triadafilopoulos, 2011) [15], ANE seems to act mainly by strengthening mucosal defense and enhancement of antioxidant capacity, rather than through acid suppression alone, as reflected by its modest but significant elevation of gastric pH. Therefore, this forms the basis for a complementary or adjunctive role of ANE in the prevention or treatment of ulcers-particularly in conditions where oxidative stress and disruption of protective barriers predominate (Robert *et al.*, 1979; Bansal & Goel, 2012) [20, 21]. Not all studies with *A. nilotica* report uniformly positive outcomes, giving importance to the standardization of extracts, type of plant part used, solvent, and dosing. For example, one ethanolic fruit extract did not heal aspirin-induced ulcers in rats (Gabi Baba *et al.*, 2022) [5], which suggests that certain activities might be model-dependent and may rely on phytochemical composition and duration of treatment. Such variable results direct the need for the phytochemical characterization of extracts, such as HPLC fingerprinting or quantification of marker compounds, along with dose-response validation to ensure reproducibility and translational reliability (Hafez *et al.*, 2024) [7].

Recent toxicology and pharmacology studies generally support a favorable safety profile at doses similar to those used here, but systematic sub-chronic and reproductive-

toxicity data remain limited. Several contemporary studies have expanded its pharmacological scope to include hepatoprotective, antimicrobial, anti-obesity, and neuroprotective properties, while reinforcing its multifunctional bioactivity and highlighting the need for standardized extracts and rigorous toxicological evaluation before human application (Manzo *et al.*, 2019; Hafez *et al.*, 2024) [9, 7].

Conclusion

Ethanol extract of the pods of *Acacia nilotica* was found to demonstrate strong gastroprotective activity in Wistar rats that were exposed to ethanol-induced gastritis. Antioxidant activity, reduction of lipid peroxidation, as well as preservation of the gastric mucosa, played important roles in this activity. These findings buttress the ethnomedicinal use of *A. nilotica* in the treatment of gastric ailments. It is recommended that further mechanistic and clinical studies should be explored.

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