International Journal of Pharmacy and Pharmaceutical Sciences

Print ISSN: 2656-0097 | Online ISSN: 0975-1491 Vol 18, Issue 5, 2026

Original Article

EVALUATION OF GASTROPROTECTIVE PROPERTIES OF *COMBRETUM MOOREANUM* LEAF

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Received: 09 Oct 2025, Revised and Accepted: 20 Feb 2026

ABSTRACT

Objective: This study aimed to evaluate the antiulcer activity of the leaves of *C. mooreanum.*

Methods: Methanol-dichloromethane extract of *C. mooreanum* (MDECM) was prepared and used for the study. The antiulcer activity of the leaf extract was evaluated *in vivo* using ethanol-, acidified ethanol-, aspirin-, and indomethacin-induced ulcers in rats. The MDECM was also subjected to phytochemical analysis.

Results: The MDECM showed a significant reduction in ulcer index in the four models. Compared to the other treatment doses, the 200 mg/kg demonstrated the highest inhibition in acidified ethanol-and aspirin-induced models, and was only marginally more effective than 400 mg/kg in the indomethacin-induced gastric lesion models. The MDECM (400 mg/kg) elicited a higher reduction of ulcer index compared to the standard drug, omeprazole, in the ethanol-induced ulcer model ($p<0.05$). Also, the highest inhibition of gastric ulcer elicited by MDECM in the other three models were comparable to that of the standard drug. Phytochemical analysis showed the presence of alkaloids, flavonoids, tannins, phenolics, saponins, terpenoids, anthocyanins, and glycosides in MDECM.

Conclusion: *C. mooreanum* effectively protected the gastric mucosa against ulcer, supporting its traditional use in the treatment of gastric ulcers. Further studies are ongoing to isolate and characterize the active molecule(s).

Keywords: Acidified ethanol-induced ulcer, Aspirin-induced ulcer, *Combretum mooreanum*, Ethanol-induced ulcer, Indomethacin-induced ulcer

DOI: https://dx.doi.org/10.22159/ijpps.2026v18i5.57131 Journal homepage: https://innovareacademics.in/journals/index.php/ijpps

INTRODUCTION

Peptic ulcer disease (PUD) is a common gastrointestinal disorder involving gastric ulcers, duodenal ulcers, and gastritis [1]. It is a disease of public health concern, with a global prevalence of over eight million, and has remained high, especially in low and middle-income countries [2, 3]. Peptic ulcer disease involves mucosal damage in the gastrointestinal region and is characterized by an imbalance between the aggressive and defensive factors affecting the mucosa [4, 5]. Acid, pepsin, and *Helicobacter pylori* infection are the key aggressive factors, while the defensive factors include mucin, nitric oxide, prostaglandin, bicarbonate, and growth factors [6]. Additionally, ethanol and non-steroidal anti-inflammatory agents (NSAIDs) are known ulcerogenic agents, leading to PUD induction [6].

Conventional therapies, such as proton pump inhibitors and histamine-2 receptor antagonists, have been widely used to manage PUD, demonstrating effectiveness in reducing gastric acid secretion and promoting the healing of ulcers. However, these agents have shown some drawbacks, such as resistance, side effects, and recurrence of ulcers [7]. Medicinal plants have gained interest as an alternative strategy in the treatment of various diseases and ailments. Hence, validating the efficacy of medicinal plants used in folkloric medicine for the treatment of PUD is essential to overcome the drawbacks of the conventional therapies [8].

Combretum mooreanum Exell is a species within the Combretaceae family commonly known as Moore's bushwillow with small, oval-shaped leaves. It is characterized as a straggling shrub reaching up to 2 meters in height, and has flowers known for their red colouration [9]. The plant is indigenous to parts of West and Central Africa, such as Sierra Leone and Congo, and thrives in marshy areas [10]. The young leaves of the plant are useful in food preparation, as they are added to soups. They are also used indigenously in the treatment of fevers, headaches, and stomach aches [11]. Despite the lack of studies evaluating the pharmacological actions of *C. mooreanum* species, other species within the family have demonstrated a range of therapeutic activities. For instance, *C. calobotrys* has been demonstrated to exhibit antimicrobial, anti-inflammatory, and antinociceptive activities [12, 13], while *C. micranthum* is noted for its antibacterial, antifungal, and antidiabetic activities [14–17]. *C. erythrophyllum* has demonstrated antibacterial activities [18], whereas *C. molle* has exhibited anthelmintic, anti-asthmatic, antibacterial, antifungal, and antitussive activities [19–21]. Also, *C. paniculatum, C. racemosum,* and *C. leprosum* [22–24] have all been reported to possess antiulcer activities.

Given the folkloric use of *C. mooreanum* in treatment of gastric ulcer and stomach aches [11], and the activity of related species in other gastrointestinal disorders [25, 26], we performed this study to evaluate the antiulcer activity of the methanol-dichloromethane extract of the leaves of *C. mooreanum.*

MATERIALS AND METHODS

Chemicals and reagents

Absolute ethanol, acidified ethanol (hydrochloric acid in ethanol), aspirin, and indomethacin were purchased from Sigma-Aldrich (St. Louis, MO, USA). Omeprazole (reference standard) was obtained from AstraZeneca Pharmaceuticals (Macclesfield, UK). Methanol and dichloromethane (analytical grade) were supplied by BDH Chemicals Ltd. (Poole, England).

All other chemicals and reagents used, including those for phytochemical tests such as ferric chloride, Dragendorff's reagent, Mayer's reagent, Molisch's reagent, and Liebermann–Burchard reagent, were of analytical grade and obtained from Merck (Darmstadt, Germany).

Distilled water was freshly prepared in the laboratory and used throughout the experiments

Plant material

Fresh leaves of *C. mooreanum* were collected in February 2024 from Agu Orba in Udenu L. G. A in Enugu State, Nigeria. The plant was authenticated by Mr. Felix Nwafor, a taxonomist at the Department of Pharmacognosy and Environmental Medicine, University of Nigeria Nsukka, Enugu State, Nigeria. The voucher specimen was deposited at the herbarium of the Department of Pharmacognosy and Environmental Medicine, University of Nigeria, Nsukka, with a voucher number UNN/11726. Unwanted materials were removed from the plant to prevent adulteration.

Sample preparation and extraction procedure

The plant material was washed in running tap water and chopped into small pieces. The leaves were allowed to dry in a well-ventilated room, at room temperature (25-28 °C). A milling machine was used to grind the dried plant material; subsequently, the dried and ground plant leaves powder (800 g) was extracted using cold maceration comprising a 1:1 (v/v) methanol: dichloromethane mixture (1600 ml methanol and 1600 ml dichloromethane) for 48 h with intermittent agitation and then filtered. The solvent was then evaporated at 40°C under reduced pressure using a rotary evaporator, and the extract (MDECM) obtained was used for the study.

Experimental animals

Male and female Sprague Dawley rats (100-150 g) and conventional grade UN-FERH: NS outbred strain of albino mice (*Mus musculus*) (19 – 29 g) were obtained from the Laboratory Animal Facility of Department of Pharmacology and Toxicology, University of Nigeria, Nsukka. Animals were housed under standard laboratory conditions (12 h light/dark cycle, 25 ± 2 °C) and fed with commercial rat feed and water *ad libitum*. All procedures involving animals were conducted in accordance with institutional ethical guidelines for animal research. Ethical clearance was obtained from the institutional review board of the Faculty of Pharmaceutical Sciences, University of Nigeria, Nsukka, with the number: FPSRA/UNN/24/0103.

Phytochemical analysis

Phytochemical screening of the methanol-dichloromethane leaf extract of *C. mooreanum* (MDECM) was conducted to determine the presence of alkaloids, terpenoids, flavonoids, phenolics, saponins, tannins, carbohydrates, and glycosides according to the methods described by [27]. Additionally, quantitative analysis of the present phytochemicals was conducted according to methods described by Harborne *et al.* [27] and Madhu *et al.* [28].

Acute toxicity test

The acute toxicity of MDECM was determined using Lorke's method (1983) [29] with slight modifications. Three mice each were randomly placed in three groups for the first phase. Each group of animals were administered different doses (10, 100, and 1000 mg/kg) of the test substance orally. The animals were placed under observation for 24 h to monitor their behaviour and check if mortality will occur. The second phase involved the use of six mice, and they were distributed into three groups of two animals each. The animals were administered higher doses (1600, 2900, 5000 mg/kg) of the test substance and then were observed for 24 h to monitor their behaviour and check for mortality.

Evaluation of anti-ulcer activity

Ethanol-induced ulcer

Sprague Dawley rats were fasted for 24 h and randomized into five groups (n = 6). Groups I–III received MDECM at 100, 200, and 400 mg/kg body weight, orally, respectively. Group IV received omeprazole (20 mg/kg) as a standard antiulcer drug, and Group V received distilled water (5 ml/kg), all orally. One hour after treatment with MDECM, gastric ulcers were induced using absolute ethanol (1 ml). Rats were sacrificed after 1 h by overdose of pentobarbital (150 mg/kg) administered intraperitoneally, and the stomachs were excised, opened along the greater curvature, and rinsed with distilled water. Ulcer in each stomach was graded on a 0-7 scale as follows [30]:

0 = no ulcer; 1= one ulcer of length ≤ 0.5 cm; 2 = more than one grade 1 ulcer; 3 = one ulcer of length>0.5 cm but<1 cm; 4 = more than one grade 3 ulcer; 5 = one ulcer of length ≥ 1 cm; 6 = more than one grade 5 ulcer; 7 = complete haemorrhagic lesion of the gastric mucosa

Mean ulcer score for each group was calculated and expressed as the ulcer index (UI). Inhibition of ulcer (%) was calculated using the relation:

$$\text{Inhibition of Ulcer (\%)} = 100\ [(UI_{(}C_{)} - UI_{(}T_{)})/UI_{(}C_{)}]$$

Where $UI_{(}C_{)}$ = ulcer index of control group

$UI_{(}T_{)}$ = ulcer index of the treated group [31]

Acidified ethanol-induced ulcer

The method described by Gong *et al.* [32] was adopted. After 24 h fasting, rats were grouped as noted above. Groups I-III received the MDECM treatment at doses of 100, 200, and 400 mg/kg, respectively. Group IV and V received omeprazole (20 mg/kg) and distilled water (5 ml/kg), respectively. They were all administered orally. Ulceration was induced one hour later using 1 ml of acidified ethanol (0.15 M HCl in 98% ethanol). One hour post ulcer induction, the rats were sacrificed using pentobarbital (150 mg/kg) administered intraperitoneally, and their stomachs removed and opened along the greater curvature. Each stomach was then rinsed to remove the gastric contents and examined for presence of gastric lesions. Similar ulcer grading and calculations as above were done.

Aspirin-induced ulcer

After 12 h of fasting, rats were grouped similarly as stated above. Groups I–III were treated with MDECM at doses of 100, 200, and 400 mg/kg, orally, respectively; Group IV received omeprazole (20 mg/kg), and Group V received distilled water (5 ml/kg) orally. After 45 min, all animals were administered oral aspirin (40 mg/kg) to induce ulcers. Four hours later, the animals were sacrificed, and the stomachs were excised, opened along the greater curvature, rinsed, and examined for ulcers [33]. The severity of the ulcers was noted and scored using a scale of 0-3 based on the length of the ulcer where:

0 = normal; 1 =<1 mm; 2 = 1-2 mm; 3 =>2 mm [30, 34]

The ulcer index and inhibition of ulcer (%) were determined as stated above.

Indomethacin-induced ulcer

The rats were fasted for 24 h and grouped similarly as stated above. Groups I-III received oral MDECM treatment at doses of 100, 200, and 400 mg/kg, respectively. Groups IV and V received omeprazole (20 mg/kg) and distilled water (5 ml/kg), respectively. Ulceration was induced 30 min later using oral 40 mg/kg indomethacin. Eight hours post-ulcer induction, the animals were sacrificed by overdose of pentobarbital (150 mg/kg) administered intraperitoneally, and their stomachs were removed, opened along the greater curvature, rinsed, and examined for ulcers [31]. The severity of the ulcers was noted and scored using a scale of 0-3 based on the length of the ulcer, where:

0 = normal; 1 =<1 mm; 2 = 1-2 mm; 3 =>2 mm [30, 34]

The ulcer index and inhibition of ulcer (%) were determined as stated above.

It is important to note that different ulcer-grading scales were used depending on the ulcerogen applied. Ethanol and acidified ethanol produce extensive, hemorrhagic mucosal damage with variable lesion lengths and depths; hence, a broader 0–7 scoring system was used to capture this range. In contrast, aspirin and indomethacin typically induce smaller, more localized or linear lesions, for which a narrower 0–3 scoring scale is more appropriate.

Statistical analysis

Data was analyzed using one-way ANOVA in GraphPad Prism 10.2.0 and the results expressed as mean±SEM. The results were further subjected to Dunnett's post hoc test for multiple comparisons, and differences between means were accepted as significant at $p<0.05$ or<0.01, as appropriate.

RESULTS

Phytochemical analysis

Phytochemical analysis of MDECM revealed the presence of alkaloids, flavonoids, phenolics, saponins, tannins, terpenoids, carbohydrates, soluble carbohydrates, and glycosides in varying amounts (table 1).

Table 1: Phytochemical constituents of MDECM

Phytochemical		Presence	Quantity (mg/100g±SEM)
1.	Alkaloids	+	3.26±0.02
2.	Flavonoids	+	5.80±0.04
3.	Phenolics	+	10.92±0.01
4.	Tannins	+	4.79±0.02
5.	Saponins	+	6.27±0.04
6.	Soluble Carbohydrates	+	9.80±0.06
7.	Terpenoids	+	2.10±0.02
8.	Anthocyanins	+	1.29±0.02
9.	Glycosides	+	8.88±0.00

+= Present; – = Absent, Quantitative values are expressed as mean±SEM (n = 3) and represent milligrams of each phytochemical per 100 g of the dried methanol–dichloromethane extract (MDECM).

Oral acute toxicity of MDECM

There was no mortality in Phase 1, and the animals showed no symptoms of toxicity. In phase two, there was also no mortality even up to the 5000 mg/kg dose.

Effects of MDECM on ethanol-induced ulcer

Damage to the gastric mucosa caused by ethanol was massive in the control group, with an ulcer index of 29.2±5.2. However, treatment with either omeprazole or MDECM at all doses tested displayed significantly ($p<0.05$ or 0.01) fewer gastric lesions. The ulcer inhibition elicited by MDECM was dose-dependent, with the maximum ulcer inhibition of MDECM (99%) seen at the dose of 400 mg/kg, while omeprazole caused 98% inhibition (fig. 1, table 2).

Table 2: Effects of MDECM on ethanol-induced ulcer

Treatment	Dose (mg/kg)	Ulcer index	Inhibition of ulcer (%)
Water	5 ml/kg	29.2±5.2	_
Omeprazole	20	0.7±0.3*	98.0
MDECM	100	2.4±1.1**	92.0
MDECM	200	1.5±0.5*	95.0
MDECM	400	0.4±0.2*	99.0

Value are expressed as mean±SEM; n=6; * indicates $p<0.05$, ** indicates $p<0.01$ significant difference relative to negative control; MDECM = methanol/dichloromethane extract of *C. mooreanum* leaf

Effects of the MDECM on acidified ethanol-induced ulcer

Acidified ethanol caused large damage to the gastric mucosa in the control group with an ulcer index of 24.0±5.5. Groups administered either omeprazole (20 mg/kg) or MDECM (100, 200, 400 mg/kg) displayed significantly (p<0.05) fewer gastric lesions. The maximum ulcer inhibition of MDECM (98.3%) was seen at the dose of 200 mg/kg, while omeprazole elicited 99% inhibition (fig. 2, table 3).

Table 3: Effects of MDECM on acidified ethanol-induced ulcer

Treatment	Dose (mg/kg)	Ulcer index	Inhibition of ulcer (%)
Water	5 ml/kg	24.0±5.5	-
Omeprazole	20	0.3±0.2*	99.0
MDECM	100	3.5±2.4*	85.4
MDECM	200	0.4±0.3*	98.3
MDECM	400	1.2±0.7*	95.0

Value are expressed as mean±SEM; n=6; * indicates p<0.05 significant difference relative to negative control; MDECM = methanol/dichloromethane extract of *C. mooreanum* leaf

Effects of MDECM on aspirin-induced ulcer

Damage to the gastric glandular mucosa caused by aspirin was immense in the control group, with an ulcer index of 30.2±3.5. On the contrary, treatment with either omeprazole (20 mg/kg) or MDECM (100, 200, 400 mg/kg) caused significantly (p<0.01 or 0.05) fewer gastric lesions. The maximum ulcer inhibition of MDECM (89%) was seen at the dose of 200 mg/kg, while omeprazole produced 96% inhibition (fig. 3, table 4).

Table 4: Effects of MDECM on aspirin-induced ulcer

Treatment	Dose (mg/kg)	Ulcer index	Inhibition of ulcer (%)
Water	5 ml/kg	30.2±3.5	-
Omeprazole	20	1.3±0.5**	96.0
MDECM	100	6.7±2.0**	78.0
MDECM	200	3.4±1.6**	89.0
MDECM	400	7.5±4.5*	75.2

Value are expressed as mean±SEM; n=6; * indicates p<0.05, ** indicates p<0.01 significant difference relative to negative control; MDECM = methanol/dichloromethane extract of *C. mooreanum* leaf

Effects of MDECM on indomethacin-induced ulcer

Indomethacin produced enormous gastric lesions in control rats with an ulcer index of 34.6 ±2.1. The 200 and 400 mg/kg doses of MDECM showed significance (p<0.05) in inhibiting gastric ulcer relative to the negative control. Omeprazole produced an inhibition of 84%, while the highest inhibition of gastric ulcer showed by MDECM was 80.6% which occurred at 200 mg/kg (fig. 4, table 5).

Table 5: Effect of MDECM on indomethacin-induced ulcer

Treatment	Dose (mg/kg)	Ulcer index	Inhibition of ulcer (%)
Water	5 ml/kg	34.6±2.1	_
Omeprazole	20	5.6±3.3*	84.0
MDECM	100	17.3±2.6	50.0
MDECM	200	6.7±2.9*	80.6
MDECM	400	10.0±4.6*	71

Value are expressed as mean±SEM; n=6; * indicates p<0.05 significant difference relative to negative control; MDECM = methanol/dichloromethane extract of *C. mooreanum* leaf

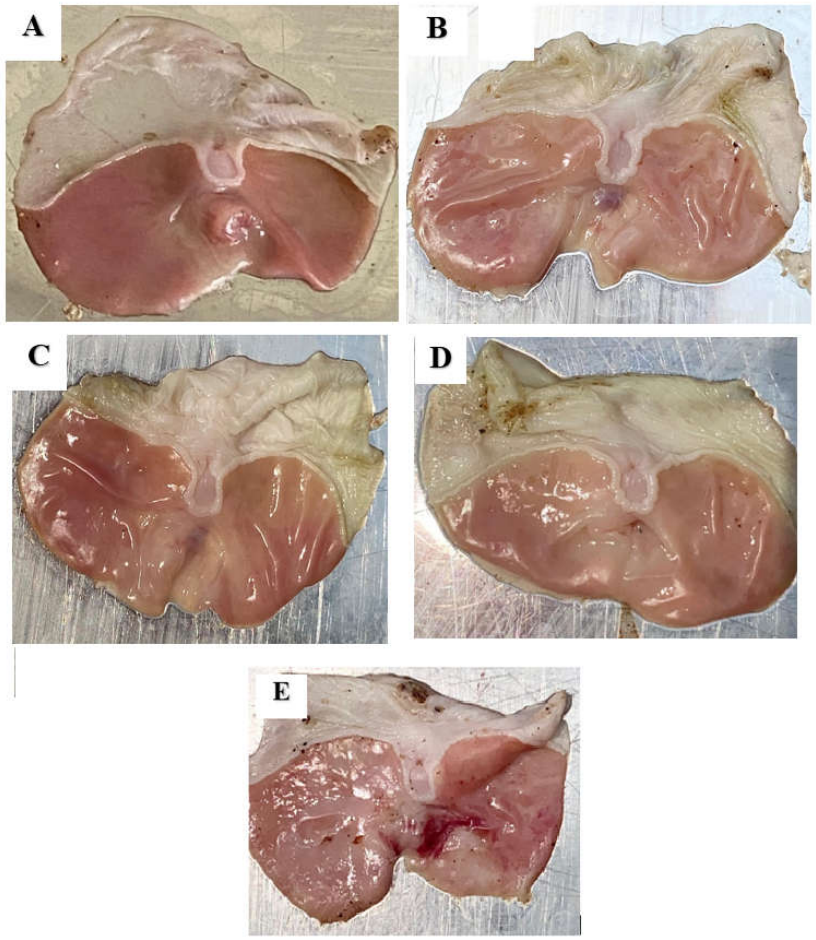

Fig. 1: Effects of MDCME on ethanol-induced gastric ulcer in rats

Representative gastric mucosal photographs showing the effect of MDECM on ethanol-induced gastric lesions. (A) MDECM 100 mg/kg; (B) MDECM 200 mg/kg; (C) MDECM 400 mg/kg; (D) Omeprazole (20 mg/kg); (E) Control (distilled water, 5 ml/kg); Marked reduction in hemorrhagic streaks and mucosal damage is observed in all treated groups relative to control.

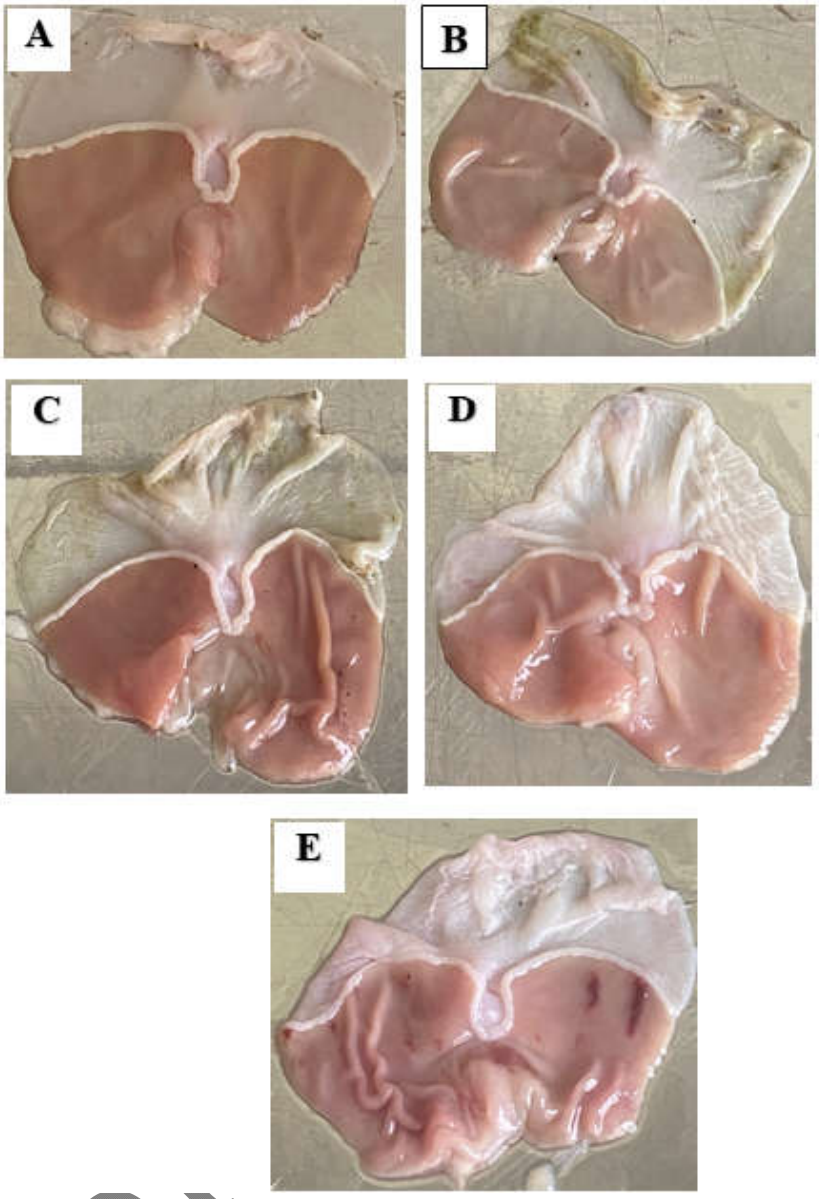

Fig. 2: Effects of MDCME on acidified ethanol-induced gastric ulcer in rats

Representative gastric mucosal photographs showing the effect of MDECM on acidified ethanol-induced gastric lesions. (A) MDECM 100 mg/kg; (B) MDECM 200 mg/kg; (C) MDECM 400 mg/kg; (D) Omeprazole (20 mg/kg); (E) Control (distilled water, 5 ml/kg); Marked reduction in hemorrhagic streaks and mucosal damage is observed in all treated groups relative to control.

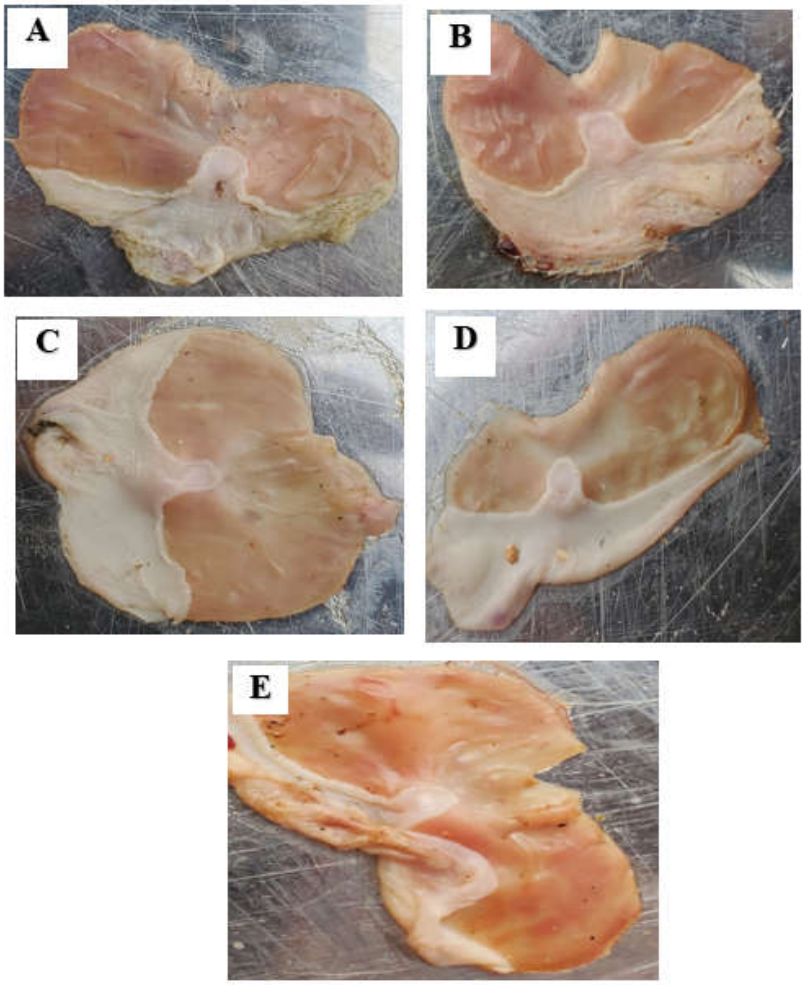

Fig. 3: Effects of MDCME on aspirin-induced gastric ulcer in rats

Representative gastric mucosal photographs showing the effect of MDECM on aspirin-induced gastric lesions. (A) MDECM 100 mg/kg; (B) MDECM 200 mg/kg; (C) MDECM 400 mg/kg; (D) Omeprazole (20 mg/kg); (E) Control (distilled water, 5 ml/kg); Marked reduction in hemorrhagic streaks and mucosal damage is observed in all treated groups relative to control.

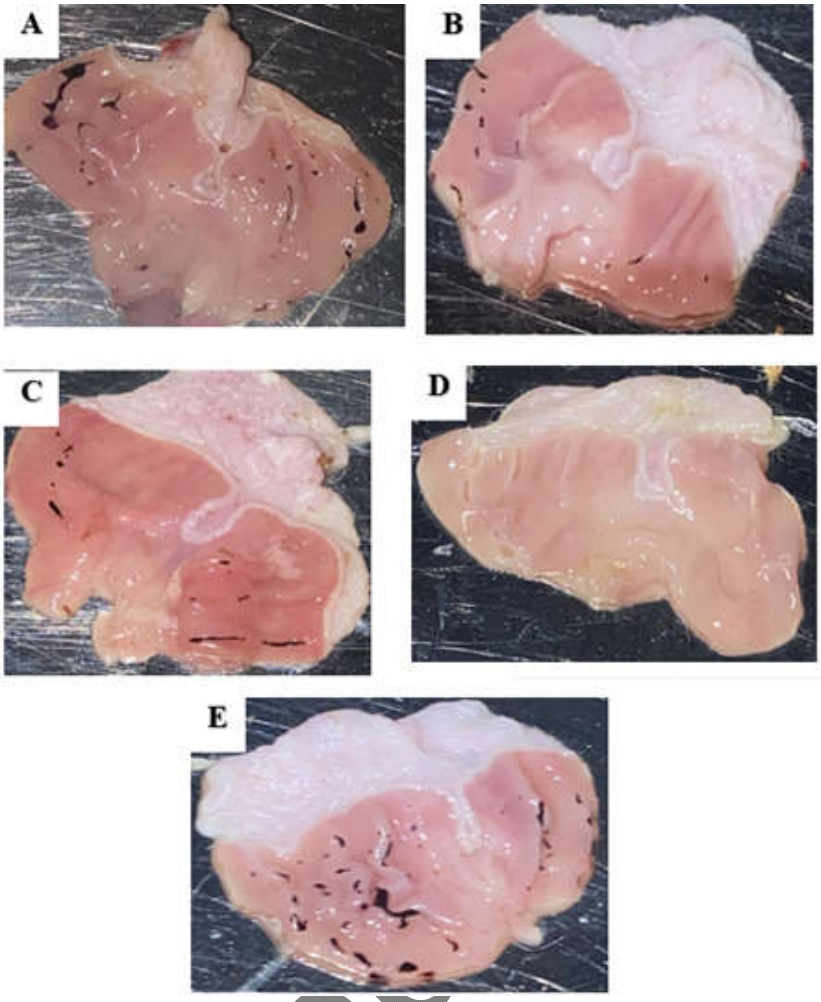

Fig. 4: Effects of MDECM on indomethacin-induced gastric ulcer in rats

Representative gastric mucosal photographs showing the effect of MDECM on indomethacin-induced gastric lesions. (A) MDECM 100 mg/kg; (B) MDECM 200 mg/kg; (C) MDECM 400 mg/kg; (D) Omeprazole (20 mg/kg); (E) Control (distilled water, 5 ml/kg); Marked reduction in hemorrhagic streaks and mucosal damage is observed in all treated groups relative to control.

DISCUSSION

Peptic ulcer disease (PUD) remains a significant clinical challenge, causing epigastric pain, heartburn, dyspepsia, and other gastrointestinal disorders. Conventional treatment options have shown effectiveness in the treatment of PUDs, but are limited by shortcomings such as side effects, drug resistance, and ulcer recurrence [7, 35, 36]. To overcome these shortcomings, researchers have constantly searched for alternative treatment options, including the use of medicinal plants. In this study, we evaluated the anti-ulcer effects of MDECM on rats using four models: ethanol-, acidified ethanol-, aspirin-, and indomethacin-induced ulcer models. Our findings show that MDECM exhibited gastroprotective effects across the models.

Ethanol, acidified ethanol, aspirin, and indomethacin are ulcerogens used to induce gastric lesions in rats. Ethanol has been shown to induce ulcer through multiple mechanisms. It penetrates the mucosal barrier, disrupting the phospholipid bilayer of the cells [30]. This disruption causes the exposure of the affected tissues to gastric acid and pepsin [37]. Additionally, ethanol metabolism through alcohol dehydrogenase leads to the generation of reactive oxygen species (ROS) such as superoxide ion, hydrogen peroxide, and hydroxyl radicals, which are known for their ulcerogenic activities [38]. Postulations have also been made on the ability of ethanol to deplete endogenous glutathione and prostaglandin levels, and increase the release of histamine and generation of free radicals [39]. Our findings demonstrate that MDECM showed a dose-dependent response in the reduction of the ulcer index for the ethanol-induced model. Here, the 400 mg/kg dose of MDECM demonstrated a higher inhibition than omeprazole. Since ethanol induces ulcer through the generation of ROS and depletion of glutathione [39], this emphasizes the gastroprotective activity of MDECM as well as its antioxidant effects.

Acidified ethanol acts through similar mechanisms to ethanol, and also lowers the gastric pH to produce rapid mucosal damage [40]. In the acidified ethanol-induced ulcer, all three doses of MDECM significantly reduced the ulcer index, with 200 mg/kg showing the greatest ulcer index reduction. The ulcer inhibition elicited by the 200 mg/kg dose was comparable with the standard drug, omeprazole, demonstrating the effectiveness of the extract against ulcer.

Aspirin is a non-steroidal anti-inflammatory drug and a non-selective irreversible inhibitor of the cyclooxygenase enzymes-COX-1 and COX-2. These enzymes, especially COX-1, are precursors of prostaglandin (PG), which have gastroprotective properties [41]. The PGE2 and PGI2 play important protective roles in the gastric mucosa by stimulating mucus secretion, bicarbonate secretion, mucosal blood flow, and epithelial cell regeneration [42]. Therefore, the inhibition of PG through COX enzymes leads to a decrease in the mucosal defensive factors, leading to gastric ulcer. In this model, there was a non-linear dose-response relationship as the 200 mg/kg dose showed the greatest inhibition. The reduced inhibition observed at 400 mg/kg

compared to 200 mg/kg may suggest a non-monotonic trend. However, the relatively high standard error at this dose indicates considerable within-group variability, which could have contributed to the apparent reversal in effect. While a biphasic response or counteractive effects from phytochemical constituents at higher doses are plausible explanations [43], this finding should be interpreted cautiously. Further experiments using larger sample sizes are needed to confirm whether the pattern reflects a true pharmacodynamic phenomenon or experimental variability.

Similarly, indomethacin induces gastric ulcer through COX inhibition, but even more severely than aspirin [44]. In addition to the COX inhibition, indomethacin promotes the adherence of neutrophils to the vascular endothelium, leading to the release of ROS, inflammatory mediators, and mucosal injury [45]. Both 200 mg/kg and 400 mg/kg MDECM exhibited significant ulcer index reduction in the indomethacin-induced model, with inhibition comparable to omeprazole. From the considerable inhibition of aspirin-and indomethacin-induced ulcers elicited by MDECM, it is plausible to hypothesize that MDECM modulates prostaglandin synthesis, has antioxidant properties, and preserves the mucosal integrity. However, these interpretations are speculative, as no direct mechanistic assays were performed in this study. Thus, these mechanisms should be viewed as hypotheses that need confirmation through future mechanistic and biochemical studies.

The antiulcer effects of MDECM may involve the reduction of gastric acid secretion, antioxidant effects, scavenging of ROS, and enhancement of mucosal defensive factors. These postulations are in alignment with the findings about some *Combretum* species [24, 46, 47]. With the demonstrated safety of the extract in acute toxicity test, even at the 5000 mg/kg dose, this extract can be potentially applied in therapeutic settings. However, chronic toxicity tests are needed to understand its long-term safety and potential delayed toxicities. Phytochemical screening of MDECM showed the presence of several constituents shown to be beneficial in ulcer. Alkaloids such as berberine, piperine, to mention but a few, have shown protective effects against gastric ulcer [48, 49]. Therefore, the alkaloidal content of the extract may be partly responsible for the anti-ulcer effect. Likewise, flavonoids also have antiulcer properties as they demonstrate cytoprotective, anti-inflammatory, antioxidative, and antibacterial activity [50, 51]. They have also been found to inhibit histamine-stimulated gastric acid secretion and promote blood flow to the mucosa, enhancing its gastroprotective effects [50, 52]. Saponins also show gastroprotective properties, improving the mucosal integrity and protecting the mucus [53–55]. Since MDECM has shown gastroprotective effect, it is also important to fully understand the mechanisms through which it acts. Future studies should evaluate and elucidate the gastroprotective mechanisms of MDECM.

CONCLUSION

C. mooreanum effectively protected the gastric mucosa against ethanol-induced, acidified ethanol-induced, aspirin-induced, and indomethacin-induced gastric ulcers. This study justifies the use of *C. mooreanum* in traditional medicine to manage ulcers. Further studies are needed to isolate and characterize the active molecule.

LIST OF ABBREVIATIONS

MDECM: Methanol-dichloromethane extract of *C. mooreanum*

PUD: Peptic Ulcer Disease

ROS: Reactive Oxygen Species

ACKNOWLEDGMENT

None

FUNDING

No funding was received for this study

AUTHORS CONTRIBUTIONS

Conceptualization: A. C. E; Methodology: ACE, MMN, ECN, CEA; Formal Analysis: ACE, MMN, ECN, CEA and ENO; Investigation: ACE, MMN, ECN, CEA and ENO; Writing – Original Draft Preparation: ACE, MMN, ECN, CEA, ENO, JOM, and IKU; Writing – Review and Editing: ACE, MMN, ECN, CEA, ENO, JOM, and IKU; Supervision: ACE.; Project Administration: ACE, MMN, ECN, CEA, ENO, JOM, and IKU.

CONFLICT OF INTERESTS

The authors declare that they have no competing interests

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