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Original Article

Gastroprotective Effect of Ethanolic Extract of *Vigna Subterranea* in Ethanol-induced Gastric Mucosal Ulceration in Rats

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Abstract

Objective(s): The aim of the study was to investigate the gastroprotective effects of ethanolic seed extract of *V. subterranea* (EEVS) in ethanol-induced gastric mucosal ulceration in rats.

Methods: Gastric ulceration was induced experimentally using ethanol. Group 1 was the vehicle (normal) group and was fed with normal rat chow and water ad libitum; groups 2 was given normal rat chow and water while group 3 was pre-treated with 20 mg/kg omeprazole. Groups 4 and 5 were pre-treated with 200 and 400 mg/kg body weight of EEVS respectively once daily for 21 days. Ulcer index and gastric secretion indices were determined.

Results: The extract significantly (P<0.05) reduced the ulcer index from 9.2±2.14 (ulcer control) to 1.4±1.02 (400 mg/kg). Pre-treatment with the EEVS significantly (P<0.05) increased gastric mucus secretion as compared to the ulcerated control. The EEVS also produced a significant (P<0.05) and dose-dependent reduction in the level of gastric juice volume and acidity in comparison to ulcer control group.

Conclusion: The results suggest that the EEVS possesses significant gastroprotective effects against ethanol-induced gastric mucosal damage in rats which could be attributed to increase in gastric mucus secretion and also decrease in the level of gastric juice volume and acidity.

Introduction

The incidence of gastric ulcer disease globally has been on the increase due to rapid development and civilization constraints. It remains a major health problem in developed and developing countries that poses economic challenges due to number of hospitalization from complications and mortality rates (1). Gastric ulcer is a benign lesion on the stomach mucosal layer. It is one of the most prevalent gastrointestinal disorders. Epidemiological studies have shown that the prevalence is increasing rapidly among the populations (about 3 to 10%) in under developed countries (2, 3). Gastric ulcers in most

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cases occur due to imbalance of activity between the aggressive factors (acid and pepsin) and protective factors (mucus and bicarbonate) (4).

The pathogenesis of gastric ulcer is multifactorial in nature, these include: increased acid-pepsin secretion (5), impaired bicarbonate neutralization and reduced mucus secretion (6). Also poor feeding habits, too much consumption of nonsteroidal anti-inflammatory drugs (NSAIDs) (7), stress (8), smoking (9), and infection by *Helicobacter pylori* (10) have also been implicated in the pathogenesis of gastric ulcer. Numerous antiulcer drugs (such as proton pump inhibitors, histamine receptor antagonists and antibiotics) are available behind the counter for treatment and management of gastric ulcers. But the major challenges remain the high cost, adverse effects and resistance of these drugs with prolonged use (11). These prompted the renewed interest in identifying new antiulcer drugs from natural sources. Numerous medicinal plants used in animal experiments have shown to possess antiulcer effects (12). One of the medicinal plants that may have a great potential as an antiulcer agent is *Vigna subterranea.*

Vigna subterranea (*V. subterranea*) commonly known as Bambara groundnut, is an emerging plant of interest in the treatment, management and prevention of gastric ulcer. It is a legume belonging to the family of *Fabaceae*, and genus of *Vigna*. It has its origin in West Africa (13) and exists in both wild and cultivated forms. The plant is known as jugo beans in South Africa, aboboi in Ghana and nzama in Malawi. In Nigeria, different tribal groups have their indigenous names as “Okpa” among the Igbos in Eastern part of Nigeria; “Gurjiya or Kwaruru” among the Hausas in Northern Nigeria and “Epiroro” by the Yorubas in Western part of Nigeria (14).

The seed extracts of *V. subterranea* have many therapeutic applications in folkloric medicine owing to their effective secondary metabolites such as alkaloids, flavonoids, saponins, resins, and glycosides (15). In Africa, infusions and decoctions of the seeds are used to treat venereal diseases, diarrhoea, cataract, beriberi and polymenorrhea (16, 17). The raw seeds are chewed to alleviate nausea and vomiting in pregnant women (18). According to previous reports, *V. Subterranea* seed extracts have been shown to possess some biological properties such as antioxidant (19) and hepatoprotective (20) activities in animal studies. In spite of multi-functional properties of *V. subterranea*, its gastroprotective effect has not been explored. This study therefore seeks to investigate the gastroprotective effect of ethanolic seed extract of *V. subterranea* (EEVS) in ethanol-induced gastric ulceration in rats.

Materials and Methods

Chemicals and reagents

Analytical grade chemicals and reagents were used in this study. Tween 80, ethanol and diethyl ether were purchased from Sigma-Aldrich Chemical Company (St. Louis Missouri, USA).

Drug preparation

Omeprazole: Omeprazole (Micro Lab India), procured from Godal pharmacy Abakaliki, Nigeria, was used as reference antiulcer drug in this study. The drug was dissolved in 3% Tween-80 (2 ml/kg) and administered to the animals orally in a dose of 20 mg/kg body weight prior to gastric ulcer induction (21). The reference drug was freshly prepared before use.

Experimental animals

Male albino rats weighing 184.7±1.53 g were used for the study. The animals were obtained from the Central Animal House of Faculty of Medicine, Ebonyi State University, Abakaliki. They animals were kept under standard laboratory conditions and fed with standard rat pellets (Vital feed: growers grand cereals, Jos, Plateau state) and water *ad libitum.* The animals were allowed one week acclimatization before the experiment commenced.

Ethical issues

This research protocol was approved by the ethics committee for animal experimentation of the Faculty of Medicine, Ebonyi State University, Abakaliki (Ethic

No. EBSU/REC/MPC/15012/07) and animal handling was according to accepted guidelines for laboratory animal use and care by the National Institute of Health (22).

Collection and authentication of plant materials

Dry seeds of *V. subterranea* were purchased from Abakpa market, Abakaliki, Ebonyi State. They were identified and authenticated by Chijioke Onyeukwu, a botanist in the department of Plant Science and Biotechnology, University of Nigeria, Nsukka, Enugu State. A voucher specimen (UNH 154a) was deposited in the herbarium of the same department for future reference**.**

Preparation of ethanolic extract

The dry seeds of *V. subterranea* were dehulled and ground into flour using a suitable grinder. The seed powder (1000 g) was extracted with absolute ethanol (96%, v/v) in a soxhlet extractor at 70°C. The mixture was vacuum-filtered through Whatman No. 1 filter paper and the filtered extract was concentrated using a vacuum rotary evaporator (Eyla N-1000, Japan) maintained at 45°C. The resulting residue which weighed 93.4 g (recovery 9.3%) was later stored under 4°C before use. Prior to oral administration, the extract was reconstituted in 3% Tween-80 to give required doses of 200 and 400 mg/kg body weight (23).

Qualitative phytochemical analysis

The ethanolic extract of *V. Subterranea* (EEVS) was subjected to qualitative phytochemical screening to identify the secondary constituents (alkaloids, saponins, terpenoids, anthraquinones, flavonoids, tannins, resins, glycosides, steroids and phenols) present in the seed using standard phytochemical methods described by Siddiqui and Ali (24) and Harborne (25).

Experimental design and animal treatment

In this study, ethanol-induced ulcer model was employed to induce ulceration experimentally. The rats were divided randomly into five groups of five rats in each. Group 1 (vehicle) received 3% Tween-80 (2 ml/kg) only. Group 2 (ulcer control) received 3% Tween-80. Group 3 (standard) received 20 mg/kg body weight of omeprazole while groups 4 and 5 (test groups) received 200 and 400 mg/kg body weight of EEVS respectively. All the groups were pre-treated for 21 days between 08.00 am and 09.00 am daily by oral gavage.

Ethanol induced gastric ulceration

This was carried out according to the method described by Mahmood *et al*. (26). Following 21 days of drug and extract pre-treatment, gastric ulcer was induced in 18 h fasted rats by oral administration of 1 ml of absolute ethanol (96%) from groups (2 to 5). After 1 h of ethanol administration, the animals were sacrificed by an overdose of diethyl ether anaesthetization. The gastric content was collected for determination of gastric juice volume, pH, free and total acidity, acid output, and mucus level (27). The stomachs were then dissected and analysed for ulcer index.

Quantification of ulceration and percentage inhibition

The mucosal layer of each stomach was viewed under a magnifying lens (x10) to quantify the gastric ulcerations. The severity of ulcerations was counted and scored using the method of Kulkarni (28) as described below:

00: Normal colouration	0.5: Red colouration
1.0: Spot ulcers	1.5: Haemorrhagic streaks
2.0: Deep ulcers	3.0: perforations

The ulcer index was expressed as the sum of scores given to gastric lesions (29).

Percentage of ulcer inhibition (%I) was calculated according to Hojage *et al*. (30) using the formula:

$$\%I = \frac{(\text{Control mean ulcer index} - \text{Test mean ulcer index}) \times 100\%}{\text{Control mean ulcer index}}$$

Determination of gastric juice volume and pH

The content obtained from each rat stomach was centrifuged at 2000 rpm for 10 mins. The supernatant fluid volume was measured in millilitre (ml) using micro syringe (31). The pH of gastric juice was determined using a digital pH meter.

Determination of free and total acidity and gastric acid output

Free and total acidity were determined by the method of Grossman (32). One ml of gastric juice was titrated with 0.01N NaOH in a conical flask using phenolphthalein (two drops) as indicator until light pink solution indicating pH 7.0 was obtained.

Acid output was expressed as micro equivalents per hour (µEq/hr) and calculated by multiplying the total acidity in mEq/L by the volume of gastric juice in ml (33). The result was divided by 4 to give output per hour.

Acid output (µEq/hr) = Acidity (mEq/L) x Volume of gastric juice (ml) / 4 hr.

Determination of gastric wall mucus secretion

This was determined by the method of Corne *et al* (34). After the rats had been sacrificed, the glandular portion of each rat stomach was weighed and soaked in 1% alcian blue solution in 10% sucrose. The glandular mucus was allowed to bind to alcian blue for 10 mins. The uncomplexed dye which adhered to the stomach tissue was removed by rinsing with sucrose solution. The complexed dye with mucus was extracted for 15 mins in 5 ml of 5% magnesium chloride solution. The resulting blue solutions were shaken with equal volume of diethyl ether. The resulting emulsions were centrifuged at 3000 rpm for 10 mins and the absorbancies of the supernatant were measured at 580 nm using UV-spectrophotometer. The amount of alcian blue concentrated for every gram of wet glandular tissue was afterwards calculated.

Macroscopic examination of the gastric mucosal ulceration

The gastric mucosal layer of the stomach of each rat was rinsed with normal saline to remove blood clot if any. The stomach was then pinned to a flat board to observe any changes in the physical appearance of the mucosa. Photographs of the gastric lesions were taken for proper observation and documentation.

Histological studies of the gastric mucosa

Histopathological studies were conducted using the method described by Bancroft and Stevens (35). After gastric content collection and scoring of gastric lesions, samples from the stomachs representing each group were fixed in 10% formalin for 24 h. The formalin fixed specimens were embedded in paraffin wax and sections of 5 mm thick were cut in a microtome, fixed in 20% alcohol and mounted on glass slides using standard techniques. The slides were viewed under a light microscope (x10, x20 and x40 magnification) after staining the tissues with haematoxylin-eosin stain. Photographs of the gastric lesions were taken with a photo microscope for proper observation and documentation of histopathological lesions.

Statistical analysis

The values were expressed as Mean±Standard error of mean (SEM). For data comparison, one way analysis of variance (ANOVA) was used followed by Tukey's multiple comparison tests. Differences between groups were considered statistically significant at $P<0.05$ using Graph pad Prism Version 6.0 for Windows (GraphPad® Software, San Diego, CA, USA).

Results

Qualitative phytochemical analysis

The result of the preliminary qualitative phytochemical studies of EEVS is presented in Table I.

TABLE I : Qualitative phytochemical analysis of *EEVS*.

Secondary metabolites	*EEVS*
Saponins	+
Tannins	–
Flavonoids	+
Glycosides	+
Alkaloids	+
Steroids	–
Terpenoids	+
Phenols	–
Anthraquinones	–
Resins	+

Keywords: EEVS = Ethanolic extract of *V. Subterranea*; + = present; – = absent.

Effect of EEVS on ulcer index and inhibition

Table II showed the effects of EEVS on ulcer index and % ulcer inhibition in the experimental animals. Oral administration of ethanol caused a significant (P<0.05) increase in ulcer index compared to vehicle control group. The results show that the EEVS significantly (P<0.05) reduced the ulcer index from 9.2±2.14 (ulcer control) to 1.4±1.02 (400 mg/kg) in the ethanol-induced ulcerated rats. Pre-treatment with either omeprazole or the extract significantly (P<0.05) reduced the severity of ethanol-induced gastric lesions. The extract exhibited a dose-dependent inhibition against ulceration in rats. However, maximum inhibition was observed in omeprazole pre-treated group.

TABLE II : Effects of *EEVS* on ulcer index and ulcer inhibition in ethanol-induced ulcerated rats.

Groups	*Treatment & dose*	*Ulcer index*	*Ulcer inhibition (%)*
1 (vehicle)	Tween 80 (2 ml/kg)	0.0±0.00	100
2 (ulcer control)	Ethanol (1 ml)	9.2±2.14#	0.00
3 (standard)	Omeprazole (20 mg/kg)	0.6±0.33*	93.48
4 (test)	Extract (200 mg/kg)	2.0±0.59*	78.26
5 (test)	Extract (400 mg/kg)	1.4±1.02*	84.78

All values are expressed as Mean±SEM (n = 5 in each group), #P<0.05 *vs* vehicle; *P<0.05 *vs* ulcer control.

Effects of EEVS on gastric juice volume and pH

The effects of EEVS on gastric juice volume and pH value are reported in Tables III, the ulcer control group showed a significant (P<0.05) increase in gastric juice volume with corresponding significant (P<0.05) decrease in pH when compared to vehicle control group. Pre-treatment with either the extract or omeprazole caused a significant (P<0.05) decrease in gastric volume coupled with significant (P<0.05) increase in pH value in comparison to ulcer control group. However, the extract exhibited dose-dependent effects on gastric juice volume and pH against ethanol-induced ulcerated rats.

TABLE III : Effects of *EEVS* on gastric juice volume and pH in ethanol-induced ulcerated rats.

Groups	*Treatment & dose*	*Gastric volume (ml)*	*pH*
1 (vehicle)	Tween 80 (2 ml/kg)	1.58±0.75	6.05±0.88
2 (ulcer control)	Ethanol (1 ml)	7.23±0.11#	2.71±0.50#
3 (standard)	Omeprazole (20 mg/kg)	2.31±0.84*	5.48±0.96*
4 (test)	Extract (200 mg/kg)	4.06±0.59*	4.40±0.37*
5 (test)	Extract (400 mg/kg)	3.12±0.63*	5.26±0.13*

All values are expressed as Mean±SEM (n = 5 in each group), #P<0.05 *vs* vehicle; *P<0.05 *vs* ulcer control.

Effects of EEVS on free and total acidity

Table IV showed the effects of EEVS on free and total acidity in ethanol-induced ulcerated rats. Oral administration of ethanol caused a significant (P<0.05) increase in free and total acidity in comparison to vehicle control group. Omeprazole group showed a significant (P<0.05) decrease in free and total acidity as compared to ulcer control group. Pre-treatment with the extract produced a significant (P<0.05) and dose-dependent reduction in free and total acidity in comparison to ulcer control group.

TABLE IV : Effects of *EEVS* on free and total acidity in ethanol-induced ulcerated rats.

Groups	*Treatment & dose*	*Free acidity (mEq/L)*	*Total acidity (mEq/L)*
1 (vehicle)	Tween 80 (2 ml/kg)	12.63±0.70	21.38±0.51
2 (ulcer control)	Ethanol (1 ml)	49.25±1.34#	78.33±0.09#
3 (standard)	Omeprazole (20 mg/kg)	17.01±0.26*	23.64±0.35*
4 (test)	Extract (200 mg/kg)	28.32±0.59*	48.02±0.66*
5 (test)	Extract (400 mg/kg)	20.57±0.82*	39.30±1.58*

All values are expressed as Mean±SEM (n = 5 in each group), #P<0.05 *vs* vehicle; *P<0.05 *vs* ulcer control.

Effect of EEVS on gastric acid output

The effect of EEVS on gastric acid output is shown in Fig. 1, the ulcer control group showed a significant (P<0.05) increase in acid output in comparison to vehicle control group. Omeprazole group produced a significant (P<0.05) decrease in gastric acid output as compared to ulcer control group. The extract produced a significant (P<0.05) and dose-dependent decrease in acid output in comparison to ulcer control group.

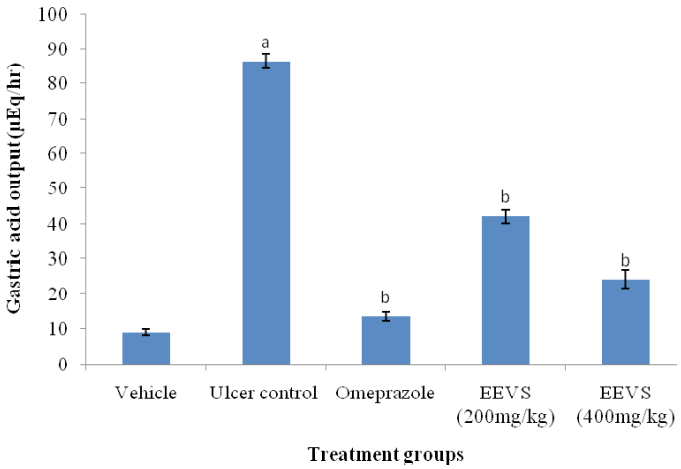

Fig. 1: Effect of EEVS on gastric acid output in ethanol-induced ulcerated rats. Values are expressed as Mean±SEM (n = 5 in each group), [a]P<0.05 *vs.* vehicle group; [b]P<0.05 *vs.* ulcer control group.

Effect of EEVS on gastric mucus secretion

Fig. 2 showed the effect EEVS on gastric mucus secretion in ethanol-induced ulcerated rats. The ulcer control group showed a significant decrease (P<0.05) in gastric mucus secretion compared to vehicle control group. Omeprazole group produced a significant (P<0.05) increase in gastric mucus secretion as compared to ulcer control group. Pre-treatment with the extract also significantly (P<0.05) increase gastric mucus secretion when compared to ulcer control group in a dose-dependent effect.

Macroscopic evaluation of the gastric lesions

In Fig. 3, the vehicle control group showed normal gastric mucosal architecture. Ulcer control group showed severe injuries with extensive haemorrhagic

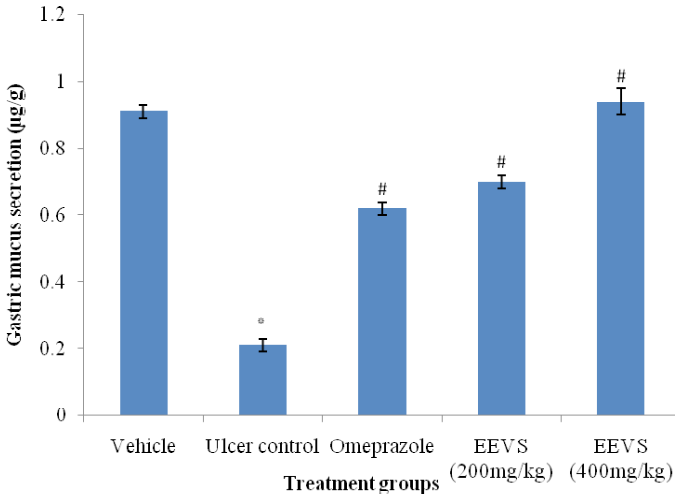

Fig. 2: Effect of EEVS on gastric mucus secretion in ethanol-induced ulcerated rats. Values are expressed as Mean±SEM (n = 5 in each group), [m]P<0.05 *vs.* vehicle group; [#]P<0.05 *vs.* ulcer control group.

necrosis of the gastric mucosa (white arrow) are seen. Groups pre-treated with EEVS (200 and 400 mg/kg) exhibited a fairly protected mucosa in ethanol-induced ulceration model.

Microscopic evaluation of the gastric lesions

In Fig. 4, histological evaluation of ethanol-induced gastric ulceration showed severe disruption to the epithelial surface of the gastric mucosa, oedema of the submucosa with leucocytes infiltration in ulcer control groups. Rats pre-treated with EEVS (200 and 400 mg/kg) and omeprazole had better gastric mucosal protection which is manifested through mild disruption to the epithelial surface with reduced or absence of submucosal oedema and leucocytes infiltration.

Discussion

The available research data from this study clearly demonstrate the protective effect of oral administration of ethanolic seed extract of *V. subterranea* against gastric mucosal ulceration induced experimentally by ethanol in rats. The findings from the study indicated that EEVS offered significant protection against experimentally induced mucosal ulceration. The extract at low and high dose levels offered 78.26% and 84.78% protection

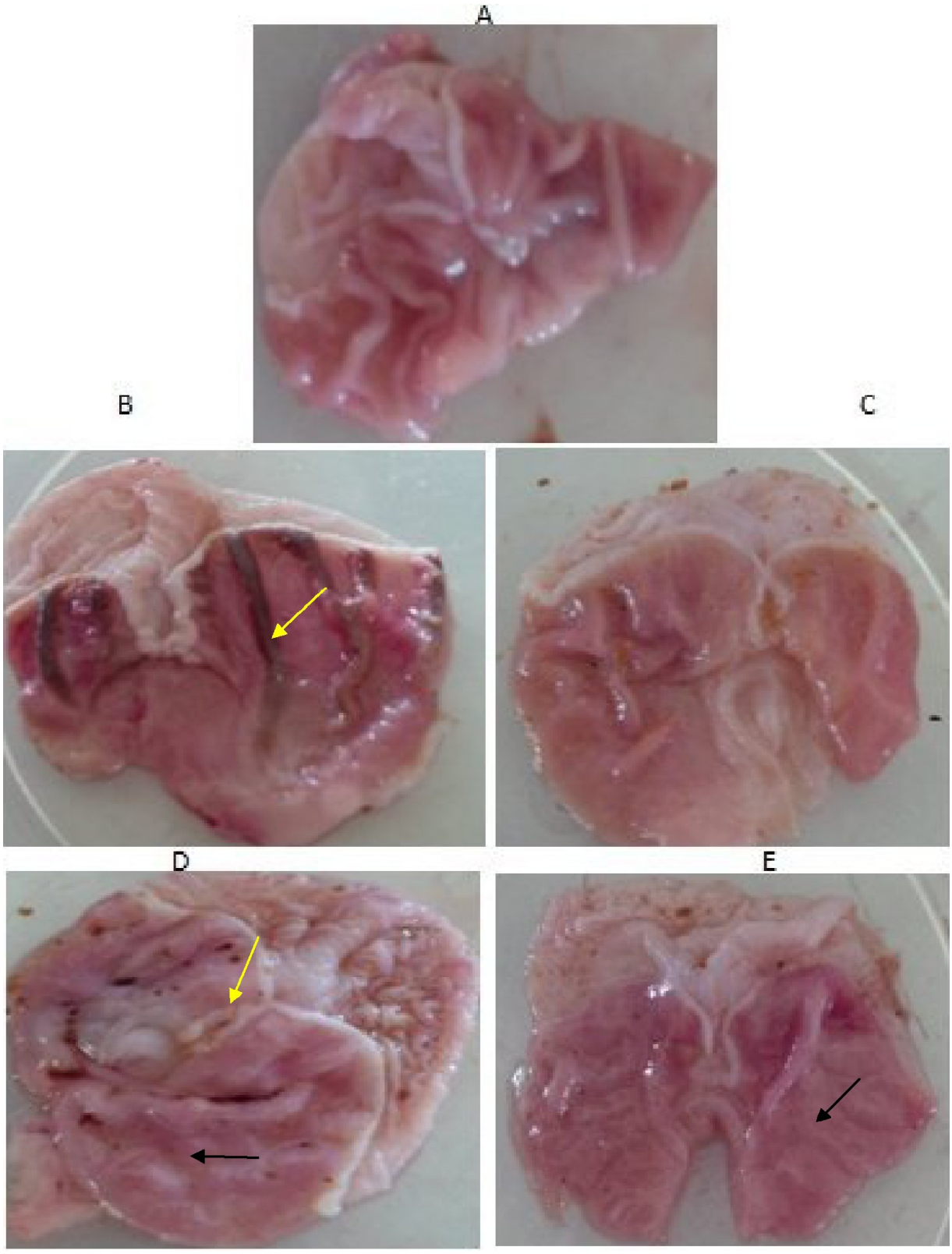

Fig. 3: Macroscopic appearance of the gastric mucosa in ethanol-induced ulcerated rats. A: vehicle control group. Has no injury of gastric mucosa. B: ulcer control group. Severe mucosal injuries with extensive haemorrhagic streaks (yellow arrow) are seen. C: omeprazole group. No mucosal injury is seen. D: EEVS (200 mg/kg). Milder mucosal injury (yellow arrow) compared to that seen in ulcer control rats with flattening of gastric mucosal folds (black arrow). E: EEVS (400 mg/kg). No mucosal injury is seen but there is flattening of the gastric mucosal fold (black arrow).

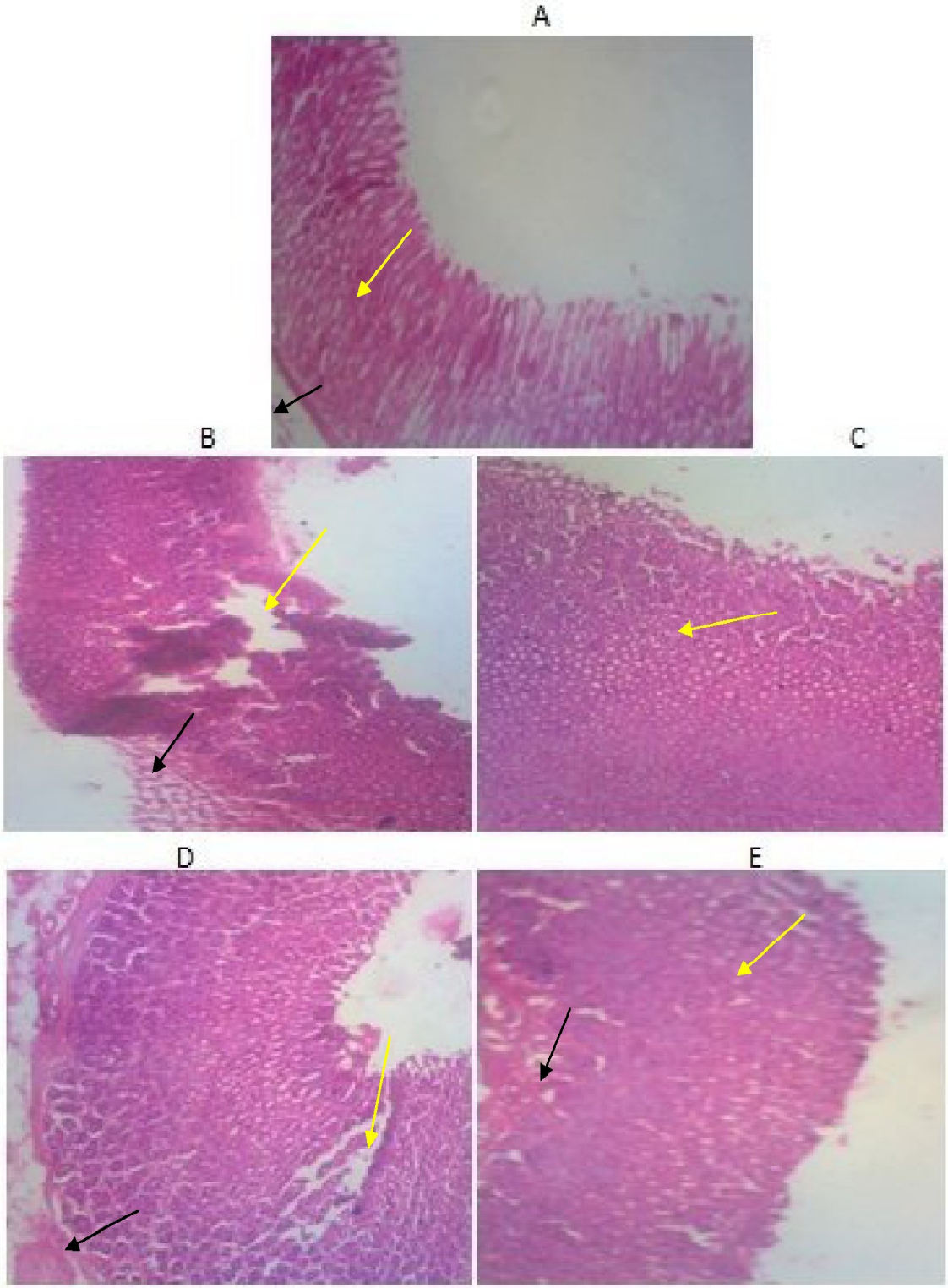

Fig. 4: Histological evaluation of ethanol-induced gastric mucosal damage in rats. A: vehicle group. Normal mucosal epithelium (yellow arrow) and submucosal layer (black arrow) are seen. B: ulcer control group. Severe damage to the epithelial surface with deep mucosal penetration of necrotic lesions (yellow arrow), submucosal oedema and leucocytes infiltration (black arrow) are present. C: omeprazole group. There is absence of epithelial surface disruption, submucosal oedema and leucocytes infiltration. D: EEVS (200 mg/kg). There is moderate disruption of the epithelial surface with no deep mucosal damage (yellow arrow). Submucosal oedema and leucocytes infiltration is reduced (black arrow). E: EEVS (400 mg/kg). Has no mucosal epithelial surface disruption (yellow arrow). Submucosal oedema and leucocytes infiltration is absent (black arrow).

respectively against ethanol-induced gastric mucosal damage. It is interesting to note that the extract at both doses, showed an increase in gastric mucus secretion over omeprazole. This is an indicative of better activity than omeprazole. Nevertheless, the mechanisms behind these antiulcer effects of EEVS in rats have not yet been established.

Ethanol penetrates the stomach mucosa rapidly, damaging the plasma and cell membrane which leads to increase in the permeability of intracellular membrane to water and sodium resulting to damage of the gastric mucosa (36). Ethanol in the stomach disrupts the mucosal barrier by dissolving the gastric mucus thereby causing backflow of acid (37). It stimulates the production of free radicals which causes increase in lipid peroxidation destroying the mucosal membrane. Ethanol also depletes the level of protein in the gastric tissues leading to stasis in mucosal blood flow which is responsible for the haemorrhage and necrosis of the gastric mucosa (38). In the present study, following oral administration of ethanol to the rats, hemorrhagic red streaks of various sizes were observed on the gastric mucosa of the control group. Groups pre-treated with EEVS significantly decrease the rate of damage induced by ethanol to the gastric mucosa in comparison to the control group. The ulcer index was significantly reduced and mucus production significantly increased in EEVS pre-treated groups compared with the control group. This gastroprotective effect of the extract may be mediated partly by gastric mucus restoration. That is, the extract prevents mucus dissolution induced by ethanol.

The pathogenic effects of ethanol-induced gastric mucosal lesions has been established to include increased secretion of gastric acid, which resulted in increased gastric volume, reduced pH, and increased free and total acidity, acid output and ulcer index (39). The auto digestion of the mucosal wall by accumulated gastric juice causes mucosal barrier breakdown leading to gastric ulcer formation (40). Findings from the current study demonstrate that in ethanol-induced gastric ulceration method, the EEVS pre-treated groups produced a significant decrease in free and total acidity, acid output and volume of gastric fluid with a corresponding increase in pH compared to the ulcer control group. This decrease in gastric juice volume in the extract pre-treated groups may be due to a decrease in acid production as evidenced from the gastric juice free and total acidity. This is an indication that EEVS contains some biological active compounds that reduce the acidity of gastric secretions which was increased by ethanol-induced ulceration.

Oxidative stress has been implicated in pathogenesis of many diseases such as gastric ulcer. Antioxidants have been reported to protect the gastric mucosa against different ulcerogens (41). Antioxidants protect cells from oxidative damage while improving the body's immune system against degenerative diseases. The antioxidant level in the stomach is decreased by ethanol leading to release and accumulation of free radicals which is toxic to the structure and function of the membrane. Ethanol metabolism has been found to involve the release of hydroperoxy and superoxide free radicals as oxygen derived free radicals which play a role in acute and chronic gastric ulceration (42). Large amounts of antioxidant compounds have been reported to be present in the seeds of *V. subterranea* (19). The active compounds flavonoids and glycosides which are present in the seeds have been reported by Mota *et al.* (43) and Gill and Bali (44) as antioxidant materials. Flavonoids have been found to display antioxidant properties by scavenging the free radicals and reactive oxygen species produced by ethanol (45). Therefore, it is suggested that the antiulcer property of this extract could be attributed to its antioxidant potential which offers a first line of defence against any ulcerogenic agent by bolstering the mucosal defence system.

The gastric mucosal folds were observed to be flattened in rats pre-treated with low and high doses of EEVS. In rat's stomach, ethanol can cause the circular muscles to contract resulting in compression of the mucosa at the crests of mucosal folds. This mucosal compression can lead to necrosis and ulceration. Flattening of mucosal folds relaxes the circular muscles and increases the area of the mucosa exposed to ulcerogens. This reduces the volume of the gastric irritants at the apex of mucosal

folds and hence protects the mucosa. Takeuchi *et al.* (46) reported that changes in gastric motility may be responsible for the prevention and formation of experimental gastric ulcers. This suggests that by decreasing gastric motility the extract is manifesting its gastroprotective potential.

Histological evaluation revealed that the gastric mucosa is protected and infiltration of leucocytes into the submucosa is inhibited in rats pre-treated with EEVS. The gastric mucosa would extensively be damaged by ethanol leading to increased infiltration of neutrophils into the submucosa. Inflammatory mediators are mainly formed by neutrophils that can release free radicals which are harmful to cells and tissues (47). According to Shimizu *et al.* (48), a decrease in infiltration of neutrophils into ulcerated gastric tissue enhances gastric ulcer healing in rats. This suggests that *V. subterranea* extract possess anti-inflammatory property which could also play a role in gastric ulcer prevention as reported by Swarnakar *et al.* (49).

Ethanolic seed extract of *V. subterranea* has been reported to contain phytochemicals such as flavonoids, glycosides and terpenoids which are among the cytoprotective materials known to possess antiulcer (43, 50), antioxidant (44) and anti-inflammatory (51) activities respectively. It is suggested that these secondary metabolites could enhance bicarbonate, mucus and prostaglandin secretion and attenuate the damaging effects of free radicals in gastrointestinal lumen (52). Therefore, it is pertinent that the decrease in gastric lesions, decrease in volume and acidity of gastric fluid and increase in gastric mucus secretion produced by EEVS in this study could be due to the presence of these active components or some other mechanisms yet to be unravelled. Therefore, further studies are required to establish the exact mechanism of action and isolate the active ingredients in the seeds responsible for the observed gastroprotective effects so as to open the gateway for potential drug development in the future.

Conclusion

Findings from this study suggest that EEVS possesses significant gastroprotective effects against ethanol-induced gastric ulceration in rats. This observed gastroprotective effect could be attributed to decrease in the level of gastric juice volume and acidity with corresponding increase in gastric mucus secretion. Our findings may have beneficial application in the management of gastric mucosal lesions associated with ethanol-induced gastric ulceration.

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Conflict of interest

The authors declare that no conflict of interest exists.

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