

***Preventable Effects of Aloe Vera, Nigella sativa, Moringa oleifera oils and Mixture of them on Aspirin Induced Acute Gastric Ulcer in Experimental Rats.***

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***Abstract***

Peptic ulcer disease (PUD) is one of the most common human diseases which affect approximately 50% of the world's population characterized by mucosal damage due to many factors including infections, alcohol, smoking and certain medications. It appears as a break in the lining of the gastrointestinal mucosa that occurs by abnormal function of acid and pepsin. This study was conducted to evaluate the preventable effects of *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils and mixture of them on aspirin induced acute gastric ulcer in rats. Thirty male albino rats ( $180\pm 10$ g *b.wt.* each) were used in this study and classified into 6 groups, 5 rats each, one of them used as control negative (c-ve) group while the other 5 groups had orally taken aspirin at a dose of 200mg/kg *b.wt.*, for gastric ulcer induction, one of them was left as control positive (c+ve) and the rest four groups had orally taken *Aloe Vera*, *Nigella sativa*, *Moringa Olifera* oils and Mixture of them at a dose of 200mg/kg *b. wt.*, each, respectively. Body weight gain (BWG), stomach weights, the length of gastric ulcer, the volume of gastric juice and stomachs histopathological changes were examined. The results concluded that rat groups which orally ingested with *Aloe Vera*, *Nigella sativa*, *Moringa Olifera* oils and their mixture had increased body weight gain and stomach weights with reduced length of gastric ulcer. The volume of gastric juice increased with improvement in stomachs histological structure. Moreover, the best preventable groups were *Aloe Vera* and *Nigella sativa* oils compared with the control positive compared to other preventable groups concerning the healing of the gastric ulcer length. Accordingly, *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils and mixture of them could be used as preventable agents against gastric ulcer in the experimental rats.

**Keywords:** Peptic ulcer, *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils, rats.

## Introduction

Peptic ulcer (PU) is one of the most common human diseases, affecting approximately 50% of the world's population. Peptic ulcer includes heterogeneous disorders, which appears as a break in the lining of the gastro-intestinal mucosa overlaid with acid and pepsin (**Snowden, 2008**). Peptic ulcers are characteristic mucosal damage due to many factors such as infections, alcohol intake, smoking or certain medications (**Al Batran et al., 2013**). Ethanol-induced gastric ulcer mediated through the free radical generation that leads to depletion of endogenous antioxidants and increases the lipid peroxide levels in mucosa (**Hernández-Muñoz et al., 2000**). Aspirin is acetylsalicylic acid often used to treat pain, fever and inflammation (**Derry et al., 2012**) and (**Thea, et al., 2009**). Despite its therapeutic benefits, it is known to cause stomach ulcers in both humans and animals (**Sørensen et al., 2012**) and (**Bashinskaya, et al., 2011**). The pathogenesis of gastric ulceration caused by aspirin that blocks the activities of cyclooxygenase (COX-1 and COX-2) resulting in decreased secretion of mucus and bicarbonate and decreased mucosal blood flow, impaired platelet aggregation, and alteration of microvascular structures resulting in damage epithelialization, increased leukocyte adhesion and elevating reactive oxygen species (ROS) production, enhancing lipid peroxidation and neutrophil infiltration as well as decreased antioxidant enzymes (**Wallace et al., 2000**) and (**Lamarque, 2004**). Hence, pre-treatment with drugs having anti-inflammatory, antioxidant and restorative properties along with cytochrome 2E1 modulating ability is the first right step in preventing gastric ulcer. Accordingly, histamine-2 receptor antagonists, Proton Pump Inhibitors (PPIs) and antacids are the commonly used antiulcer agents which work with the principle of reducing or neutralizing the stomach acid (**MacAllister, 1999**).

*Aloe Vera* plants have been used medicinally for centuries. Among them, *Aloe barbadensis* (1.8-Dihydroxy-3-hydroxymethyl-10-(6-hydroxymethyl-3,4,5-trihydroxy-2-pyranyl anthrone) known as *A.vera*, is one of the most widely used medicinal plants in human history. Two distinct preparations of *Aloe Vera* plants are used medicinally. The secretions of the leaves (*Aloe Vera*) are used as a laxative, and the gum gel (*A. vera*) extracted from the parenchyma of the leaves is used as a remedy against a variety of skin disorders (**Capasso and Gagarella, 1997**). *Aloe Vera* leaf secretions also possess anti-diabetes (**Ghannam et al., 1986**) and heart stimulating activity (**Yagi et al., 1982**). *A. Vera* also has an anti-ulcer effect that may be due to its antioxidant, anti-inflammatory, mucus secreting, cellular protective or healing activities (**Borra et al., 2011**).

*Nigella sativa* belongs to the family Ranunculaceae and is a miracle herb widely used as a medicinal plant all over the world. *Nigella sativa* seeds and oil have a long history of folklore use in various drug and food systems. Extensive research has been done on the biological and therapeutic potential of *Nigella sativa* such as antimicrobial, antispasmodic, digestive and kidney protecting properties, anti-inflammatory and antioxidant. (**Ahmad et al., 2013**). Black seed, *Nigella Sativa* (NS), a member of the Ranunculaceae family, contains more than 30% fixed oil and 0.4-0.45% w/w volatile oil. The volatile oil contains 18.4-24% thymoquinone (TQ) and 46% several monoterpenes such as p-cymene and  $\alpha$ -piene (**El-Kadi and Kandil 1987**). Experimental studies had shown that *Nigella Sativa* oil possess protective activity against gastric mucosal injury (**Syed et al., 2009**), induced by ethanol (**El-Dakhahny et al., 2000**) and (**Arslan et al., 2005**), ischemia reperfusion (**El-Abhar et al., 2002**) and alcohol toxicity (**Kanter et al., 2005**) in rats.

*Moringa oleifera* Lam., is a rapidly developing evergreen deciduous tree that belongs to the family Moringaceae. It is native in the Indian subcontinent and naturalized in tropical and subtropical regions around the world. It has antioxidant, antimicrobial, anti-inflammatory, antipyretic, antidiabetic, anti-ulcer, anti-tumor, anti-diarrheal and hypocholesterolemic properties. (Anitha et al., 2011). Various parts of the Moringa tree had been used as good sources of unique glucosinolates, flavonoids, and phenolic acids (Amaglo, et al., 2010) and (Coppin et al., 2013), carotenoids (Saini et al., 2014a), tocopherols (Saini et al., 2014b), polyunsaturated fatty acids (PUFAs) (Saini, et al., 2014c), highly bioavailable minerals (Saini, et al., 2014d) and folate (V. B9) (Saini, et al., 2016).

This study was conducted to evaluate the preventable effects of *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils and mixture of them on aspirin induced acute gastric ulcer in rats.

## **Materials and Methods**

### **Materials and rats**

#### **The oils used in the research:**

The oils of *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* were bought from oil and herbal store, Cairo, Egypt.

#### **Aspirin:**

Aspirin (acetylsalicylic acid) as a powdered material was purchased from Al-Gomhoriya Chemical Company, Cairo, Egypt.

#### **Rats:**

Thirty (30) adult male albino rats weighing  $180\pm 10$ g b.wt., were obtained from Research Institute of Ophthalmology, Medical Analysis Department, Giza, Egypt.

### **Methods**

#### **Basal diet:**

The basal diet consisted of casein (12%), corn oil (10%), mineral mixture (4%), vitamin mixture (1%), cellulose (5%), chorine chloride (0.2%), methionine (0.3%) and the remainder is corn starch (67.5%) according to Reeves et al., (1993).

#### **Preparing the rats for the experiment:**

Experimental rats were housed in an individual cage for each group under controlled condition of temperature (25 °C) in the Animal House of Ophthalmology Hospital, Giza, Egypt. All rats were fed on basal diet for seven consecutive days for adaptation in special non-scattering feeding cups to avoid loss of feed and decrease contamination to minimum. Tap water was provided to rats *ad libitum* by glass tubes projecting through wire cages from inverted bottles supported to one side of the cage.

#### **Peptic ulcer induction:**

Acute peptic ulcer was induced for rats by oral ingestion of aspirin (acetylsalicylic acid solution) at a dose of 200mg/kg b.wt., as the method described by Agrawal et al., (2000).

#### **Experimental design:**

After adaptation period (7days), rats were divided randomly into six groups as follows:  
Group (1): Fed on basal diet only for the whole study period as a control negative (C -ve) group.

## Asmaa H. Ahmed

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Group (2): Fed on basal diet only and had taken aspirin orally after adaptation period as a control positive (C +ve) group.

Group (3): Fed on basal diet with oral ingestion of *Aloe Vera* oil at a dose of 200 mg/kg b.wt., after inducing the injury for 28 days.

Group (4): Fed on basal diet with oral ingestion of *Nigella Sativa* oil at a dose of 200 mg/kg b.wt., after inducing the injury for 28 days.

Group (5): Fed on basal diet with oral ingestion of *Moringa Oleifera* oil at a dose of 200 mg/kg b.wt., after inducing the injury for 28 days.

Group (6): Fed on basal diet with oral ingestion of *mixture* of all oils equally at a dose of 200 mg/kg b.wt., after inducing the injury for 28 days.

### Measuring the stomach weights and the volume of gastric juice:

At the end of the experiment, rats were sacrificed after 12 hours fasting from food only with water allowance. After exposing the stomach, a small incision in the duodenum was made to insert a poly-ethane tube into the stomach and was fixed by pylorus ligation. Clamping of esophagus was done to prevent gastric secretions refluxing upward resulting in the loss of volume. The gastric juice was collected in tubes and centrifuged at 5000 × g for 5 min, the supernatant volume was obtained and expressed as mL/100 g b.w. A graduated cylinder was used to assess the volume of gastric juice which was expressed in ml. According to **Kiernan (2001)**.

### Measuring the length of gastric ulcer:

All rats' stomachs had been opened longitudinally, washed in saline followed by examination under dissecting microscope. The length of gastric ulcer was measured and expressed as mean+SE for each group according to the method described by **Akhtar and Ahmad (1995)**.

### Histopathological study:

Specimens from stomachs had been collected from all rats' groups, then fixed in 10% neutral buffered formalin (pH=7.0), dehydrated in ethyl alcohol, after that, cleared in xylol and embedded in paraffin; 4-6 microns thickness, sections were prepared and stained with hematoxylin and eosin (H&E) for examining both fore and glandular parts of the stomach (**Carleton, 1976**).

### Statistical analysis:

The obtained data were statistically analyzed using computerized SPSS Ver. 22 (Statistic Program Sigma stat, statistical soft-ware, SAS Institute, Cary, NC). The Effects of different treatments were analyzed by one-way ANOVA test by using Duncan's multiple range test and  $p < 0.05$  was used to indicate significance between different groups means (**Snedecor and Cochran, 1967**).

## Results and Discussions

### Effects of the tested oils on body weight gain and stomach weight in rats inflicted with gastric ulcer:

Data listed in table (1) shows the effects of *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils and mixture of them at a dose of 200mg/kg. b. wt., each on body weight gain and stomach weight in rats inflicted with gastric ulcer.

**Table (1)**  
**Effects of the tested oils on body weight gain (BWG) and stomach weight in rats inflicted with gastric ulcer**

Rat groups		Stomach weight (g)	BWG (g/28days)
		Mean ± SE	Mean ± SE
-ve Control	1	1.27 ± 0.01 <sup>d</sup>	36.00 ± 0.35 <sup>e</sup>
+ve Control	2	1.11 ± 0.03 <sup>e</sup>	25.00 ± 1.09 <sup>f</sup>
Aloe Vera oil	3	1.44 ± 0.02 <sup>a</sup>	54.00 ± 0.90 <sup>d</sup>
Nigella sativa oil	4	1.33 ± 0.01 <sup>bc</sup>	69.00 ± 0.37 <sup>a</sup>
Moringa Oleifera oil	5	1.32 ± 0.01 <sup>c</sup>	65.00 ± 0.81 <sup>b</sup>
Mix. of all oils	6	1.34 ± 0.02 <sup>b</sup>	61.00 ± 0.29 <sup>c</sup>
LSD*		3.01	0.03

Values denote arithmetic means ± standard error of the means

Means with different letters (a, b, c, d) in the same column differ significantly at  $p \leq 0.05$ , while those with similar letters are non-significant.

\*LSD: Low significant difference.

It could be observed for group No. 2 (+ve Control group) that body weight gain (BWG) was  $25.00 \pm 1.09$  g/28days, compared to  $36.00 \pm 0.35$  g/28days for group No. 1 (-ve Control group), ( $p < 0.05$ ). These results denote that there was significant decrease in body weight gain in rats inflicted with gastric ulcer compared to normal rats. All rats which were ingested Aloe Vera, Nigella sativa, Moringa Oleifera oils and Mixture of them, showed significant increase in BWG compared with the control positive rats. The least body weight gain was the rat group which ingested Aloe Vera.

It is clear from the table (1) that ulcerated gastric rats (+ve control) group showed significant decrease ( $p < 0.05$ ) in stomach weight compared with normal rats (-ve control) group. The mean values of stomach weights of group No. 3 (Aloe Vera oil), group No. 4 (Nigella Sativa oil), group No. 5 (Moringa Oleifera oil) and group No. 6 (Mix., of all oils) were  $1.44 \pm 0.02$ ,  $1.33 \pm 0.01$ ,  $1.32 \pm 0.01$ ,  $1.32 \pm 0.01$  and  $1.34 \pm 0.02$  g, respectively, were significantly higher than group No. 2 (+ve Control)  $1.11 \pm 0.03$  gm. These data illustrate that there was significant increase in stomachs weights in all groups compared to the gastric ulcer group. The highest significant increase in stomachs weights were in group No. 3 (Aloe Vera group) significantly higher than all supplemented and both (+ve) and (-ve) groups. Meanwhile, there were no significant differences between Mix. of all oils group and Nigella Sativa oil group, as well as Moringa Oleifera oil groups.

## Asmaa H. Ahmed

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These results agreed with that of *Hassan et al., (2015)* who found significant increase in the gain of body weights for rabbits on moringa oil treated groups. They attributed to the androgenic properties of moringa which possess considerable concentrations of crude protein, vitamins, calcium and iron that promotes body weight gain in rabbits fed on moringa oils compared to other groups. Several studies had shown that free radicals are important factors in causing stress-induced gastric mucosal damage. Stress has been reported to cause severe oxidative stress in gastric tissues manifested in stimulation of lipid peroxidation by increasing malonaldehyde content and decreasing gastric glutathione content. (*Rao et al., 2008*). The protective effect of *Nigella sativa* oil against stress-induced gastric ulcers can be explained by various mechanisms that *Nigella sativa* has been reported to have an antihistamine effect (*Kahraman, et al., 2003*). Inhibition induced by histamine release to reduce the level of C-Amp which may be due to inhibition of adenylate or induction of phosphodiesterase activity (*Chakravarty, 1993*) and improving the antioxidant status of animals as a result of the increased content of mucus in gastric mucosa (*Saleh et al., 2000*).

In this respect, experimental studies have proven that *Nigella Sativa* oil has a protective activity against gastric mucosal injury as reported by *Syed et al., (2009)*, meanwhile, the prostaglandin promotes mucus and bicarbonate secretions which have a significant effect on stomach weight in experimental animals suffering from gastric ulcers and treated with *Aloe Vera* oil (*Magri et al., 2007*). The anti-ulcer activity of *A. vera* is due to its anti-inflammatory (*Robert et al., 1979*), cytoprotective (*Mahattanadul, 1995*), healing (*Teradaira et al., 1993*). The anti-inflammatory effects might be due to the interaction of leukocytes with the endothelium in the gastric microcirculation reported on *H. pylori* infected rats (*Prabjone et al., 2006*).

*Nigella sativa* can protect the gastric mucosa by increasing the bioavailability of arachidonic acid, which leads to the biosynthesis of cell-protective prostaglandins in the stomach (*Tsuji et al., 1990*). Also, it has been stated by *Mansour, (2000)* that marked inhibition on the release of leukotrienes, that cause mucosal tissue injury and hypoxia might affect the stomachs in experimental animals.

Phytochemical analyzes of *Moringa Oleifera* leaves showed that it is rich in flavonoids contents (*Amaglo et al., 2010*) and (*Coppin et al., 2013*), This may be the active ingredient that exerts an anti-ulcerative effect. The antioxidant and other protective effects of plant flavonoids could occur prior to absorption, within the gastrointestinal tract and could explain the ability of flavonoid-rich foods to protect against gastric and colon ulcers affecting the stomachs weights in experimental animals after gastric ulcer healing (*Halliwell et al., (2000)*).

*Borra et al., (2011)* mentioned that *Aloe Vera* has antiulcer effect of the stomach and this activity is due to its anti-oxidant activity with anti-inflammatory effect, as soon as, mucus secreting with cytoprotective and healing activities for gastric ulcers.

### **Effects of tested oils on the length of gastric ulcer and the volume of gastric juice in rats inflicted with gastric ulcer:**

Data listed in table (2) show the effects of *Aloe Vera*, *Nigella Sativa*, *Moringa Oleifera* oils and mixture of them at a dose of 200mg/kg. b. wt., each, on the length of gastric ulcer and the volume of gastric juice in rats inflicted with gastric ulcer.

**Table (2)**  
**Effects of tested oils on the length of gastric ulcer and Volume of gastric juice in rats inflicted with gastric ulcer**

Rat groups		Gastric ulcer length (mm.)	Volume of gastric juice (mL.)
		Mean ± SE	Mean ± SE
-ve Control	1	0.00	0.40 ± 0.01 <sup>f</sup>
+ve Control	2	1.53 ± 0.03 <sup>a</sup>	0.77 ± 0.04 <sup>a</sup>
Aloe Vera oil	3	0.33 ± 0.01 <sup>d</sup>	0.43 ± 0.02 <sup>de</sup>
Nigella sativa oil	4	0.31 ± 0.02 <sup>de</sup>	0.45 ± 0.03 <sup>d</sup>
Moringa Oleifera oil	5	0.82 ± 0.01 <sup>c</sup>	0.55 ± 0.01 <sup>c</sup>
Mix. of all oils	6	0.99 ± 0.03 <sup>b</sup>	0.60 ± 0.03 <sup>b</sup>
LSD*		0.09	0.02

Values denote arithmetic means ± standard error of the means

Means with different letters (a, b, c, d) in the same column differ significantly at  $p \leq 0.05$  using one way ANOVA test, while those with similar letters are non-significant.

\* LSD: Low significant difference.

It could be observed that the length of gastric ulcer in control +ve group was  $1.53 \pm 0.03$  mm., compared to zero in control -ve group (normal rats) ( $p < 0.05$ ). All rats orally supplemented with all tested oils and mixture showed significant decrease in gastric ulcer length compared to the control positive group which were  $0.33 \pm 0.01$  mm., for group No. 3 (*Aloe Vera* oil),  $0.31 \pm 0.02$  mm., for group No. 4 (*Nigella Sativa* oil),  $0.82 \pm 0.01$  mm., for group No. 5 (*Moringa Oleifera* oil),  $0.99 \pm 0.03$  mm., for group No. 6 (Mixture of all oils) and  $1.53 \pm 0.03$  mm, for (Control +ve) group. Moreover, the best response were in the groups ingested with *Aloe Vera* and *Nigella Sativa* oils.

Table 2 shows the volume of gastric juice in ulcerated rats (control positive group) increased significantly ( $p \leq 0.05$ ) when compared to normal rats which were  $0.77 \pm 0.04$  and  $0.40 \pm 0.01$  mL., respectively. All rats which were orally supplemented with all tested oils and their mixture showed significant decrease in the volume of gastric juice compared to the control positive group. It was  $0.43 \pm 0.02$  mL., for group No. 3 (*Aloe Vera* oil),  $0.45 \pm 0.03$  mL., for group No. 4 (*Nigella Sativa* oil),  $0.55 \pm 0.01$  mL., for group No. 5 (*Moringa Oleifera* oil) and  $0.60 \pm 0.03$  mL., for group No. 6 (Mix. of all oils) revealing significant decrease in gastric juice volume for all supplemented groups compared to the gastric ulcer group (+ve Control). Moreover, the best response groups were *Aloe Vera* and *Nigella Sativa* oils when compared to groups.

The pathogenesis of gastric ulceration caused by aspirin include blocks the activities of cyclooxygenase (COX-1 and COX-2) resulting in decreased secretion of mucus and bicarbonate and decreased mucosal blood flow, impaired platelet aggregation, and alteration of microvascular structures resulting in damage epithelialization, increased leukocyte adhesion and elevating reactive oxygen species (ROS) production, enhancing lipid peroxidation and neutrophil infiltration as well as decreased antioxidant enzymes (*Wallace et al., 2000*) and (*Lamarque, 2004*).

Meanwhile, *Aloe Vera* oil is highly effective in preventing stomach ulcers through a lot of mechanisms (*Fallahi et al., 2022*). The mechanism of proton pump inhibitor, omeprazole has a mechanism of action on developing acute ulcers and accelerating the healing of pre-existing ulcers and

## Asmaa H. Ahmed

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this appears to be mainly due to its strong and long lasting anti-secretory activities (**Osamu, 1984**). Moreover, mucus protects the gastric mucosa from irritants, such as ethanol, hydrochloric acid and acetyl acid. The cytoprotective effect of *A. Vera* may be due to its active ingredients such as tannins, saponins, and flavonoids (**Rajasekaran et al., 2005**).

Regarding *Nigella Sativa*, it has anti gastric ulcer preventable according to the presence of free radical scavenging substances such as thymoquinone (**Khazadi et al., 2008**), moreover, it inhibits free radical generation and increases serum levels of antioxidants (**Kanter et al., 2005**) and (**Naciye et al., 2008**).

In respect to *Moringa oleifera*, it is effective in decreasing the ulcer index that significantly increases the mucus content (**Devaraj et al., 2007**), as soon as containing a number of flavonoids, triterpenes, stimulants, alkaloids, and many other chemical components (**Ross, 1999**). The flavonoid quercetin present in *Moringa oleifera* is well-known anti-ulcer agent (**Suzuki et al., 1998**). Furthermore, it contains rutin, a flavonoid that is reported to have a protective effect on cells in the stomach (**Casa et al., 2000**). *Moringa oleifera* contains steroids such as B-Sitosterol and B-Carotene, both of which are known to reduce the development of stomach ulcers. (**Xiao et al., 1992**). *Moringa oleifera* has significant increase in the number of red blood cells since it contains n-carotene and vitamin B12, which stimulate erythropoiesis and increase hemoglobin concentrations, stacked cell size and platelet count providing anti gastric ulcer (**Samuel et al., 2015**). Moringa's anti-acting effect is attributed to its ability to reduce gastric motility and decrease acid secretion, in addition to the gastro-protective effects of the phytochemicals (**Devaraj et al., 2008**). Accordingly, *Moringa oleifera* may be beneficial in the healing of ulcers in patients suffering from peptic ulcer disease (**Bapan et al., 2022**).

It may be noted that *Aloe Vera extract* inhibits acid secretion due to the presence of lectins in the plant (**Blitz et al., 1963**). Lectins are proteins and glycoproteins that are able to recognize and bind to carbohydrate moieties (**Bardocz et al., 1995**). Lectins had been proven to inhibit the uptake of aminopyrine by parietal cells (**Healey et al., 1998**). Thus, the ability of the extract to inhibit gastric acid production is probably the result of a direct effect on the acid-producing cells. The administration of *A. Vera* enhances the resistance of the mucous membranes and leads to a decrease in the ulcer index and the ulcerated surface with increasing stomach mucus production (**Kossi et al., 2011**).

Decreased volume of gastric juice may be due to decreased level of thyroid hormones (**Fatemeh et al., 2003**) reported that decreased level of thyroid hormone has decreased number of parietal cells secreting gastric juice. It is also possible that thyroid hormones exert their effects by affecting the size of parietal cells or affecting the metabolic activity of these cells (**Weberg et al., 1990**). Moreover, cysteamine-induced ulcer in rat is a widely used model of peptic ulcer disease. Cysteamine hydrochloride inhibits the alkaline mucus secretion from the Brunner glands and stimulates gastric acid secretion rate. Gastric emptying is also delayed, and serum gastrin concentration is increased (**Parmar and Desai, 1993**).

The results in this study were in accordance with (**Almuzafar, 2018**) who reported a significant anti-ulcer activity of *Moringa Oleifera* due to its direct effect on mucus secretion and prostaglandins, thus protecting the gastric mucosa, while the volume of gastric juice significantly decreased in *Moringa Oleifera* and linseed oil treated groups mentioning that, *Moringa Oleifera* is very rich in phenolic compounds, which have gastroprotective properties through various mechanisms and anti-secretory properties that result in decrease in gastric acid volume (**Hamedi et al., 2015**).

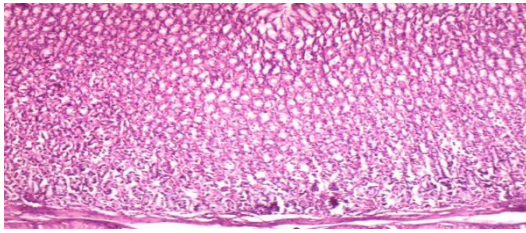


Moreover, stress plays an important role in ulcer formation. The pathophysiology of stress gastric ulcers is complex. Stress ulcers are likely mediated by histamine release while promoting acid secretion and decreasing mucus production, hence, *Moringa Olifera* oil can be used as a preventable agent (**Peters and Richardson, 1983**).

*Nigella Sativa* seeds and oil have a long history of folklore use in various drug and food systems. Extensive researches had been done on the biological and therapeutic potential of *Nigella sativa* such as antimicrobial, antispasmodic, digestive and kidney protecting properties, anti-inflammatory and antioxidant which decrease gastric juice secretion and enhancing gastric ulcer healing (**Ahmad et al., 2013**). Moreover, the methanolic extract of *Nigella Sativa* seeds reduces gastric juice secretion in rats treated with it, and results in a protective effect against ethanol-induced ulcers in rats. (**Geetha and Anitha, 2022**).

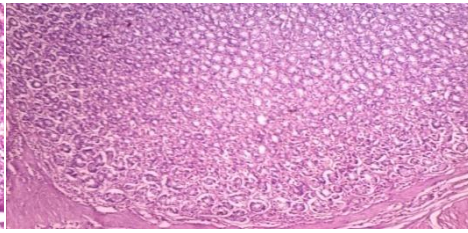
#### **5. Histopathological results:**

Microscopical examination of stomach of rat from group 1 revealed no histopathological changes with the normal histological gastric layers (Photos, 1 & 2). On the other hand, stomach of rats from group 2 revealed necrosis of gastric mucosa, associated with inflammatory cells infiltration (Photos, 3 & 4), submucosal edema (Photo, 5) and inflammatory cells infiltration (Photos, 4 & 5) with congestion of submucosal blood vessel (Photo, 5). However, stomach of rats from group 3 showed submucosal oedema and inflammatory cells infiltration with congestion of submucosal blood vessel (Photos, 6 & 7). Improved picture was observed in stomach of rats from group 4, examined sections revealed no changes except submucosal few inflammatory cells infiltration (Photos, 8 & 9). Moreover, stomach from group 5 revealed no histopathological changes and restored the normal histological structure (Photos, 10 & 11). Also, examined sections from group 6 revealed no histopathological changes (Photo, 12) except submucosal few inflammatory cells infiltration (Photo,13) in some examined sections.



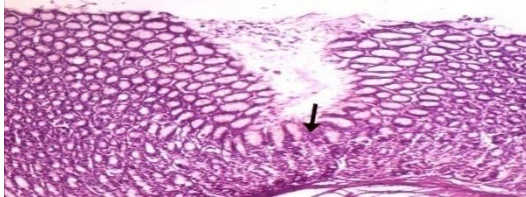
**Photo (1):**

Stomach of rat from group 1 showing no histopathological changes with the normal histological gastric layers (H & E X 100).



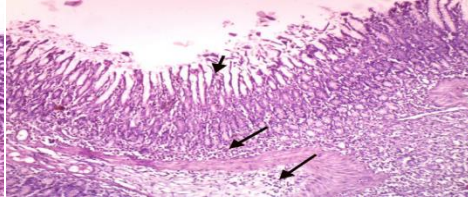
**Photo (2):**

Stomach of rat from group 1 showing no histopathological changes with the normal histological gastric layers (H & E X 100).



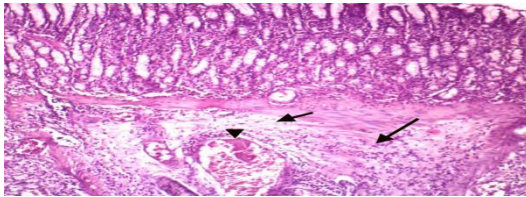
**Photo (3):**

Stomach of rat from group 2 showing necrosis of gastric mucosa, associated with inflammatory cells infiltration (head) (H & E X 100).



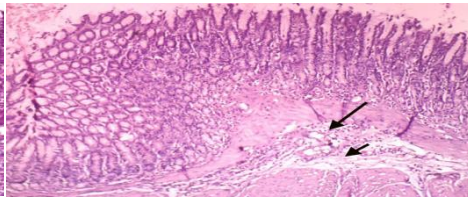
**Photo (4):**

Stomach of rat from group 2 showing necrosis of gastric mucosa, (small arrow) associated with mucosal and submucosal inflammatory cells infiltration (large head) (H & E X 100).



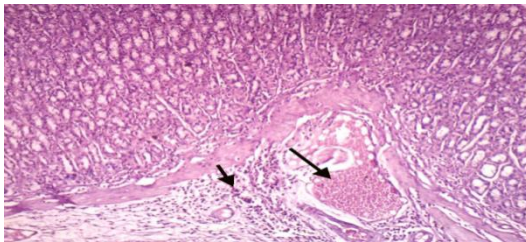
**Photo (5):**

Stomach of rat from group 2 showing submucosal oedema (small arrow) and inflammatory cells infiltration (large head) with congestion of submucosal blood vessel (arrow head) (H & E X 100).



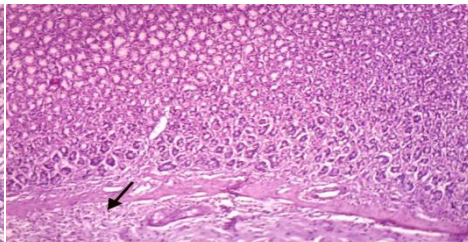
**Photo (6):**

Stomach of rat from group 3 showing submucosal oedema (small arrow) and inflammatory cells infiltration (large arrow) (H & E X 100).



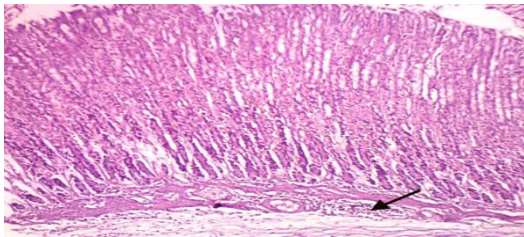
**Photo (7):**

Stomach of rat from group 3 showing submucosal oedema and inflammatory cells infiltration (small arrow) with congestion of submucosal blood vessel (large arrow) (H & E X 100).



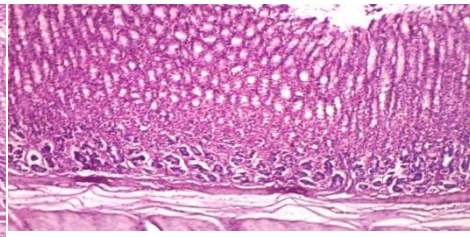
**Photo (8):**

Stomach of rat from group 4 showing submucosal few inflammatory cells infiltration (arrow) (H & E X 100).



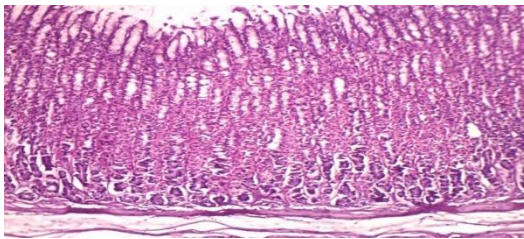
**Photo (9):**

Stomach of rat from group 4 showing submucosal few inflammatory cells infiltration (arrow) (H & E X 100).



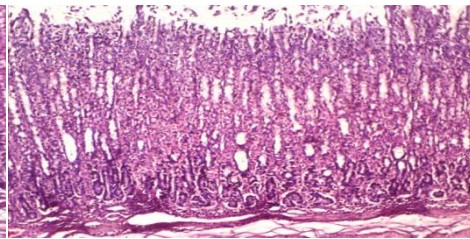
**Photo (10):**

Stomach of rat from group 5 showing no histopathological changes (H & E X 100).



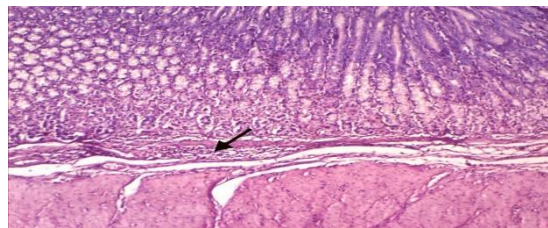
**Photo (11):**

Stomach of rat from group 5 showing no histopathological changes (H & E X 100).



**Photo (12):**

Stomach of rat from group 6 showing no histopathological changes (H & E X 100).



**Photo (13):**

Stomach of rat from group 6 showing submucosal few inflammatory cells infiltration (arrow) (H & E X 100).

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التأثيرات الوقائية لزيت الصبار وحب البركة والمورينجا والمزيج منهم على قرحة المعدة التي يسببها الأسبرين في فئران التجارب.

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### الملخص العربي

يعد مرض القرحة الهضمية من أكثر الأمراض البشرية شيوعاً، حيث يصيب ما يقرب من 50% من سكان العالم ويتميز بتلف الغشاء المخاطي بسبب العديد من العوامل مثل الالتهابات أو تناول الكحول أو التدخين أو بعض الأدوية، وتظهر القرحة على شكل جرح في بطانة الغشاء المخاطي المعدي بسبب الوظيفة الغير طبيعية للحامض وانزيم الببسين المعدي. أجريت هذه الدراسة لتقييم التأثير الوقائي لزيت الصبار وحب البركة والمورينجا ومزيجهم على قرحة المعدة الحادة التي يسببها الأسبرين في الفئران. تم استخدام 30 فأر ذكر بالغ من نوع الألبينو ذات أوزان (180+10جم) في هذه الدراسة وتم تقسيمهم إلى 6 مجموعات (5 فئران للمجموعة)، تركت مجموعة واحدة كمجموعة ضابطة سالبة بينما الخمس مجموعات الأخرى فقد تم حقنها فمويًا بالأسبرين بجرعة 200 مجم/كجم من وزن الجسم لإحداث القرحة المعدية، تركت إحدى هذه المجموعات كمجموعة ضابطة موجبة بينما المجموعات الأربعة المتبقية فقد تم حقنهم وقائياً عن طريق الفم بزيت الصبار وحب البركة والمورينجا ومزيج منهم بجرعة 200 مجم/كجم لكل منهم على التوالي. ثم تم فحص زيادة وزن الجسم وأوزان المعدة، وطول القرحة المعدية، وحجم العصارة المعدية والتغيرات النسيجية لمعدة كل الفئران. وقد خلصت الدراسة إلى أن تناول زيت الصبار وحب البركة والمورينجا والمزيج منهم كوقاية لقرحة المعدة عن طريق الفم أحدثت زيادة في وزن الجسم وأوزان المعدة وقللت من طول القرحة المعدية وحجم العصارة المعدية مع تحسن في التركيب النسيجي للمعدة. علاوة على ذلك كانت أفضل المجموعات التي أحدثت أفضل وقاية هي زيت الصبار وحب البركة عند مقارنتهما بالمجموعات الأخرى وفقاً لدرجة الشفاء من طول القرحة المعدية. ووفقاً لذلك يمكن استخدام زيت الصبار وحب البركة والمورينجا والمزيج منهم كعوامل وقائية ضد القرحة المعدية في فئران التجارب وننصح بتجربة ذلك على الإنسان المصاب بالقرحة.

الكلمات المفتاحية: قرحة المعدة، زيت الصبار وحب البركة والمورينجا، الفئران.