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Effect of Aqueous Licorice (*Glycyrrhiza glabra*) Extract on Gastric Ulcer Induced by Aspirin in Rats

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Abstract:

The main objective of the present study was to investigate the treatment effect of decoction aqueous licorice extract (ALE) on peptic ulcer in rats. Twenty eight adult male Albino rats were used in this study. Rats were divided into two main groups. The first group (7 rats) was fed on basil diet as negative control. The second group (21 rats) was given orally aspirin (400 mg/kg) to induce peptic ulcer and divided into three equal subgroups as follow: subgroup 1, fed on basil diet as positive control; subgroup 2, fed on basil diet plus 10% ALE, and subgroup 3, fed on basil diet plus 15% ALE for 28 days. Ulcer index, Curative (%), gastric pH, reduced glutathione (GSH), malondialdehyde (MDA) content were determined and histopathological parameters were examined. All biochemical investigations referred to treatment efficacy of ALE in treatment groups (3 and 4). Histopathological examination of treatment groups showed that licorice has strong treatment effect on peptic ulcer in both two tested concentrations (10 and 15%). Therfore, the results of this study suggest that licorice can be used for the treatment of peptic ulcer and can protect the stomach against non-steroidal anti-inflammatory drugs induced ulcers.

Kay words: Licorice, peptic ulcer, aspirin, glycyrrhizin, gastric pH, reduced glutathione, malondialdehyde.

Introduction

Peptic ulcer disease (PUD) is an ulceration that develops in the gastrointestinal mucosa when gastric acid and pepsin overwhelm mucosal defenses and destroy mucosal tissue **(Cryer and Spechler, 2006)**. Peptic ulcer is a most popular disease all over the world. It is the most common gastrointestinal tract (GIT) disorder in clinical practice. Recent survey revealed that 1.84% of population in the United States, 2.7% in Australia, and about 1.8% in Canada, also United Kingdom and Egypt suffer from this disease **(Cai *et al.*, 2009)**.

The primary causes of peptic ulcers are *Helicobacter pylori* infection and non-steroidal anti-inflammatory drugs (NSAIDs) (**Goenka *et al.*, 2011**). It leads to the development of chronic gastritis, PUD, mucosal-associated lymphoid tissue (MALT) lymphoma and gastric carcinoma **(Malnick *et al.*, 2014).** In addition, some reports showed that stress, smoking and alcohol consumption are the main causes of peptic ulcer (**Prawit, 2008**). The most common symptom related to PUD is abdominal pain and a burning sensation, which may be precipitated by certain types of foods or accentuated by food intake. For a duodenal ulcer, pain characteristically occurs from 90 minutes to 3 hours after eating, and is usually relieved within minutes either by eating or by use of anti-acids **(Harbison and Dempsey, 2005)**.

Herbal medicine has been opened its way in therapy of gastric ulcer **(Mehrabani *et al.*, 2009)**. In modern medicine, licorice extract has been used for peptic ulcer and as an alternative to bismuth that has a protective role against acid and pepsin secretions by covering the site of lesion and promoting the mucous secretion **(Rahnama *et al.*, 2013 and Memariani *et al.*,2017)**.

Licorice is one of the most commonly used herbs in Western and Eastern herbal medicine and has a very long history of use, both as a medicine and also as a flavoring to disguise the unpleasant flavor of other medications (**Akram *et al.*, 2011**).

Traditionally, licorice used to treat liver diseases, hepatotoxicity (**Lateef *et al.*, 2012**), the compound glycyrrhizic acid found in licorice is routinely used throughout Japan for the treatment and control of chronic viral hepatitis. It can also be used for autoimmune conditions including lupus, scleroderma, rheumatoid arthritis and animal danger allergies **(Rekha and Parvathi, 2012)**. Licorice (*Glycyrrhiza glabra*) used to

treat dyspepsia, peptic ulcers, sore throats, asthma, bronchitis, Addison's disease and rheumatoid arthritis and has been used as a laxative, antitussive and expectorant. Among its most consistent uses are as a demulcent for the digestive system, to treat coughs, to soothe sore throats, and as a flavoring agent (**Xiao and Zhou 2012**).

Licorice has been used since many years ago for reliefing of epigastric pain, and healing of gastric ulcers (**Shibata, 2000).** It stimulates the production of mucus membrane, and may cause symptomatic improvements of peptic ulcer. Also, licorice acts as anti-*H. pylori* (**Mukherjee *et al.*, 2010**), anti-inflammatory (**Adel *et al.*, 2005**) and act to healing peptic ulcer **(Mesut *et al.*, 2009)**. It stimulates the production of mucus membrane, and may cause symptomatic improvements of peptic ulcer. Moreover, licorice might inhibit leukotriene synthesis and prostaglandin; however, it may also stimulate of adrenocortical axis by inhibition of 11-β hydroxyl steroid dehydrogenase *In vitro* antibacterial and antiviral effect of licorice was shown in some studies **(Momeni *et al.*, 2014).** Its derived compounds can raise the concentration of prostaglandins in the digestive system that promote mucus secretion from the stomach; it was also reported that licorice prolongs the life span of surface cells in the stomach and has an antipepsin effect **(Adel *et al.*, 2005).** Therefore, the main objective of the present study is to investigate the treatment effect of aqueous licorice extract (ALE) on gastric ulcer in rats induced by aspirin.

Materials and Methods

Materials

Animals: Twenty eight adult male Albino rats weighing, 130-140 g, were obtained from the animal house colony of Vacsera, Helwan, Egypt. They were housed individually in stainless steel cages under a 12 h light-dark cycle at 20±5°C. Animals were maintained at free access to tap water and were fed a standard pelleted feed for at least 7 days before starting the experiment.

Licorice: Dried roots of licorice were obtained from the local herbal market Cairo, Egypt.

Aspirin: Aspirin (acetylsalicylic acid) as Aspegic® injection was obtained from Ameriyah Pharmaceutical Industries, Alexandria, Egypt.

Diet: Casein was obtained from El-Gomhoriya Company for Trading Drugs, Chemicals and Medical Instruments, Cairo, Egypt. Vitamins and minerals mixture were obtained from the Cairo Company for Chemical Trading, Cairo, Egypt. Starch was obtained from the local market, Cairo, Egypt.

Methods

Preparations of the aqueous licorice extract (ALE)

Licorice water extract was prepared by adding licorice roots 50g or 75g to 500 ml of hot water at 51°C for 3 h then filtered through a cloth (**Lee *et al.,* 2009**). Rats received the ALE ad-libitum.

Preparation of basal diet

Basal diet was prepared according to **Reeves *et al.,* (1993)**.

Induction of peptic Ulcer

Aspirin solution (acetylsalicylic acid) freshly prepared by dissolving one vial (1g) in 5 ml distilled water. Aspirin solution was given orally to the rats on an empty stomach at a single dose of 4 ml (equal to 400 mg Aspirin) for gastric ulcer induction in rats (**Al-Dalain *et al.,* 2008**).

Experimental Design

Twenty eight adult male Albino rats were divided into two main groups. The first group (7 rats) was fed on basil diet as negative control. The second group (21 rats) was given orally aspirin (400 mg/kg) to induce peptic ulcer and divided into three equal subgroups as follow: subgroup 1, fed on basil diet as positive control; subgroup 2, fed on basil diet plus 10% ALE, and subgroup 3, fed on basil diet plus 15% ALE for 28 days. The experimental period was 28 days. After the experiment, the animals fasted overnight, before scarifying. After scarifying, stomach was removed and opened to determine the ulcer index and the pH of the stomach solution as well as the histopathological changes. Body weight (BW) and feed intake (FI) were recorded daily for 4 weeks of the experimental period. Feed efficiency ratio (FER) was calculated according to **Champman *et al.,* (1959).**

Analytical methods

Ulcer index (UI) was measured according to **Garg *et al.,* (1993).** The curative ratio from the ulcer was calculated for the treated groups according to the method described by **Akhtar and Ahamed, (1995)**. Malondialdehyde (MDA) and reduced glutathione (GSH) were

measured according to **Satoh, (1978) and Beutler *et al.*, (1963)**, respectively.

Histopathology

Autopsy samples were taken from the stomach of the scarified rats for histopathological examination (**Banchroft *et al.*, 1996**).

Statistical Analysis

Statistical analysis was carried out using analysis of variance (ANOVA) test with the statistical analysis system, (**SAS, 1996**). Results were expressed as mean ± SD at $P \leq 0.05$ significance.

Results

Table (1) illustrates the effect of ALE on body weight gain (BWG), feed intake (FI) and feed efficiency ratio (FER) of experimental rats. The mean value of BWG shows higher values for groups received ALE. The values of BWG were 40.32±12.32 and 47.60±9.52 for treated groups with ALE in the concentration of 10% and 15%, respectively. With regard to daily FI, the mean values were not significantly different. The mean values of FI were in the range of 15.51± 1.56 to 16.93± 1.36 g/day in all groups. Concerning FER, the treated groups which were received high level of ALE recorded significantly increasing values when comparing with the positive control groups.

Table (2) illustrates the effect of ALE on gastric lesion surface of rats inducted by aspirin. Peptic ulcer lesions were marked in the experiment (+ve control) group, were 3.82±0.49. Administration of ALE was significantly reduced the ulcer index the values were 0.90±0.089 and 0.25±0.098 in treated groups received ALE in the concentrations of 10% and 15% respectively. Also, the effect of treated groups with 10% and 15% concentrations of ALE were measured by curative (%) also indicates that were effective in the gastric ulcer treatment.

Table (3) illustrates the effect of ALE on pH values of gastric juice in rats. The mean values±SD of pH values of control groups received aspirin solution decreased significantly ($p \leq 0.05$), as compared to the negative control group (-ve group). ALE raises the pH of the stomach significantly to the level near that of the negative control group. Peptic ulcer induction of experiment group2 (+ve control) induced significant increase ($p \leq 0.05$) in acidity. The mean values of pH were 1.59±0.19, while treated groups administrated ALE caused significant decrease in stomach acidity. The means values of pH were 2.23±0.12

and 2.43±0.17 in the treated groups received 10% and 15% ALE, respectively.

Table (1): Effect of aqueous licorice extract (ALE) on mean values of body weight gain (BWG), feed intake (FI) and feed efficiency ratio (FER) of rats

Groups	Parameters		
	BWG (g)	**FI (g/day)**	**FER**
G1:– ve control	35.56 ± 13.72^{b}	16.67 ± 1.36^{a}	0.076 ± 0.027^{b}
G2:+ve control	37.52 ± 11.48^{b}	15.83 ± 1.37^{a}	0.084 ± 0.033^{c}
G3: 10% ALE	40.32 ± 12.32^{a}	16.93 ± 1.36^{a}	0.085 ± 0.031^{c}
G4: 15% ALE	47.60 ± 9.52^{a}	15.51 ± 1.56^{a}	0.109 ± 0.024^{a}

All values represented as mean ± SD. Means with different superscript aresignificantly different ($P < 0.05$).

Table (2): Effect of aqueous licorice extract (ALE) on gastric lesion surface of rats inducted by aspirin

Groups	Ulcer index	Curative %
G1:– ve control	0.00	100
G2:+ve control	3.82 ± 0.49^{a}	0
G4: 10% ALE	0.90 ± 0.089^{b}	76.44
G5: 15% ALE	0.25 ± 0.098^{c}	93.46

All values represented as mean ± SD. Means with different superscript are significantly different ($P < 0.05$).

Table (3): Effect of aqueous licorice extract on pH of gastric juice in rats

Groups	pH
G1:– ve control	2.05 ± 0.15^{a}
G2:+ve control	1.59 ± 0.19^{b}
G4: 10% ALE	2.23 ± 0.12^{a}
G5: 15% ALE	2.43 ± 0.17^{a}

All values represented as mean ± SD. Means with different superscript are significantly different ($P < 0.05$).

Table (4) exhibits the effect of ALE on serum reduced glutathione (GSH) and malondialdehyde (MDA) of rats. The antioxidant as measured by GSH was increased significantly ($p \leq 0.05$) in the treatment

groups received ALE in the concentration of 10% and 15%, compared with positive control group. The mean values of treatment groups 3 and 4 were 2.93±0.15 mg/dl and 3.40±0.17 mg/dl, respectively compared with positive control group which show the value of 2.09±0.18 mg/dl. The oxidative stress were measured by malondialdehyde (MDA) which showed increased significantly ($p \leq 0.05$) in the positive control group and recorded 48.6 ± 3.67 nmol/ml as compared with treatment groups (3 and 4) which show the mean values of 39.10±3.00 n mol/ml, 35.80 ±3.07, respectively.

Histopathological examination

The histopathological changes of the stomach as a result of aspirin induced peptic ulcer and the effect of treatment of ALE administration are illustrated in histopathological (Table, 5 and Photos, 1-4). Histopathological examination for stomach of rats from group 1 (-ve control) showed no alteration and normal histopathological structure of the mucosal, submucosal and muscular layer were recorded (photo 1). Meanwhile, stomach of rats from group 2 (+ve control) revealed that diffuse mononuclear leucocytes inflammatory cells infiltration in mucosal layers associated with congestion in the blood vessels on the same layers (photo 2). Stomach of rats from group 3 (treated by 10% ALE) showed inflammatory cells infiltration in mucosal and submucosal layer (photo 3). Stomach from group 4 (treated with 15% ALE) showed oedema with few inflammatory cells infiltration in the submucosal layer (photo 4).

Discussion

Peptic ulcer is a very common disease that affects millions of people in all parts of the world (**Mukherjee *et al.*, 2010**). Medical treatment of peptic ulcer is intended to relieve pain, accelerate healing of the ulcer creator, and prevent complications and recurrences. For this, many of synthetic drugs are used. However, all these synthetic drugs may result in a number of side effects **(Fatima *et al.*, 2008).** Among the antiulcer herbal product is that from an indigenous plant *Glycyrrhiza glabra* belonging to family *Fabaceae* is commonly known as licorice **(Fatima *et al.*, 2008),** and in Egypt licorice known as erq'soos (**Wassef,**

Table (4): Effect of aqueous licorice extract on serum reduced glutathione (GSH) and malondialdehyde (MDA) of rats

Groups	Parameters	
	GSH (mg/dl)	**MDA (n mol/ml)**
G1:– ve control	3.79 ± 0.12^{a}	33.1 ± 3.08^{b}
G2:+ve control	2.09 ± 0.18^{c}	48.6 ± 3.67^{a}
G4: 10% ALE	2.93 ± 0.15^{b}	39.1 ± 3.00^{b}
G5: 15% ALE	3.40 ± 0.17^{b}	35.8 ± 3.07^{b}

All values represented as mean ± SD. Means with different superscript are significantly different ($P < 0.05$).

Table (5): Effect of aqueous licorice extract (ALE) on the histopathological changes in the stomach of rats

Histopathological Alterations	**G1 (-ve)**	**G2 (+ve)**	**G3 (10% ALE)**	**G4 (15% ALE)**
Inflammatory cells infiltration in mucosal layer	–	+++	++	–
Inflammatory cells infiltration in sub mucosal layer	–	–	++	+
Inflammatory cells infiltration in mucosalar layer	–	++	–	–
Congestion in blood vessels of sub mucosal layer	–	+	–	–
Congestion in mucosal blood vessels	–	+	–	–
Odema in sub mucosal	–	–	–	+

– = nil + = mild ++ = moderate +++ = severe

2004). Licorice is useful in the treatment of peptic ulcer because it act as anti-*H. Pylori* (**Mukherjee *et al.,* 2010**), anti-inflammatory (**Akram *et al.,* 2011**) and healing peptic ulcer **(Mesut *et al.,* 2009)**. The fresh root contains about 20% of water-soluble extractives, and much of this is composed of glycyrrhizin, which is considered the primary active ingredient. Several flavonoids are present also in licorice (**Isbrucker and Burdock, 2006**).

Glycyrrhizin has anti-inflammatory activity and increase the rate of mucus secretion by gastric mucosa. Deglycyrrhizinated licorice

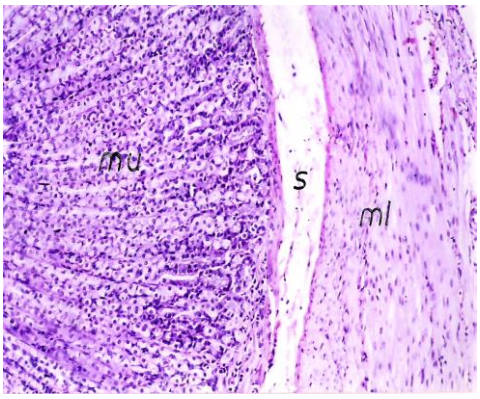

Photo 1. Stomach of rat from group 1 (–ve control) showing no histopathological alteration and normal histopathological structure of the mucosal, submucosal and muscular layer (H and Ex 40)

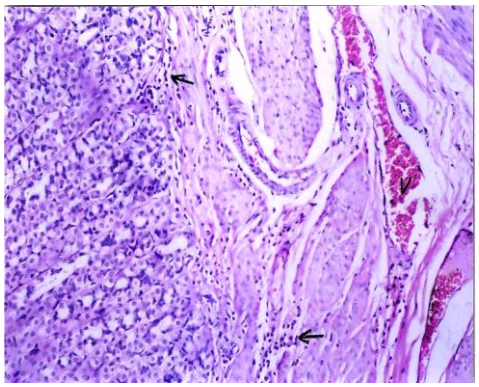

Photo 2. Stomach of rat from group 2 (+ve control) showing diffuse mononuclear leucocytes inflammatory cells infiltration in mucosal layers associated with congestion in the blood vessels on the same layers (H and Ex 40)

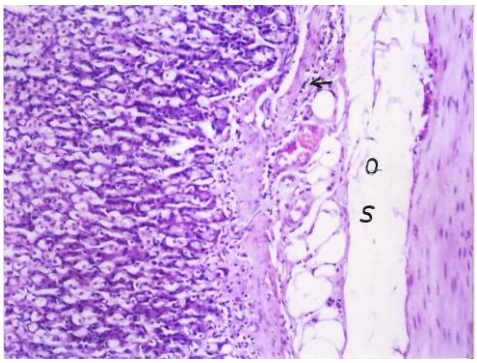

Photo 3. Stomach of rat from group 3 (10% ALE) showing inflammatory cells infiltration in mucosal and submucosal layer (H and Ex 40)

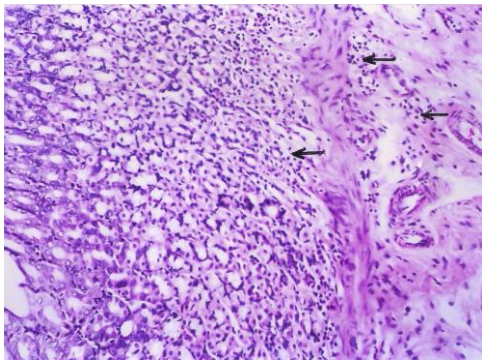

Photo 4. Stomach of rat from group 4(15% ALE) showing odema with few inflammatory cells infiltration in the submucosal layer (H and Ex 40)

effectively treated stress induced ulcers in animal models. The mechanism of antiulcer activity involves acceleration of mucus excretion through increasing the synthesis of glycoprotein at gastric mucosa, prolonging the life of the epithelial cells and antipepsin activity (**WHO, 2010**). However, WHO report warning against use of large doses of *G. glabra* (> 50g/day) for extended period (> 6 weeks). So, the aim of the

present study was to investigate the treatment effect of licorice on peptic ulcer in rats using 10% and15% of ALE.

Mean values of BWG was increased in the treatment groups received ALE, as compared with the control groups. **Shalaby *et al.*, (2004)** recorded a significant increase in BWG as a result of orally administration of ethanol licorice extract. With regard to FI and FER, the results indicated that FI was not significantly different between all groups. FER was significantly different, it was relatively high in the treated groups especially group 4 which administer ALE in the concentration of 15%. These increases in FER may reflect the recorded increase in BWG in corresponding groups. **Shalaby *et al.*, (2004**) in their study shows that the protective effect of ethanol licorice extract against peptic ulcer in rats recorded a significant decrease in feeding intakes and marked increases in FER as a result of protective administration of ethanol licorice extract.

The results of ulcer index (UI) and curative (%) of the experimental groups indicated significant marked UI in positive control group. In the treatment groups the results indicated a significantly reduction in UI as a result of ALE in dose related manner. The reduction in UI as a result of ALE in 10% concentration is significant but significantly less than treated with the concentration of 15% of ALE. The results of curative (%) were reflection of reduction in UI, either in treatment pattern. **Mesut *et al.*, (2009)** in their study to evaluate the comparative effectiveness of licorice vs. omeprazole and misoprostol for treatment peptic ulcer induced by aspirin in rats, found that licorice can be used in treatment, but not protection from NSAID-induced ulcers. Also, **Fatima *et al.*, (2008) and Memariani *et al.*, (2017)** in their study suggested that licorice could be a good source of alternative medicine for ulcer therapy. Furthermore, **Revers, (1956**) was one of the first authors to systematically study anti-ulcer properties of licorice extract. In an un-blinded and un-controlled study, 45 patients with confirmed gastric ulcers were given 10 g/day of powdered licorice extract (duration unknown). The ulcers were found to disappear in 17 of the cases, were diminished in 22 cases, and were unchanged in six of the cases. Patients with duodenal ulcers did not react as favorably. The author added that uncertainty still remains surrounding the involvement of glycyrrhizate compounds in the anti-ulcerogenic effect of licorice.

The results of the effect of ALE on gastric juice pH on rats suffering from peptic ulcer indicated that an ulcer induction cause significant increases in gastric juice pH. Treatment with ALE was significantly decreasing the gastric juice acidity. The decrease in gastric juice acidity is due to the anti-pepsin effect of licorice constituents. **Chaturvedi, (1979)** reported that oral administration of glycyrrhizin to 15 patients with peptic ulcer reduced symptoms and improved healing in 75% of the cases. **Adel *et al.,*** (**2005**) reported that licorice prolongs the life span of surface cells in the stomach and has an anti-pepsin effect. The combined effect may lead to the healing of ulcers. Licorice has also been shown to help inhibit the growth of potentially harmful intestinal bacteria, such as *Helicobacter pylori*, through the flavonoids that it has.

Results of the effect of ALE in case of induced peptic ulcers on antioxidant/oxidant stress balance as measured by balance between reduced glutathione and malondialdehyde are illustrated indicated that peptic ulcer induction in group 2 (+ve control) significantly decrease the antioxidant reduced glutathione and increase the oxidant stress substance MDA. Administration of aqueous licorice extract in concentration of 10% or 15% reduce the adverse effect of induced peptic ulcer and significantly increase the levels of serum glutathione and significantly decreases the level of serum MDA in dose related manner. These result more or less were in agreement with the results reported by **(Muralidharan *et al.*, 2009; Hye-Jin *et al.*, 2010; Kanimozhi and Karthikeyan, 2011; Makky *et al.*, 2012; Hajiaghamohammadi *et al.*, 2016 and Memariani *et al.*, 2017)** which showed the improvement effect of ALE on antioxidant and oxidant stress balanced related to the antioxidant content of licorice.

The histopathological changes as a result of aspirin induced peptic ulcer and the effect of treatment or protective effect of ALE administration indicated that peptic ulcer induction of experiment (+ve control group 2) cause moderate to mild histopathological alterations in stomach mucosa of rats. Treatment with ALE ameliorates these pathological changes to great extent especially in case of treatment with 15% ALE. These histopathological changes conform to treatment and protective effect of ALE on peptic ulcer as measured by ulcer index and curative percent.

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تأثير المستخلص المائي للعرق سوس علي قرحة المعدة المستحثة بالأسبرين في الفئران

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أجريت الدراسة الحالية بهدف التحقق من التأثير العلاجي للمستخلص المائي الساخن للعرق سوس على قرحة المعدة المستحث بالأسبرين في الفئران. واستخدم لهذه الدراسة ثمانية وعشرون فأراً من الذكور البالغين تم تقسيمهم الى مجموعتين رئيسيتين. المجموعة الأولى (7 فئران) تم تغذيتها على الوجبة الأساسية كمجموعة ضابطة سالبة، المجموعة الثانية (21 فأر) تم إعطاؤها الأسبرين عن طريق الفم بتركيز 400 ملليجرام / كجم من وزن الجسم على معدة فارغة للحث على الإصابة بقرحة المعدة ثم تم تقسيمها الى ثلاث تحت مجموعات على النحو التالى: تحت المجموعة الأولى، تم تغذيتها على الوجبة الأساسية كمجموعة ضابطة موجبة، تحت المجموعة الثانية، تم تغذيتها على الوجبة الأساسية كمجموعة ضابطة موجبة مضافا اليها 10 % مستخلص العرق سوس المائى، تحت المجموعة الثالثة، تم تغذيتها على الوجبة الأساسية كمجموعة ضابطة موجبة مضافا اليها 15% مستخلص العرق سوس المائى، واستمرت التجربة لمدة 28 يوم. وأظهرت التحاليل البيوكيميائية و الفحص النسيجي للمعدة للمجموعات العلاجية أن العرق السوس له تأثير علاجي قوي على القرحة المعدية عند التركيزات التى تم إختبارها (10، 15%) مقارنة بالمجموعة الضابطة الموجبة. لذلك، فإن نتائج هذه الدراسة تشير إلى أن العرق سوس يمكن استخدامة لعلاج القرحة المعدية ويمكن أن يحمي المعدة ضد العقاقير المضادة للالتهابات التي قد تسبب القرحة.

الكلمات المفتاحية: العرق سوس، القرحة المعدية، الأسبرين، تركيز الأس الأيدروجيني للمعدة، الجلوتاثيون، المالونالدهيد.