Anti-ulcer effect of Jasminum grandiflorum L. Extract on Gastric Ulceration in Rats

Eman S. Ibrahem2* , Aml F. EL-Ghazar1 , Fathia K. Gamal1

1: Nutrition and Food Science Dept., Home Economics Faculty, Helwan University

2: Regional Center for Food and Feed, Agricultural Research Center, Giza, Egypt.

*Corresponding author: profemy2000@gmail.com

Abstract

Globally, gastric ulcer is one of the most serious diseases. Although, there are several antiulcer drugs, however most of them have adverse reactions. This research aims to investigate the protective effects of Jasminum Grand Flower Extract (JGFE) in three levels against indomethacin (IND)-induced gastric ulcer in rats. Gastric ulcer was induced by a single oral dose of IND (30 mg/ kg). JGFE were administered orally at a dose of 200,400 and 600 mg/ kg b.wt. for 14 days prior to IND ingestion. Gross evaluation of gastric mucosal lesions showed that JGFE at the three tested levels significantly ($P<0.05$) diminished ulceration of surface epithelium, and maintained the normal histological structure of gastric mucosa induced by IND. Besides, significant ($P<0.0.5$) reduction in length of Gastric Ulcer, volume of gastric juice as well as decreased serum malondialdehyde as compared with the +ve group. Pretreatment with JGFE at the three tested levels significantly ($P<0.05$) increased the gastric PH and serum Superoxidase dismutase, catalase and glutathione. Moreover, JGFE at 400 and 600 showed a better ulcer healing capacity as compared with the dose of 200 mg/kg b.wt. Conclusion, pretreatment with JGFE alleviated gastric mucosal ulceration due to its high activity of active antioxidant.

Keywords : Jasminum grandiflorum L., Extract, Gastric ulcer, Indomethacin, Antioxidant

INTRODUCTION

The gastric mucous membrane is a thick layer of mucus that acts as a barrier against invading pathogens, also protects the underlying tissues from the digestive juices. Gastric ulcers are a break in the mucosa of the stomach lining that penetrates through the muscular is mucosa and extends more than 5 mm in diameter **(Woolf and Rose, 2022)**. When various factors and components of mucosal defense are insufficient to limit injury to the mucosa, an ulcer form **(Wallace, 2008)**. Peptic ulcers are lesions that form when digestive juices containing hydrochloric acid (HCL) erode the lining of the gastric mucosa, which can lead to gastrointestinal bleeding, stomach perforation, and even stomach cancer **(Mota et al., 2009)**. Gastric damage and ulcers can be induced by drinking, smoking, stress, poor diet and pathogenic infections. High concentrations of ethanol directly corrode the gastric mucosal tissue, causing inflammation, mucosal congestion, edema, bleeding, mucosal erosion and ulcers **(Rocco et al., 2014).** Although the exact pathological basis of gastric ulcer is unknown, oxidative stress and inflammation have been implicated as the main driving factors **(Koc et al., 2020).** Oxidative damage to the gastric epithelial and the ensuing apoptosis also play an important role in the progression of gastric diseases **(Zeng et al., 2017)**.

Gastric ulcers repair is a highly organized and complicated process involving inhibition of inflammation and promotion of cell proliferation, granulation tissue formation and angiogenesis **(Wallace, 2008).**

In addition, phytochemicals and several kinds of flavonoids have also demonstrated therapeutic effects against gastric injury with fewer side effects **(Miyazak et al., 2017).** Jasminum grandiflorum Linn. (family: Oleaceae) is a medicinal plant that has been used to anti-inflammatory, antioxidant, anthelmintic, diuretic and for treatment of toothache, ulcer, stomatitis, and wounds treat spasmolytics, infections, skin diseases such as conjunctivitis and dermatitis, mental illness and cancer **(Arun et al., 2016).** The flower of J. grandiflorum is also widely consumed as a beverage with heat-clearing and detoxifying effects in China. It has been reported that the ethanolic extract from the leaves of J. grandiflorum exerts antiulcer and antioxidant activities in rats **(Umamaheswari et al., 2007)**. Several bioactive compounds have been isolated from J. grandiflorum, including iridoid-type compounds, secoiridoid

glucosides, triterpenes, flavonoids and lignans **(Arun et al., 2016 and Zhao et al., 2009).** Because of the valued medicinal properties of this plant, there have been efforts to conserve it.

The aim of the present study is to investigate the protective effects of Jasminum Grand Flower Extract at three levels against indomethacin induced gastric ulcer in rats.

Materials and Methods

Animal: adult male albino rats of Sprague Dawley strain (n=35) weighing 180±5g were purchased from Helwan Experimental Animals Farm, Egypt. **Plant:** Jasminum flower were purchased from the local market, then was identified at the Agricultural Research Centre, Cairo, Egypt.

Diet: Basal diet constituents were obtained from El- Gomora Company, Cairo, Egypt. **Chemicals and Kits:** Indomethacin (IND) was purchased from Hikma Pharmaceuticals PLC, Amman, Jordan, (provided as 25 mg/capsule). Other chemicals and reagents were purchased from Sigma-Aldrich (Sigma Company, Cairo, Egypt).

Preparation of dried Jasminum flower: Fresh Jasminum flowers were washed with tap water and soaked in a water bath to remove the possible potential Pathogenic microorganisms and dust. Afterwards, the Jasminum was dried by Cotton cloth to remove the excess liquid prior to drying by Solar Energy. Then a grinder mill and sieves were used to obtain a powder particle. Preparation of Jasminum flower extract: The powdered Jasminum Flowers were extracted with 90% ethyl alcohol and concentrated at low Temperature (50°C) using a Rotary evaporator apparatus. Dried ethanol Extract was dissolved in a mixture of carboxyl methylcellulose and few Drops of Tween 80 as a suspending agent to obtain 10% concentration liquid Extract **(Brusotti et al., 2014)**.

Preparation of basal diet: The basal diet (AIN-93M) was formulated to meet the recommended nutrients levels for rats according to **(Reeves et al.,1993).**

Induction of gastric ulcer: Gastric ulcer was induced for all rats at the end of the experiment except for the negative control group as described **by Bhattacharya et al., (2007).** The animals were fasted for 24 h before oral administration of a single dose of IND (30 mg/kg b.wt.). Different degrees of gastric mucosal injuries were detected 4 h after IND administration **(Chatterjee et al., (2012).**

Experimental design: The experiment was conducted using thirty-five male rats. The animals were kept in healthy condition at room temperature (20-25 °C), exposed to a 12:12-h light-dark cycle and fed on the basal diet, and water were provided ad libitum for one week before starting the experiment for acclimatization. After the acclimatization period, rats were randomly divided into two main groups: The first main group (7 rats), which was presented as a negative control group (-ve) and was fed only on the basal diet. The second main group was divided into 4 subgroups (7 rats each), as follows: The first subgroup was kept as a positive control group (ve+) and was fed only on the basal diet. The other Subgroup were fed basal diet and given orally with Jasminum flower extract at a dose of 200, 400, 600 mg/kg b. wt. respectively for 14 days.

Determination of feed intake (FI), body weight determined every day. Body weight gain percent (BWG%) and feed efficiency ratio (FER) at the end of the experimental period were calculated using the following formulas:

$$\textbf{Body weight gain percent (\%)} = \frac{\text{Final body weight (g)} - \text{Initial body weight}(g)}{\textit{intial body weigh t}} \times 100$$

$$\textbf{Feed efficiency ratio (FER)} = \frac{\text{Body weight gain (g)}/}{\text{Food consumed (g)}}$$

At the end of the experimental period (14 days) the second main group were fasted and given the prescribed dose of IND. After 4 h post IND administration, rats were sacrificed, blood samples were collected, serum samples were separated and kept frozen until used for biochemical determination.

The stomach was dissected out and cut along its greater curvature, then its content was evacuated into a centrifuge tube, diluted with distilled water and centrifuged at 12000 g for 10 min. Gastric pH and total gastric acidity were detected in the supernatant. The stomach was kept clean in 10% formalin solution and processed for macroscopic examination and histopathological examination.

Gastric ulcer index: The method described by **Agrawal et al., (2000)** was employed in the present study. In brief, after 4 hours of aspirin administration, all rats were sacrificed after using an overdose of diethyl ether and their stomachs removed and washed with saline. The gastric juice was collected in a test tube. Then stomachs were opened along the greater curvature, washed with saline and examined under a dissecting microscope for gastric ulcers. The sum of the length of all lesion areas for each animal was measured and served as the ulcer index.

The Curative ratio (CR) was calculated using the following equations:

The Curative ratio (CR) was calculated using the following equations:

$$\textbf{Curative ratio (CR)} = \frac{LC-LT}{LC} \times 100$$

LC: The length of gastric ulcer in positive group.
LT: The length of gastric ulcer in treated group.

Determination of gastric juice volume:

Gastric juices from all groups were collected in test tubes, centrifuged at 5000 r.p.m. for 10 minutes and their volume of were measured by a graduated cylinder. percentages of the decrease in volume of the gastric juice of the treated groups compared to the (+ve) control group were calculated according to the method described by **Agrawal et al., (2000)** using the following equation:

Percentage of the decrease = VJC -VJT ×100
Where: VJC = volume of gastric juice of the positive control group
VJT = volume of gastric juice of the treated group.

Determination of gastric pH: The gastric juice was collected by a needle after tying the neck and the end of the stomach. Gastric juice (1 ml) was diluted by distilled water (1ml) in an aliquot to measure pH using pH meter **(Dashputre and Naikwade, 2011).**

Determination of serum antioxidant biomarkers: Oxidative stress biomarkers (malondialdehyde (MDA), superoxide dismutase (SOD), glutathione (GSH) and catalase (CAT)) were determined in serum using ELISA kits obtained from My Biosource, San Diego, California, USA.

Histopathological examination: The formaldehyde fixed stomach is paraffin-embedded, cut into sections, then stained with Hematoxylin-Eosin (H&E). The slides were examined microscopically.

Statistical analysis: Ulcer Inhibition was expressed in percentage. Results are reported as mean ± SE. Data were compared by one-way analysis of variance (ANOVA), followed by LSD using SPSS version 25. $P < 0.05$ indicates significance difference **(Armitage and Berry, 1987).**

Results and Discussion

The obtained results in table (1) showed no significant differences between all groups in the initial body weight and final body weight. Moreover, Indomethacin treated groups, at levels (200 and 600) had no significant differences ($P<0.05$) in the main values of BWG%, however, there was a significant decrease in BWG% for the group treated with 200 mg/kg b.wt as compared to the other treated groups. On the other hand, there was a significant decrease ($P<0.05$) in the main value of FER between treated groups and the positive or negative control groups.

These results were in the same line as the result of **(Ramadhan and Yuniarto, 2017)** who found that Jasminum extract dose at 100, 300, 500 mg/kg BW indicated a significant decrease in body weight, fat index and organs index compared to control rats. Observations on feed intake were based on the mechanism of action of JGFE that can reduce body weight by reducing appetite, and decreased appetite leads to lower calorie intake and body weight **(Lestari, 2007)**

Table (1) Effect of Jasminum grandiflorum L. Extract on daily feed intake and body weight gain percent in rats with gastric ulcer

Parameters / Groups		IBW (g)	FBW (g)	BWG (%)	FER	FI (g\day)
Negative control group (-ve)		207.21±2.63[a]	233.34±2.07[a]	12.63±0.52[a]	0.11±0.01[a]	17.01
Positive control group (+ve)		208.42±2.29[a]	234.01±2.70[a]	12.28±0.45[a]	0.10±0.01[a]	17.25
Treated groups with IND+ JGFE at	**200**	±0.61[a]٩,٢٢20	229.20±1.16[ab]	9.45±0.28[a]	0.06±0.00[b]	20.40
	400	±1.32[a]١٩.05.٢	224.41±1.28[b]	7.06±0.48[b]	0.04±0.01[c]	21.40
	600	205.00±1.84[a]	218.81±2.05[b]	6.73±0.50[c]	0.05±0.01[c]	19.81

Value expressed as means ± SE

Means with different letters in each Colum are significantly differs at ($P < 0.05$).

The positive control group had highly significant increase ($P < 0.05$) in the main value of length of gastric ulcer and gastric juice volume as compared to the -ve group as seen at Table (2), moreover IND groups treated with JGFE at all levels (200, 400 and 600 mg/kg b.wt.) had significant decrease ($P < 0.05$) in the main values of length of gastric ulcer and gastric juice volume as compared to the + ve group.

Regarding to the PH in the positive control group there was a significant increase ($P<0.05$)) in the mean value of the PH as compared to the -ve group or other treated groups. On the other side treated groups with JGFE at all levels had significant increase ($P<0.05$)) in the mean values of PH as compared to the positive control group. The most protective dose of JGFE was 600 mg/kg b.wt.

These results were in the same line as the result of **Bech et al., (2000)** found that IND, caused a significant increase in curative Ratio and PH. The obtained results revealed that IND groups treated with JGFE at all levels had significant decrease in the main value of the length of gastric ulcer (mm) and gastric juice volume as compared to the +ve group. Inhibitory action of indomethacin on prostaglandin synthesis which due to the presence of alkaloids, glycoside, flavonoid, terpenes, tannin, resin, and salicylic acid **(Priya et al., 2008).**

Table (2): Effect of Jasminum grandiflorum L. Extract on The Length of Gastric Ulcer (Mm), Volume of Gastric Juice, PH and Curative Ratio in Rats with Gastric Ulcer.

Groups \ Parameters		length of Gastric Ulcer (mm)	Gastric Juice Volume (µg)	PH	Curative Ratio %
Negative control group (-ve)		00.00±0.00^{e}	101.53±1.04^{e}	6.11±0.04^{a}	100.0
Positive control group (+ve)		83.83±1.17^{a}	415.14±1.01^{a}	1.08±0.06^{d}	00.00
Treated groups with IND+ JGFE at	**200**	52.09±0.65^{b}	321.18±0.43^{b}	2.83±0.05^{c}	37.83±0.82
	400	30.68±0.47^{c}	251.44±0.71^{c}	2.99±0.12^{bc}	63.36±0.71
	600	18.53±0.61^{d}	142.93±2.01^{d}	3.13±0.05^{b}	77.87±0.81

Results are expressed as mean ±SE

Values at the same column with different letters are significantly at ($P < 0.05$)

There was a significant decrease ($P < 0.05$) in the mean value of catalase, GSH and SOD for the positive control group as compared to the -ve group. Moreover, treated groups with different levels of JGFE had significant increase ($P < 0.05$) in the main value of Catalase, GSH and SOD as compared to the +ve group. On the other hand, concerning to the serum levels of MDA, the +ve group had significant increase ($P < 0.05$) in the mean value of MAD as compared to the -ve group. Moreover, IND groups treated with JGFE at all levels had significant decreased ($P < 0.05$) in the main value of MAD as compared to the +ve group. The highest improvement of antioxidant status was observed at the group treated with 600 mg/kg b.wt.

Mansouri et al., (2015) found that the increased concentration of MDA as well as reduced activity of SOD in the stomach of indomethacin-ulcerated rats is a manifestation of facilitated lipid peroxidation and over production of free radicals resulting in mucosal damage. Moreover, **Murat et al., (2016)** found that IND significantly decreased the GSH levels in the liver, kidney, and brain tissues of the treatment groups compared with the control groups. Our results are in line with a reported study, where GSH level was quite low in the IND group when compared to all other experimental groups resulting in gastric ulceration.

The reduction in the activities and level of this antioxidant system in this study suggest overwhelming generation of ROS and exhaustion of these antioxidant cascades to cope with their detoxification resulting in their accumulation in the liver and kidney of rats. Our result is similar to the previous **(Zahran et al., 2020).** On the other wise elevated MDA levels indicate oxidative damage and act as oxidative stress indicators **(Geyikoglu et al., 2018)**

Also results were in agreement with **Bafna et al., (2005)** who found that rats fed on damascene Rosa have a significant decrease in serum level of MAD and increase in serum activities of GSH and SOD enzymes compared with the +ve groups.

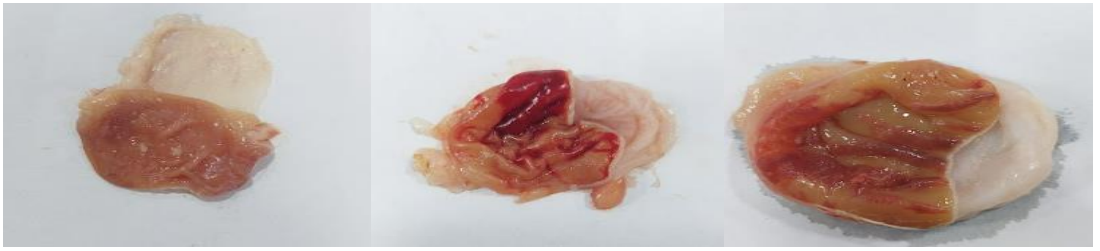
Table (3) Effect of Jasminum grandiflorum L. Extract on serum Catalase, Malondialdehyde, Reduced Glutathione and Superoxide Dismutase in rats with gastric ulcer.

Parameters / Groups		Catalase (µmol/L)	MDA (µmol/L)	GSH (mg/L)	SOD (µm/L)
Negative control group (-ve)		5.25±0.052^{a}	0.85±0.019^{e}	4.59±0.13^{a}	3.89±0.26^{a}
Positive control group (+ve)		1.05±0.027^{e}	4.65±0.08^{a}	0.95±0.016^{e}	0.91±0.03^{e}
Treated groups with IND+ JGFE at	**200**	2.65±0.084^{d}	3.59±0.14^{b}	1.85±0.096^{b}	1.57±0.13^{d}
	400	3.25±0.101^{c}	2.91±0.07^{c}	2.88±0.087^{c}	2.34±0.11^{c}
	600	4.14±0.043^{b}	1.59±0.10^{d}	3.61±0.143^{d}	2.96±0.12^{b}

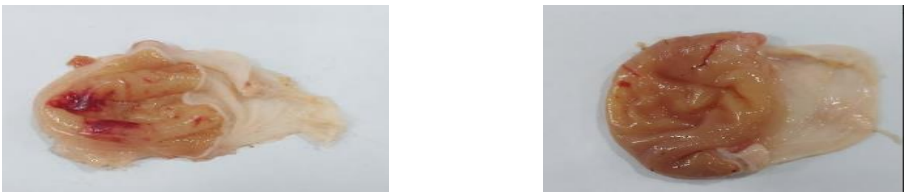
Results are expressed as mean ±SE Values at the same column with different letters are significantly at ($P < 0.05$)

Gastric gross structure: In the IND group, the stomach showing diffuse and multiple ulcer formation with depressed center and elevated borders. While stomach of the JGFE 200+IND pretreated group showing starting of ulcer healing signs. In the JGFE 400+IND pretreated group stomach showing advanced ulcer healing signs. While in the JGFE 600+IND pretreated group the stomach showing complete ulcer healing with apparently healthy intact mucosa was seen (Photo. 1). The stomach of the –ve control group showing intact mucosa. In IND group the stomach showing diffuse multiple ulcer formation with depressed center and elevated borders. While stomach of JGFE 200+IND

pretreated group showing starting of ulcer healing signs. Stomach of JGFE 400 +IND pretreated group showing advanced ulcer healing signs. In JGFE 600 +IND pretreated group the stomach showing complete ulcer healing with apparently healthy intact mucosa.



Negative control group IND group 200 JGFE group



400 JGFE group 600 JGFE group

Photo. (1): Gastric gross structure

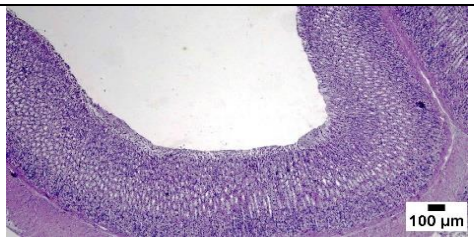
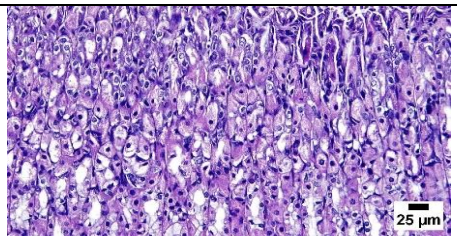
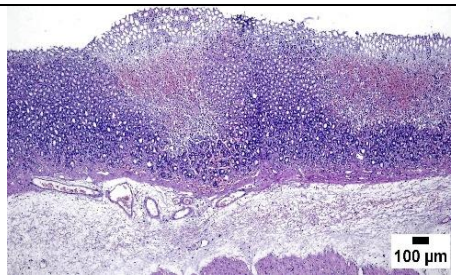
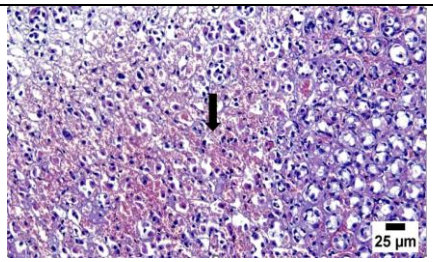
Histological examination of control group revealed normal structure of the glandular gastric mucosa and submucosa **(Photo. 2-3).** In contrast to the +ve group, serious histopathological alterations were detected. diffuse ulceration was detected in the glandular mucosa which characterized by desquamation of the epithelial lining admixed with hemorrhages and accumulation of necrotic tissue. Numerous sections showed excessive inflammatory cells infiltration in the submucosal layer. The submucosa layer revealed dispersion of the connective tissue with abundant edema, congested blood vessels and inflammatory cells infiltration (**Photo. 4-5).**

Mild improvement was detected in a group treated with JGFE at 200 mg/kg b.wt. which was characterized by epithelial sloughing with variable number of inflammatory cells infiltration in the submucosa, necrosis of glandular acini and multifocal hemorrhages. Cystic dilation of gastric was less frequently observed

(Photo. 6-7). Group treated with JGFE at 400 mg/kg b.wt. achieved higher protection compared to group 200 and PC group. Microscopic examination showed multifocal areas of mononuclear inflammatory cells infiltration in the submucosa with necrosis of mucosal surface in fewer sections. Several examined sections revealed apparently normal histological structure of glandular mucosa **(Photo. 8-9).** Examination of group treated with JGFE at 600 mg/kg b.wt. showed apparently normal glandular stomach in almost examined sections **(Photo. 10-11).**

Finally: pretreatment with JGFE alleviated gastric mucosal ulceration due to its high activity of active antioxidant.

Effect of Jasminum grand flower extract on the gastric tissue histopathological changes

<table>
<tr><td> 
</td><td> 
</td></tr>
<tr><td>Photo. 2: stomach from control group showing normal histological structure of stomach mucosa (H&E).</td><td>Photo. 3: stomach from control group showing normal histological structure of stomach mucosa (H&E).</td></tr>
<tr><td> 
</td><td> 
</td></tr>
<tr><td>Photo. 4: stomach from +ve group showing severe destruction of gastric mucosa with hemorrhages (H&E).</td><td>Photo. 5: stomach from +ve group showing severe destruction of gastric mucosa with extensive hemorrhages (arrow) (H&E).</td></tr>
</table>

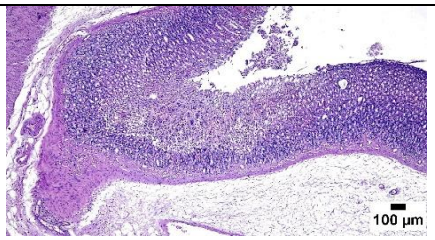


Photo. 6: stomach from group JGFE at 200 mg/kg b.wt. showing submucosal edema, necrosis of the glandular mucosa (**H&E**).

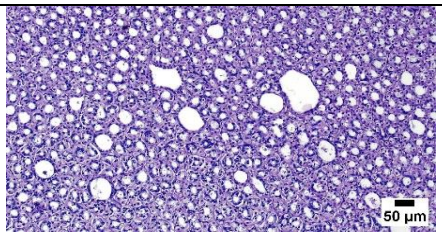


Photo. 7: stomach from group JGFE at 200 mg/kg b.wt. showing cystic dilation of some gastric glands (**H&E**).

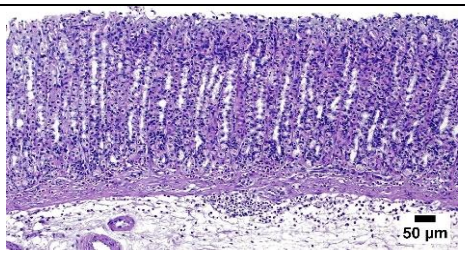


Photo. 8: stomach from group JGFE at 400 mg/kg b.wt. showing inflammatory cells infiltration in the submucosa (**H&E**).

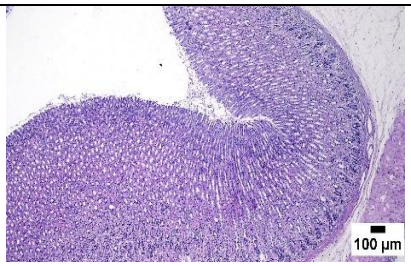


Photo. 9: stomach from group JGFE at 400 mg/kg b.wt. showing apparently normal gastric mucosa (**H&E**).

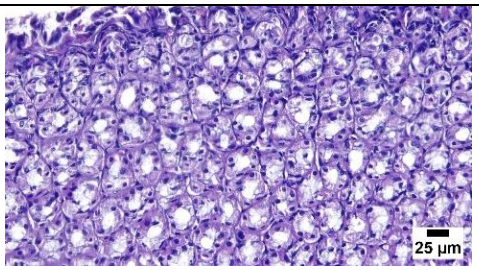


Photo. 10: stomach from group JGFE at 600 mg/kg b.wt. showing apparently normal gastric mucosa (**H&E**).

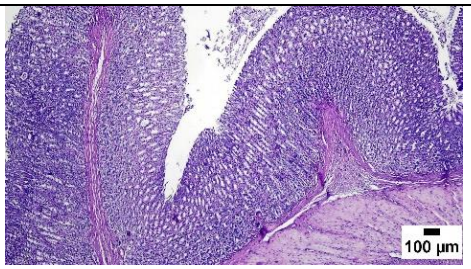


Photo. 11: stomach from group JGFE at 600 mg/kg b.wt.showing apparently normal gastric mucosa (**H&E**).

REFERENCES

Agrawal, A., Rao, C., Sairam, K., Joshi, V. and Goel, R. (2000). Effect of Piper longum Linn, Zingiber officianalis Linn and Ferula species on gastric ulceration and secretion in rats.

Armitage, G. and Berry W. (1987): Statistical methods 7th Ed Ames., Iowa State University. Press .39- 63.

Arun, M., Satish, S. and Anima, P. (2016). Phytopharmacological profile of Jasminum grandiflorum Linn.(Oleaceae). Chinese journal of integrative medicine, 22, 311-320.

Bafna, P. (2005): Balaraman R. Anti-ulcer and antioxidant activity of pepticare, a herbomineral formulation. Phytomedicine.;12:264–70.

Bech, P., Xavier, R., Lu, N.; Nanda, N., Dinauer, M. and Podolsky, D. (2000): Mechanisms of NSAID induced gastrointestinal injury defined using mutant mice. Gastroenterology. ;119(3):699–705.

Bhattacharya, S., Chaudhuri, S., Chattopadhyay, S. and Bandyopadhyay, S. (2007). Healing properties of some Indian medicinal plants against indomethacin-induced gastric ulceration of rats. Journal of Clinical Biochemistry and Nutrition, 41(2), 106-114.

Brusotti, G., Cesari, I., Dentamaro, A., Caccialanza, G. and Massolini, G. (2014). Isolation and characterization of bioactive compounds from plant resources: The role of analysis in the ethno pharmacological approach, Journal of Pharmaceutical and Biomedical Analysis, 87:218-28.

Chatterjee A., Chatterjee S., Biswas A., Bhattacharya S., Chattopadhyay S. and Bandyopadhyay, S. (2012). Acid. Enriched Fraction of Phyllanthus emblica Potentiates Indomethacin-Induced Gastric Ulcer Healing via. e-NOS-Dependent Pathway. Evid Based Complement. Alternat Med., 2012: 487380.

Dashputre, N.L. and Naikwade, N.S. (2011): Evaluation of antiulcer activity of methanolic extract of Abutilon indicum Linn leaves in experimental rats. Int. J. Pharm. Sci. Drug Res., 3:97-100.

Geyikoglu, F., Yilmaz, E. and Erol, H.S. (2018): Hepatoprotective role of thymol in drug-induced gastric ulcer model. Ann Hepatol. ;17(6):980–991

Koc, K., Cerig, S., Ucar, S., Colak, S., Bakir, M., Erol, H. S. and Geyikoglu, F. (2020). Gastroprotective effects of oleuropein and thymol on indomethacin-induced gastric ulcer in Sprague-Dawley rats. Drug and Chemical Toxicology, 43(5), 441-453.

Mansouri, E., Khorsandi, L. and Zare, M. (2015): Grape seed proanthocyanidin extract improved some of biochemical parameters and antioxidant disturbances of red blood cells in diabetic rats. Iran. J. Pharm. Res. .;1:329–34.

Miyazaki, Y., Ichimura, A., Sato, S., Fujii, T., Oishi, S., Sakai, H., and Takeshima, H. (2018). The natural flavonoid myricetin inhibits gastric H+, K+-ATPase. European journal of pharmacology, 820, 217-221**.**

Mota K., Dias G., Pinto M., Luiz-Ferreira A., Souza-Brito A., HirumaLima C., Barbosa-Filho J. and Batista L.M.(2009) Flavonoids with gastroprotective activity, Molecules 14: 979–1012.

Murat, B., Cavit, K., Selim, S., Hamdi, A., Erkmen, T. and Umit, K. (2016): The effects of lycopene on DNA damage and oxidative stress on indomethacin-induced gastric ulcer in rats| Volume 35, ISSUE 2, P428-435.

NH, L. (2007). Aktivitas antiobesitas rimpang rumput teki (Cyperus rotundus L.), bunga melati (Jasminum sambac (L.) Ait.) dan kombinasinya pada tikus yang diinduksi makanan tinggi lemak. Sekolah Tinggi Farmasi Bandung. Bandung**.**

Priya, J. and Raja, D. (2008). Anti-bacterial activity studies of Jasminum grandiflorum and Jasminum sambac. Ethnobot. Leafl. 2008, 12, 481–483

Ramadhan, M. and Yuniarto, A. (2017). Anti-obesity effect of the jasminum sambac (l.) W. ait. Ethanol extract in high fat diet induced mice. In 2nd Sari Mulia International Conference on Health and Sciences 2017 (SMICHS 2017)–One Health to Address the Problem of Tropical Infectious Diseases in Indonesia (pp. 351-358). Atlantis Press.

Reeves, P. Nielsen, F. and Fahmy, G. (1993): AIN-93. Purified diets for laboratory rodents: Final reports of the American Institute of Nutrition ad hoe 9 wriling committee of reformulation of the AIN-76 A Rodent Diet. J. Nutr., 123: 1939-1951.

Rocco, A., Compare, D., Angrisani, D., Zamparelli, M. S. and Nardone, G. (2014). Alcoholic disease: liver and beyond. World journal of gastroenterology: WJG, 20(40), 14652-14659.

Umamaheswari, M., Asokkumar, K., Rathidevi, R., Sivashanmugam, A., Subhadradevi, V. and Ravi, T. (2007). Antiulcer and in vitro antioxidant activities of Jasminum grandiflorum L. Journal of ethnopharmacology, 110(3), 464-470.

Wallace, J. (2008). Prostaglandins, NSAIDs, and gastric mucosal protection: why doesn't the stomach digest itself?. Physiological reviews, 88(4), 1547-1565.

Woolf, A. and Rose, R. (2022): Gastric ulcer. In StatPearls; StatPearls Publishing: Treasure Island, FL, USA,.

Zahran, R., Ghozy, A. and Elkholy, S. (2020): Combination therapy with melatonin, stem cells and extracellular vesicles is effective in limiting renal ischemia– reperfusion injury in a rat model. Int J Urol. ;27(11):1039– 1049.

Zeng, Q., Ko, C., Siu, W., Li, L., Han, X., Yang, L. and Leung, P. (2017). Polysaccharides of Dendrobium officinale Kimura & Migo protect gastric mucosal cell against oxidative damage-induced apoptosis in vitro and in vivo. Journal of ethnopharmacology, 208: 214-224.

Zhao, G., Yin, Z. and Dong, J. (2009). Antiviral efficacy against hepatitis B virus replication of oleuropein isolated from Jasminum officinale L. var. grandiflorum. Journal of ethnopharmacology, 125(2), 265-268.

المستخلص

التأثير المضاد للقرحة لمستخلص أزهار الياسمين L grandiflorum Jasminum. علي تقرح المعدة في الفئران

إيمان سامي إبراهيم[١] أمل فوزي الجزار[٢] ، فتحية خالد جمال الدين[٢]

١: المركز الإقليمي للأغذية والأعلاف – مركز البحوث الزراعية –الجيزة – مصر
٢: قسم التغذية وعلوم ألطعمة – كلية الاقتصاد المنزلي – جامعة حلوان

على الصعيد العالمي، تعتبر قرحة المعدة هي واحدة من أخطر الأمراض. على الرغم من وجود العديد من ألأدوية المضادة للقرحة، إل أن معظمها له ردود أفعال سلبية. يهدف هذا البحث إلى التحقق من التأثيرات الوقائية لمستخلص زهرة الياسمين في ثالثة مستويات ضد قرحة المعدة التي يسببها عقار إلندوميثاسين (IND) في الفئران .تم إحداث قرحة المعدة بجرعة فموية واحدة من ٣٠ IND ملجم / كجم من وزن الجسم .تمت المعالجة بمستخلص زهرة الياسمين الكحولي عن طريق الفم بجرع ات ٢٠٠و ٤٠٠ و ٦٠٠ ملجم / كجم من وزن الفأر لمدة ١٤ يوما قبل اعطاء جرعة .IND أظهر التقييم إلجمالي للتقرحات الموجودة في المعدة أن المستخلص الكحولي بالنسب الثالثة المختبرة قللت من تقرح سطح المعدة بشكل ملحوظ ($p<0.05$) وحافظت على التركيب النسيجي الطبيعي للغشاء المخاطي في المعدة للمجموعات التي تم إعطائها جرعة الـ .INDإلى جانب ذلك، حدث انخفاض معنوي ($p<0.05$) في طول قرحة المعدة ، وحجم عصير المعدة وكذلك انخفض مستوى المالوند اه يد في السيرم مقارنة مع المجموعة الضابطة الموجبة. كما أدت المعالجة المسبقة باستخدام مستخلص زهرة الياسمين عند المستويات الثالثة المختبرة بشكل ملحوظ إلى زيادة معنوية ($p<0.05$) في درجة حموضة المعدة ومستويات SOD ,CAT and GSH في سيرم الدم . وعلاوة على ذلك، أظهرت المجموعات المعالجة بمستخلص زهرة الياسمين عند ٤٠٠ و ٦٠٠ قدرة أفضل في التئام القرحة مقارنة بجرعة ٢٠٠ ملجم/كج م من وزن الفأر. الخالصة: المعالجة المسبقة باستخدام مستخلص زهرة الياسمين أظهرت ت حسن ملحوظ لكل من الغشاء المخاطي للمعدة بسبب النشاط العالي لمضادات الأكسدة النشطة .

الكلمات الدالة :زهرة الياسمين ، مستخلص ، قرحة المعدة ، الإندوميثاسين ، مضادات الأكسدة.