

The Corrosive Esophageal Injury Induced by NaOH and the Possible Protective Role of Kefir in Adult Albino Rat: A Histological and Scanning Electron Microscopic Study

Original Article

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ABSTRACT

Background: Corrosive substances' impact on the esophagus is a significant health concern, with an acute onset of mucosal injury. Kefir has antibacterial and immunomodulatory properties.

Aim of the Work: To study the potential protective effect of kefir on the esophageal injury induced by NaOH in adult male albino rats.

Material and Methods: Twenty-five adult male albino rats were randomized into: Group I (10 rats) was subdivided equally into: IA, negative control, IB received one ml of kefir once daily orally for seven days; group II (5 rats) received one ml of 5% NaOH orally and were sacrificed after one day. Group III (5 rats) received 1 mL of 5% NaOH orally and were left for 7 days. Group IV (5 rats): One ml of 5% NaOH was administered orally, and then one ml of kefir was administered once daily for seven days. Some esophageal specimens were processed and stained with H&E and Masson's Trichrome, and others were processed for scanning electron microscopy. Statistical analysis was done for some parameters.

Results: The corrosive esophagitis group (II) showed a loss of mucosal folds, thinning, and separation of the esophageal layers. The spontaneous healing group (III) displayed preservation of all layers, along with short mucosal folds, thin epithelium, and congested blood vessels. The kefir-treated group presented intact mucosal layers and preserved mucosal folds, with continuous keratin.

Conclusion: Kefir supplementation may contribute to better control of corrosive esophageal injury, thereby reducing the risk of esophageal stricture.

Key Words: Esophagus, kefir, NaOH, scanning microscopy, ulcer.

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INTRODUCTION

Corrosive injury to the esophagus is a major public health issue worldwide, especially in children^[1], with 90% of cases attributed to alkaline chemicals such as potassium and sodium hydroxide, leading to varying degrees of esophagitis and life-threatening perforation^[2]. Corrosive compounds, present in household items such as bleaches and detergents, can lead to serious harm to the esophagus and stomach^[3]. Chemical substances like hydrochloric and sulfuric acids can lead to esophageal and gastric stenosis more frequently than acidic toxicity^[4]. Exposure to large amounts of these substances can lead to injury of the skin, upper gastrointestinal tract, and respiratory tract, ranging from mild irritation to potentially life-threatening conditions^[5].

Esophageal burn injury treatment typically involves rest, stopping oral feedings, and administering intravenous fluids^[5]. Various treatments such as antibiotics, steroids, anti-reflux medication, stenting, and dilatations are utilized to prevent additional harm, although there is no widely agreed-upon preventive treatment. However, the priority is still for the urgent treatment, which limits the burn area with neutralizing chemical drugs^[6].

Kefir, a kind of fermented milk belonging to the Caucasus region in origin, is commonly consumed for its beneficial health effects^[7]. It is formed by fermenting milk with kefir grains, yeasts, acetic acid, and lactic acid bacteria. Research revealed that kefir has many positive aspects^[8], such as regulating glucose and cholesterol levels, having anti-hypertensive and anti-inflammatory effects^[9].

Kefir has been found in numerous studies to have antibacterial, antifungal, and immunomodulatory properties^[10]. Despite the utilization of various chemical agents and pharmacological drugs in the management of corrosive esophagitis, the application of Kefir has not been investigated as a therapeutic modality to date. Therefore, the current study aimed to explore the possible protective effect of kefir on the esophageal injury induced by NaOH in adult male albino rats.

MATERIAL AND METHODS

Drug: Kefir grains were obtained from Hikma Pharmaceutical Company in Cairo, Egypt. A total of 10 grams of the grains were mixed with 100 ml of pasteurized full-fat milk. This admixture was incubated at 23°C for 12 hours. After filtration, the grains were removed, and the resulting kefir extract was kept at 4°C. The extract was then administered orally using a gastric tube that did not extend to the cardia^[10].

Animals: Twenty-five adult male albino rats, aged 3 to 6 months and weighing 200 to 250 grams, were acquired from the Animal House of the Research Institute (MASRI) at Ain Shams University. The rats were housed in typical wire-mesh cages in a controlled environment with alternating light and dark cycles, a temperature of approximately $21 \pm 10^\circ\text{C}$, and a humidity level between 45% and 50%. Ad libitum food and water were allowed. Ten days before the experiment, the rats were housed to allow them to acclimate to the experimental conditions.

ETHICS COMMITTEE

Approval of the study protocol (Reference No: FMASU AnimalR100/2025) had been received from the Institutional Animal Care and Use Committee (ACUC) and Research Ethics Committee (FMASUS REC), Faculty of Medicine- Ain Shams University on 15/4/2025. Protocols of animal care and maintenance followed the ARRIVE guidelines.

Experimental groups: four rat groups were randomly assigned as follows:

Group I: (Control group): ten adult male albino rats, further subdivided into two subgroups, 5 rats each: subgroup Ia (negative control); not receiving any intervention, subgroup Ib (sham control); received 1 ml of kefir once per day for 7 days. Both subgroups were sacrificed on the 7th day.

The rats in groups II, III, and IV were anesthetized via subcutaneous injection of a combined solution consisting of ketamine (50 mg/kg; Ketalar, Pfizer) and xylazine (10 mg/kg; Bimeda). Following induction of anesthesia, the esophagogastric junction was occluded using a 2 mm diameter Foley catheter. During the procedure, the

animals were maintained in a semi-horizontal position at approximately 60° to the horizontal plane to minimize the risk of aspiration^[11]

Group II: (corrosive esophagitis group) (5 rats): received 1 ml of 5% NaOH orally via gastric tube, not reaching the cardia. Sacrifice was done after one day.

Group III: (spontaneous healing group) (5 rats): received 1 ml of 5% NaOH orally via gastric tube, not reaching the cardia, followed by esophageal wash using distilled water, and the animals were sacrificed on the 7th day.

Group IV: (kefir-treated group) (5 rats): received 1 ml of 5% NaOH once orally via gastric tube, not reaching the cardia, then received 1 ml of kefir once daily for 7 days, then sacrificed^[12].

At the end of the experimental period, animals will be euthanized humanely in accordance with the AVMA Guidelines for the Euthanasia of Animals (2020)^[13] using intraperitoneal injection of ketamine (50 mg/kg; Ketalar, Pfizer) and xylazine (10 mg/kg; Bimeda). Median laparotomy was performed to explore the distal esophagus, and almost 1.5 cm of the esophageal segment was dissected.

Light microscopic study:

Specimens were fixed in 10 % neutral formalin for 24 hours, processed into paraffin blocks, and 5 µm sections were cut and stained with Hematoxylin & eosin and Masson's trichrome^[14] to be examined by an Olympus light microscope (CX31).

Scanning electron microscopic study:

Specimens were fixed in 2.5% glutaraldehyde, dehydrated in a graded series of ethanol, and finally dried. After coating with a layer of gold^[15]. All specimens were studied using a scanning electron microscope (XL30, Philips, Amsterdam, Netherlands) in the Electron Microscopic Unit in the Agricultural Experimentation Station, Faculty of Agriculture, Cairo University.

Morphometric study and Statistical analysis:

Measurements of rats' body weight were obtained at the beginning and the end of the experiment for statistical analysis. Collagen fiber deposition area percentage was measured in Masson's trichrome-stained sections at a magnification of x200 in ten non-overlapping fields from the experimental groups using Image J software version 1.50i.

These data were analyzed using SPSS version 22 statistical package. Software (La Jolla, US) was used for statistical analysis and represented as mean $\pm$ standard deviation (SD). Group comparisons were done through analysis of variance (ANOVA) with a multiple comparisons

post hoc test to compare more than 2 groups. The difference was considered statistically significant when p (probability) < 0.05 and highly significant when $P < 0.001$.

RESULTS

Histological results:

Examination of H&E-stained sections of the control subgroup revealed almost similar findings; the rat esophagus was composed of mucosa, submucosa, muscularis externa, and adventitia. The mucosa appeared folded and formed of stratified squamous epithelium covered by a thin layer of keratin, lamina propria, and muscularis mucosae. The submucosa, vascular connective tissue layer, was observed, followed by two layers of muscularis externa: inner circular and outer longitudinal (Fig. 1a). The keratinized epithelium appeared with a layer of cells with granules (granular layer) and a basal layer of dark-staining cells. A dense connective tissue layer called the lamina propria was detected below the epithelial layer, separating it from bundles of smooth muscles that form the muscularis mucosae (Figure 1 b).

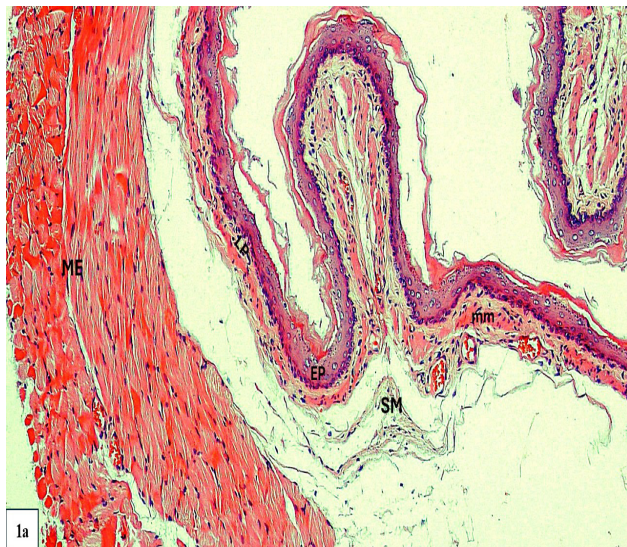


Fig. 1: A photomicrograph of a section of rat's esophagus of the control group showing that: a) the layers of rat esophagus: the mucosa has folds and is formed of stratified squamous epithelium (EP), lamina propria (LP) and muscularis mucosae (mm), the submucosa (SM), the inner circular and the outer longitudinal layers of muscularis externa (ME). b): the mucosa is formed of stratified squamous epithelium (EP) with keratin (K). The epithelium comprises granular cells (↑) and darkly stained basal cells (thick arrow). Lamina propria (LP) and muscularis mucosae (mm) are observed (H&E.: ax200, bx400).

Conversely, the corrosive esophagitis group (II) displayed pronounced histopathological changes, characterized by loss of the mucosal folds. All layers of the rat's esophagus were greatly affected. The mucosa, submucosa, and muscularis externa showed thinning and separation. The squamous epithelium was attenuated and interrupted with a very thin, mostly detached keratin layer. The epithelium also revealed separation between its layers; the deep layer was formed of basal dark cells with pyknosis, and the superficial granular layer comprised dark-stained flat cells. Some basal cells migrating upwards were also detected. The muscularis mucosae was thin and was seen only in a few areas. The submucosa and the muscularis externa were interrupted with congested blood vessels. The inner layer of muscularis externa was separated from the submucosa (Figures 2a & b).

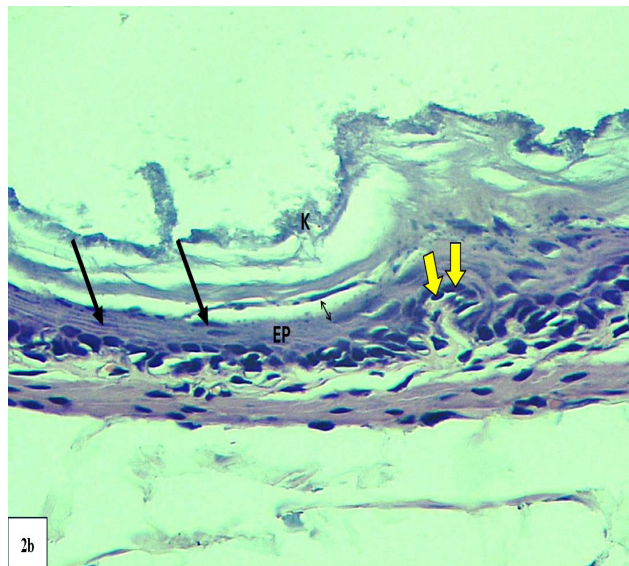
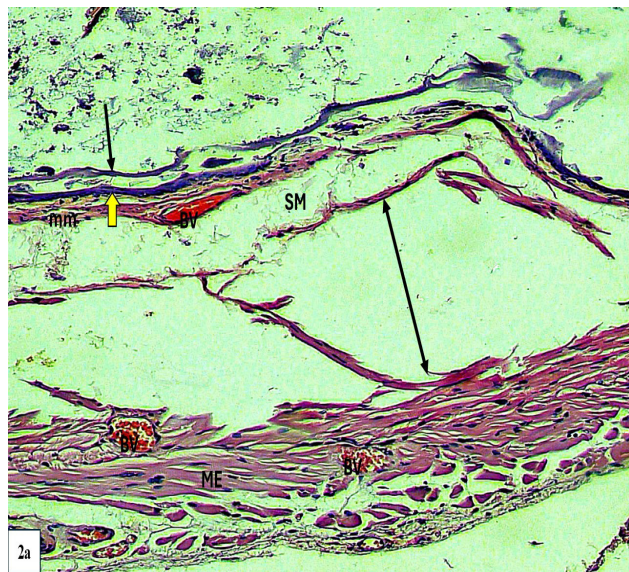


Fig. 2: A photomicrograph of a section of rat's esophagus of the corrosive esophagitis group showing: a) loss of mucosal folds with thinning and separation of all esophageal layers; the epithelium is attenuated and interrupted. Separation between epithelial layers is also detected; the deeper layer (thick arrow) and the superficial layer with thin keratin ($\uparrow$). The muscularis mucosae is thin and seen only in a few areas (mm). Congested blood vessels (BV) are seen in the submucosa (SM) and muscularis externa (ME). The muscularis externa was separated from the submucosa ($\leftrightarrow$). b) Some areas showing thinning of epithelium (EP) appear with keratin layer (K), and some areas show separation ($\leftrightarrow$). Dark-stained flat granular cells ($\uparrow$) and migration of basal cells upward (thick arrow) are detected (H&E.: ax200, bx400).

Examination of H&E-stained sections of the spontaneous healing group (III) exhibited preservation of all layers of the rats' esophagus. The mucosa appeared to have short mucosal folds and thin epithelium with no interruptions. In the epithelial layer, some granular cells were large and filled with granules, while others were flat and dark-stained. There was a continuous keratin layer and congested blood vessels in the submucosa and muscularis externa (Figures 3a & b).

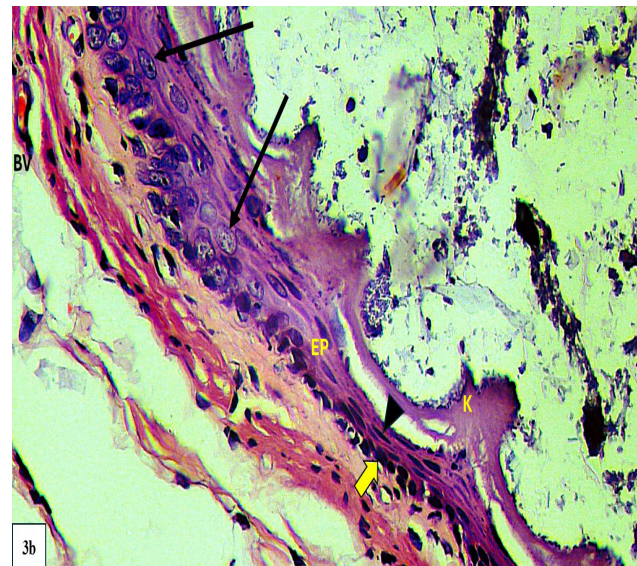
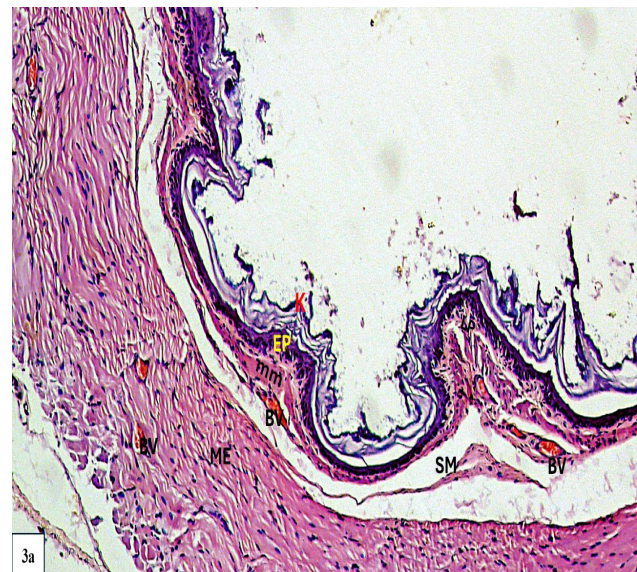


Fig. 3: A photomicrograph of a section of rat's esophagus of the spontaneous healing group showing: a) preservation of all mucosal layers but with thin epithelium (EP), lamina propria (LP), and muscularis mucosae (mm). The mucosa appeared to have short mucosal folds. Continuous keratin layer (K) and congested blood vessels (BV) in submucosa (SM) and muscularis externa (ME) are also seen. b) The epithelium (EP) is formed of granular cells and dark-stained basal cells (thick arrow). Some granular cells appear large and filled with granules ($\uparrow$), others appear flat and dark-stained (arrowhead). Intact keratin layer (K) and congested blood vessels (BV) in submucosa are also observed (H&E.: ax200, bx400).

In the kefir-treated group (IV), H&E-stained sections revealed intact mucosal layers with preserved mucosal folds. The epithelium was demonstrated to have its granular and basal layers, with a continuous keratin layer (Figures 4a & b).

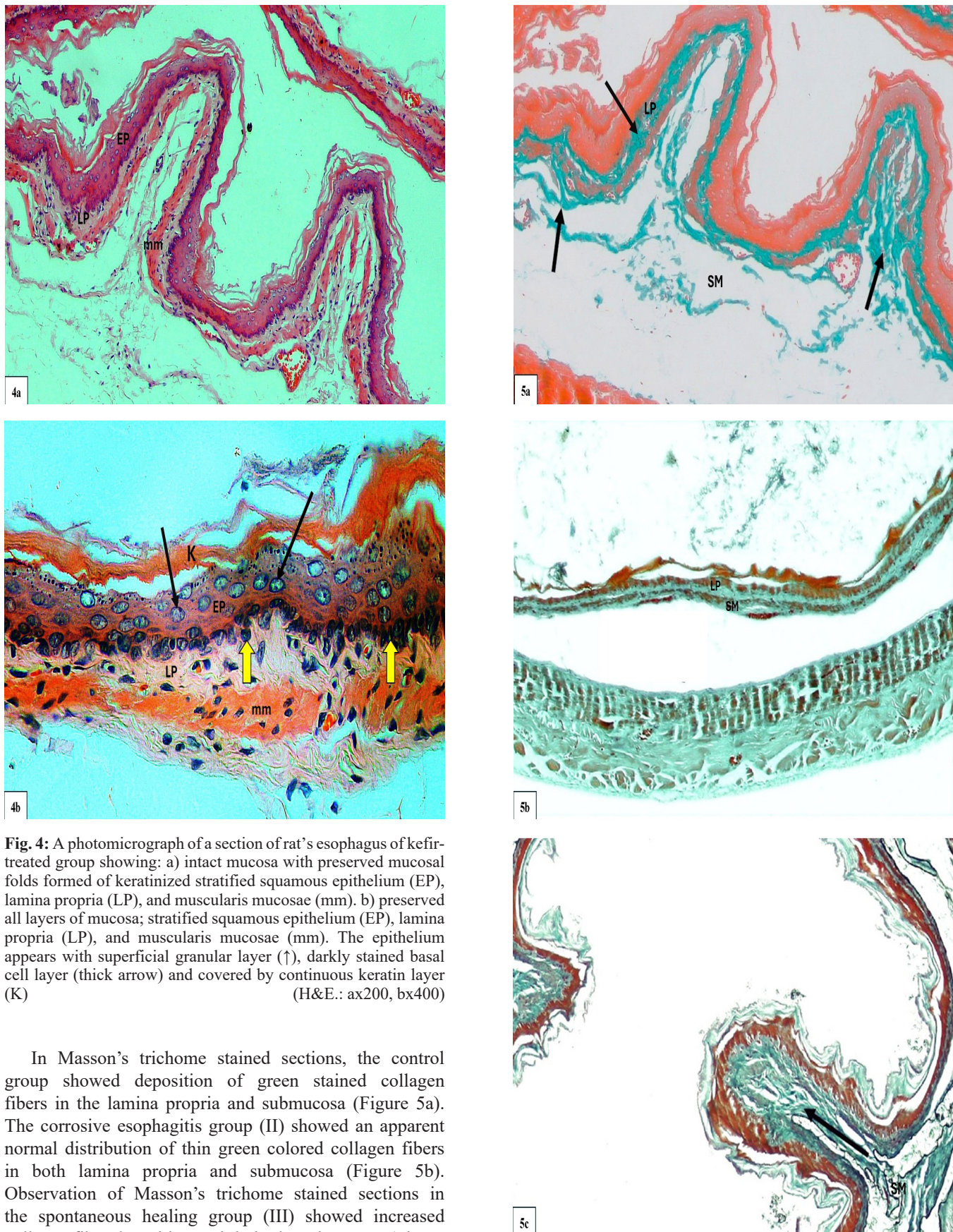


Fig. 4: A photomicrograph of a section of rat's esophagus of kefir-treated group showing: a) intact mucosa with preserved mucosal folds formed of keratinized stratified squamous epithelium (EP), lamina propria (LP), and muscularis mucosae (mm). b) preserved all layers of mucosa; stratified squamous epithelium (EP), lamina propria (LP), and muscularis mucosae (mm). The epithelium appears with superficial granular layer (↑), darkly stained basal cell layer (thick arrow) and covered by continuous keratin layer (K) (H&E.: ax200, bx400)

In Masson's trichome stained sections, the control group showed deposition of green stained collagen fibers in the lamina propria and submucosa (Figure 5a). The corrosive esophagitis group (II) showed an apparent normal distribution of thin green colored collagen fibers in both lamina propria and submucosa (Figure 5b). Observation of Masson's trichome stained sections in the spontaneous healing group (III) showed increased collagen fiber deposition mainly in the submucosa (Figure 5c). Moreover, the kefir treated sections (group IV) also showed an increase of the green colored collagen fiber deposition within the submucosa (Figure 5d).

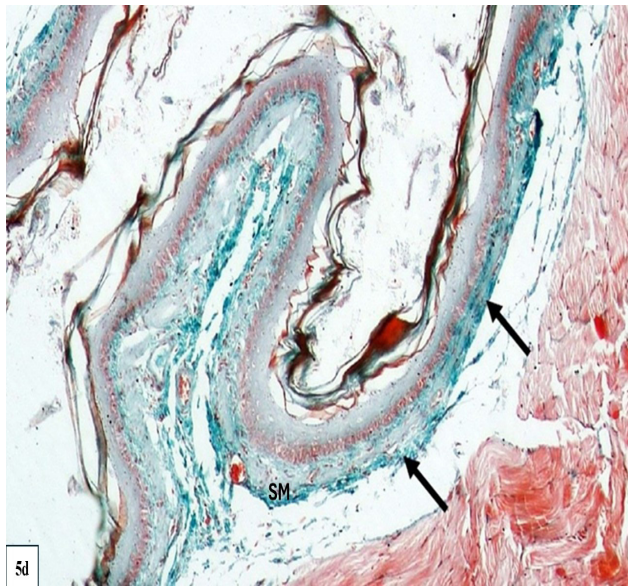


Fig. 5: A photomicrograph of a section of rat's esophagus: a) control group showing deposition of green stained collagen fibers in the lamina propria (LP) and submucosa (SM) (↑). b) corrosive esophagitis group showing apparent normal distribution of thin green colored collagen fibers in both lamina propria (LP) and submucosa (SM) (↑). c) spontaneous healing group showing increased collagen fiber deposition mainly in the submucosa (SM) (↑). d) kefir treated group showing increase of the green-colored collagen fibers within the submucosa (SM) (↑) (Masson's trichome x 200)

Ultrastructural examination:

Scanning electron microscopic examination of the control group showed intact keratinized epithelial cells with longitudinal mucosal folds. The folds were observed to be parallel and regular (Figures 6a & b). Whilst the corrosive esophagitis group (II) showed multiple surface erosions and distortion of the folds, with wide gaps in between. Moreover, an esophageal surface defect was observed (Figures 7a & b).

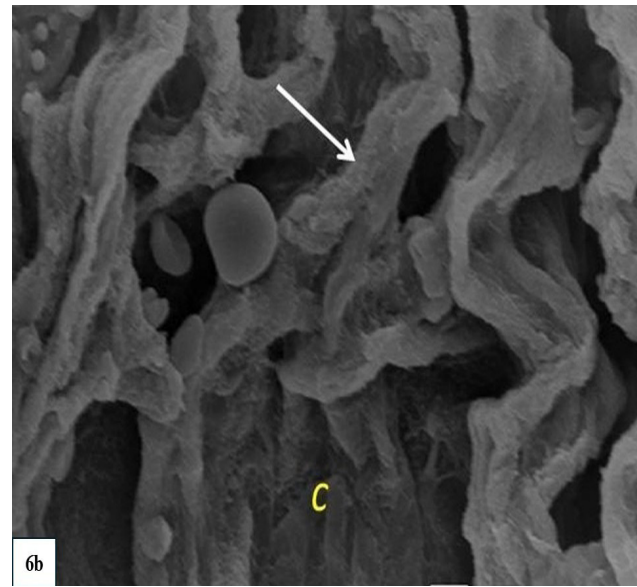
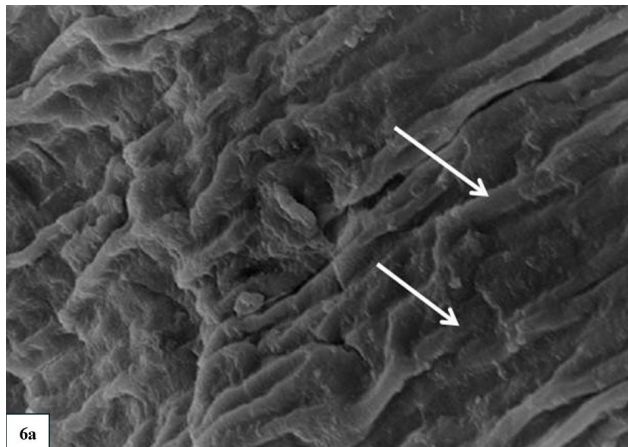


Fig. 6: Scanning electron micrograph of the esophagus from the control group showing: a) intact keratinized epithelial cells with presence of longitudinal mucosal folds (↑). The folds are more or less parallel and regular. b) Surface epithelial cell (C) with defined boundaries. Notice intact keratinized epithelial plicae (↑) (SEM: a x500, bx750)

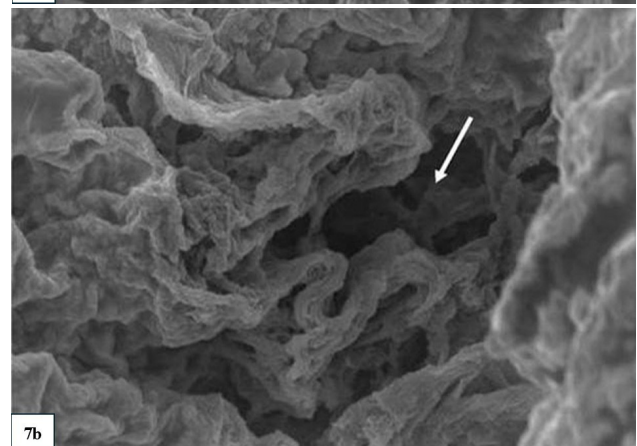
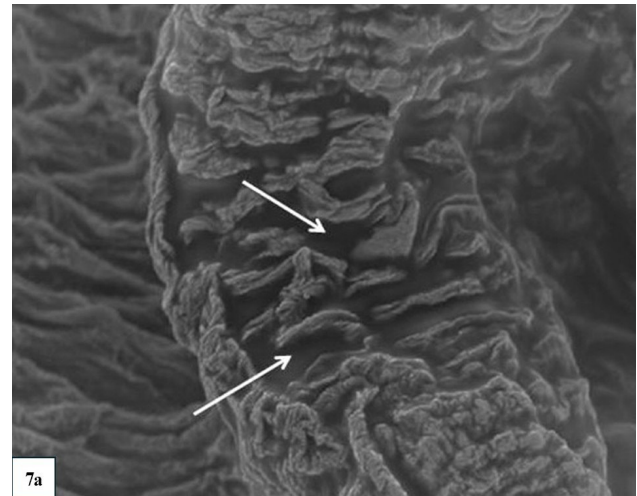


Fig. 7: Scanning electron micrograph of the esophagus from the corrosive esophagitis group showing: a) multiple surface erosions (↑), distorted esophageal folds with wide gaps in between. b) distortion to the folds. An esophageal surface defect is observed (↑)

(SEM: a x200, bx500)

Furthermore, the spontaneous healing group (III) showed an area of granulation tissue. The esophageal surface was smooth with fusion between mucosal folds (Figures 8a & b). In the kefir-treated group (IV), scanning microscopic examination of the esophagus showed reversion of the longitudinal keratinized mucosal folds with preservation of the normal architecture in most areas. The folds seem to be relatively regular and parallel (Figures 9a & b).

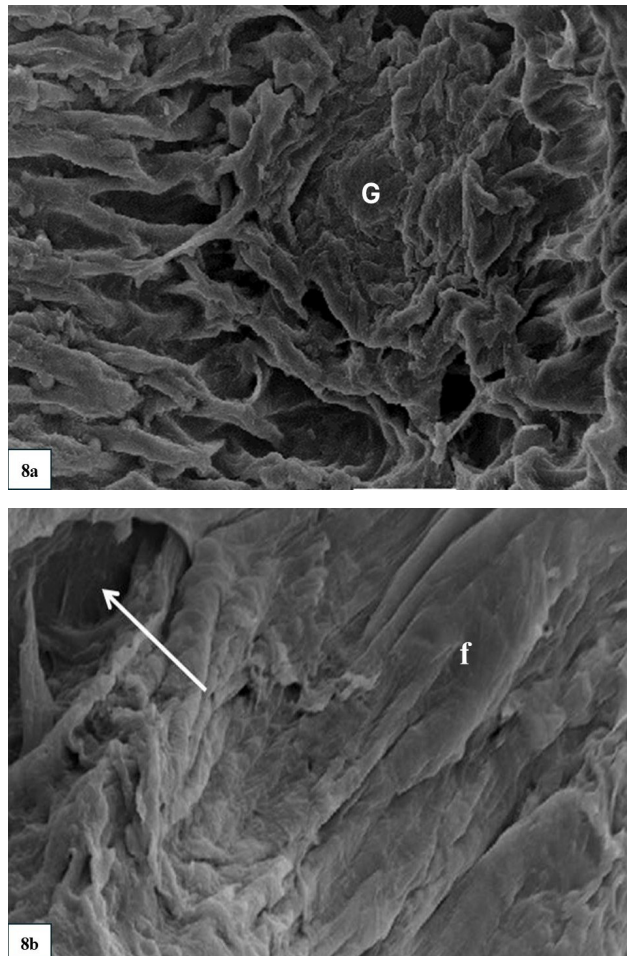


Fig. 8: Scanning electron micrograph of the esophagus of spontaneous healing showing: a) area of granulation tissue (G) b) higher magnification of mostly smooth surface with fusion between folds (f), area of surface loss is observed (↑) (SEM: a x200, bx500)

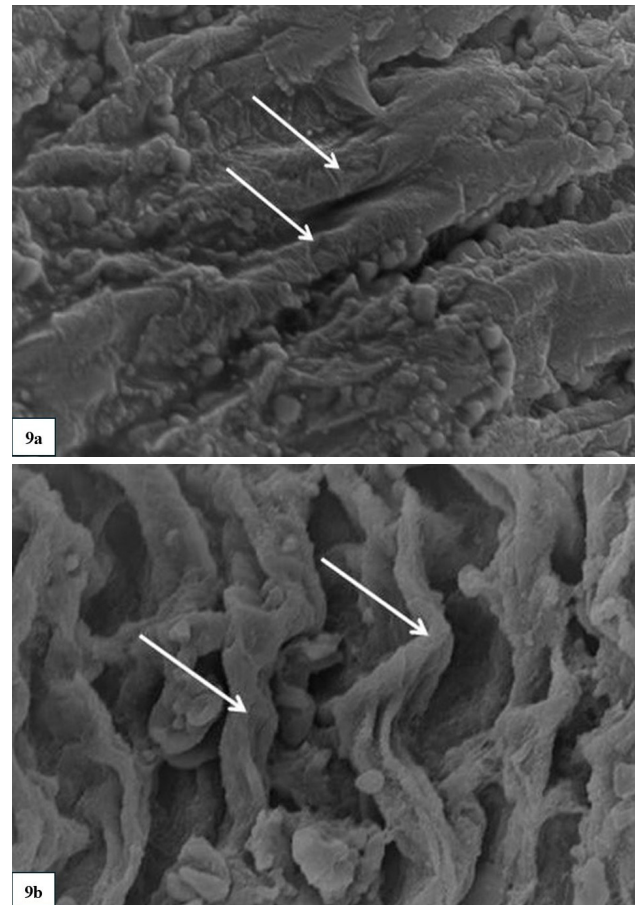


Fig. 9: Scanning electron micrograph of the esophagus of the kefir-treated group showing: a) reversion of the longitudinal keratinized mucosal folds (↑) with preservation of the normal architecture in most areas. (SEMx350) b) relatively parallel and regular keratinized mucosal folds (↑). (SEM: a x350, bx500)

Morphometric results:

Weight changes

Body weight of all rats was recorded at the beginning and the end of the study. No statistically significant difference was observed between the corrosive esophagitis group and the control groups. The spontaneous healing group exhibited a highly significant decrease in body weight compared to both the control and corrosive esophagitis groups ($P < 0.001$). Additionally, the spontaneous healing group demonstrated a statistically significant decrease in weight relative to the kefir-treated group ($P < 0.05$). Rats in the kefir-treated group (Group IV) experienced a statistically significant decrease in weight compared to both the control and corrosive esophagitis groups ($P < 0.05$), as seen in Table 1 and Histogram 1.

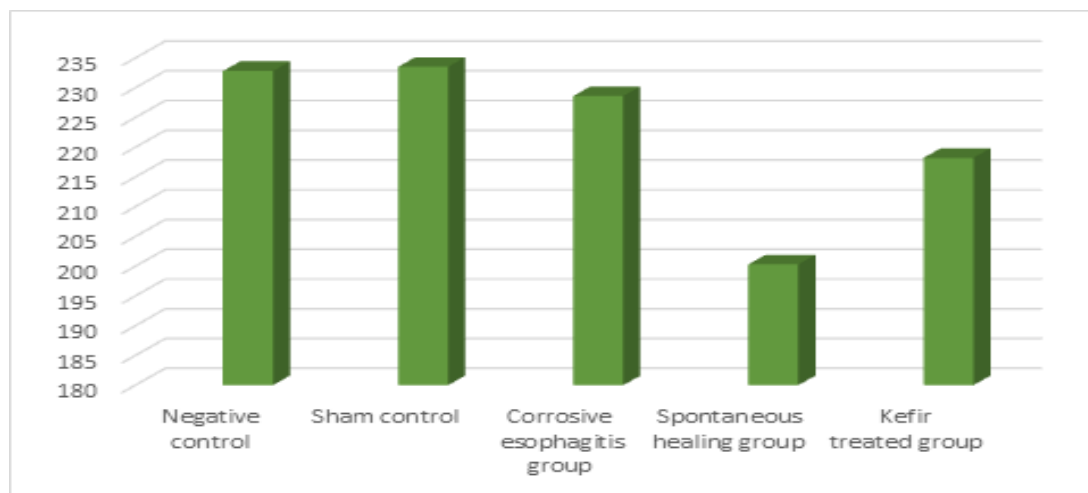
Table 1: Comparison among different experimental groups regarding the mean body weight of the rats in grams.

Control groups		Corrosive esophagitis group (II)	Spontaneous healing group (III)	Kefir-treated group (IV)
Negative control (Ia)	Sham control (Ib)			
232.8 ± 9.2	233.5 ± 7.1	228.6 ± 7.9	200.3 ± 6.1*#	218.2 ± 6.3**

*: highly significant decrease compared to control and corrosive esophagitis groups

#: significant decrease compared to kefir-treated group

**#: significant decrease compared to control and corrosive esophagitis groups



Histogram 1: The mean rats' body weight in different groups.

Collagen fiber deposition

Analysis of the mean surface area percentage of collagen fiber deposition revealed no statistically significant difference between the corrosive esophagitis group and the control group. However, the spontaneous healing group demonstrated a highly significant increase in collagen fiber

deposition compared to both the control and corrosive esophagitis groups ($P < 0.001$). Furthermore, the kefir-treated group exhibited a statistically significant increase in collagen fiber deposition relative to the control and corrosive esophagitis groups ($P < 0.05$). Also, the kefir group showed a significant decrease in collagen fiber deposition compared to the spontaneous healing group ($P < 0.05$) (see Table 2 and Histogram 2).

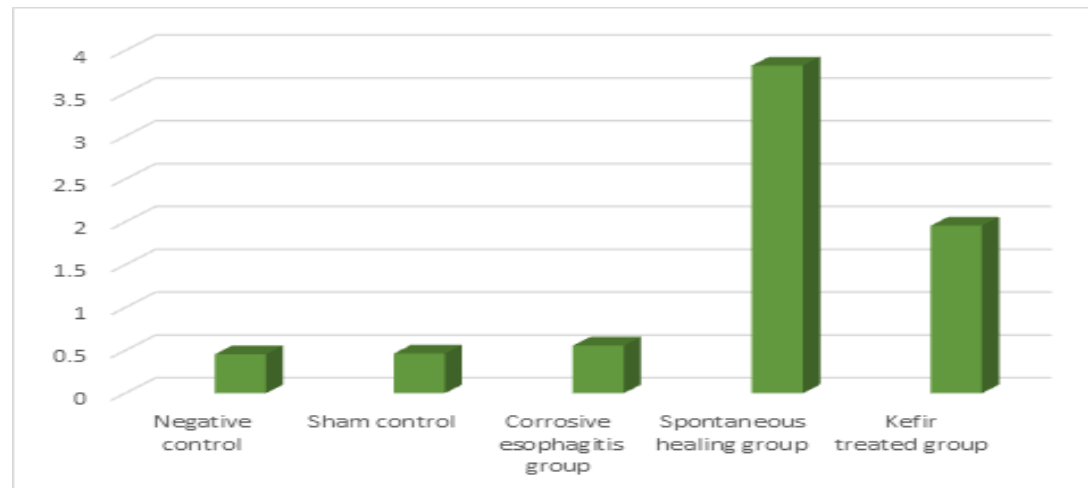
Table 2: Comparison among different experimental groups regarding the mean area% of collagen fiber deposition.

Control groups		Corrosive esophagitis group (II)	Spontaneous healing group (III)	Kefir-treated group (IV)
Negative control (Ia)	Sham Control (Ib)			
0.455 ± 0.24	0.463 ± 0.21	0.556 ± 0.298	3.824 ± 0.583*	1.956 ± 0.592#**

*: Highly significant increase compared to control and corrosive esophagitis groups.

#: significant decrease compared to Spontaneous healing group

**: significant increase compared to control and corrosive esophagitis groups.



Histogram 2: The mean area% of collagen fiber deposition in different groups.

DISCUSSION

Exposure of the gastrointestinal tract to corrosive injuries caused by household products remains one of the serious medical and social issues. The main goal of the treatment of post-corrosive burn is to induce healing and

prevent probable stenosis formation. The current work examined the histopathological characteristics of the esophageal mucosa of adult male albino rats following ingestion of 5% sodium hydroxide and simultaneous treatment with kefir, utilizing light microscopy and scanning electron microscopy.

In the present work, male albino rats were utilized to avoid the female hormonal effect; progesterone hormone is suggested to accelerate the healing process, which may affect our results^[16].

The present study showed weight changes among the groups; rats of the spontaneous healing group showed weight loss more than those of the kefir-treated group. This is most probably attributed to the post-corrosive esophageal stricture resulting from spontaneous healing^[17] and Kefir's ability to lessen the negative consequences of burn injuries, promoting a more favorable environment for recovery^[18].

In the current work, examination of the esophagus after NaOH administration revealed loss of mucosal folds, thinning, and separation of the layers of the esophagus. Spontaneous healing group showed preservation of all layers, with short mucosal folds, thin epithelium, and congested blood vessels in submucosa and muscularis externa. The kefir-treated group exhibited intact mucosal layers and preserved mucosal folds, with continuous keratin.

In the present study, sections stained with H&E of the corrosive esophagitis group revealed that the rat's esophageal layers were all greatly affected. There was thinning and separation of the mucosa, submucosa, and muscularis externa. There was an upward migration of basal cells. These results were in line with *Anwar et al.*, 2017^[19] who demonstrated that administering sodium hydroxide (NaOH) to adult Wistar rats can lead to significant distorted esophageal epithelium and debris in the lumen. Likewise, *Keşim et al.*, 2024^[20] studied the effect of sodium hydroxide on the esophageal mucosa of adult albino rats, reporting signs of epithelial disarray, deformation, and shedding. Furthermore, Basuguy & Gokalp Ozkorkmaz, 2023^[21] observed degenerative alterations in the esophageal epithelial lining following NaOH ingestion. Moreover, *Hara et al.*, 2022^[22] demonstrated that basal cells migrate upwards, identifying them as a distinct stem cell-like population possessing self-renewal capacity and the potential to generate epithelial cells.

In the current work, all layers of the rats' esophagus were preserved in the spontaneous healing group, with continuous thin epithelium and short mucosal folds. This has coincided with the findings of *Keşim et al.*, 2024^[20] who reported that the untreated corrosive esophageal burns resulted in decreased keratin and epithelium, deteriorated connective tissue, and compromised mucosal layer integrity owing to burn damage. Moreover, our study showed congestion of blood vessels. These results were in line with Tarnawski & Ahluwalia, 2021^[23] who stated that ulcer healing is a process of dedifferentiation, proliferation, migration, and re-epithelialization ending in granulation tissue formation. Angiogenesis and vasculogenesis are also involved, resulting in scar formation.

In the present study, the kefir-treated group showed almost normal esophageal mucosa compared to the corrosive esophagitis group. Almost intact covering mucosal epithelium was detected in many areas with a continuous keratin layer. This was consistent with the results of *Nascimento da Silva et al.*, 2023^[24] who reported that administration of kefir fermented milk significantly attenuated the histopathological changes associated with ulcerative colitis. Specifically, kefir supplementation diminished inflammatory responses and contributed to the preservation of cellular architecture. Similarly, *Ellatif et al.*, 2022^[25] identified kefir as possessing potent antimicrobial, anti-inflammatory, and wound-healing properties, suggesting its potential therapeutic efficacy across a spectrum of pathological conditions.

Moreover, *Oryan et al.*, 2019^[26] demonstrated that kefir enhances the phases of wound healing, improving tissue regeneration and closure. This is partly attributed to its lactic and acetic acid content, which modulate inflammation and promote re-epithelialization^[27]. Therefore, the microbiota in kefir synergistically accelerates wound contraction and healing. *Côco et al.*, 2023^[28] observed that probiotic properties of milk kefir, especially those associated with lactobacilli and bifidobacteria, promote the ability to prolong the healing period and preserve the mucosal barrier's integrity.

In the current research, sections stained by Masson's trichrome demonstrated that collagen fiber deposition was highly significantly increased in the spontaneous healing group, mainly in the submucosal layer, compared to the control subgroups. The group that received kefir revealed a significant increase in the collagen fiber deposition compared to the control subgroups. Also, there was a significant increase in collagen fiber deposition, mainly in the submucosa of the spontaneous healing group compared to the kefir-treated group. This was supported by the morphometric results and statistical analysis. *Chirica et al.*, 2017^[29] considered fibrous tissue deposition as a defensive body mechanism. Furthermore, *Yang et al.*, 2024^[30] explained the underlying mechanism of fibrosis that esophageal tissue injury triggers inflammatory and fibrotic pathways, leading to fibrosis, excessive extracellular matrix deposition, and stricture formation.

On the other hand, kefir-treated wounds showed high hydroxyproline content that may improve collagen synthesis, wound contraction, faster wound healing, and increased collagen fiber deposition^[31].

The previous findings were supported by scanning electron microscopy, which revealed distortion of the mucosal folds as well as multiple gaps in between, noticed in the corrosive esophagitis group. Granulation tissue area was observed in the spontaneous healing group. An apparently normal mucosal surface was detected in the kefir-treated group.

CONCLUSION

The current study demonstrated that 5% NaOH exposure results in significant histopathological damage to the esophageal mucosa in albino rats, whereas kefir confers a protective effect on the mucosal architecture, likely mediated by its potent antioxidant and anti-inflammatory mechanisms. Kefir could be a promising new therapeutic approach for promoting healing, as an adjuvant in the treatment of corrosive esophageal injury. However, further research should be conducted to further explore the underlying mechanisms of action.

CONFLICT OF INTERESTS

There is no conflicts of interest.

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الإصابة التآكلية للمرئ الناتجة عن الصودا الكاوية و الدور الوقائي المحتمل للكفير في الجرذان البيضاء البالغة: دراسة نسيجية و مسح مجهري الكتروني

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المقدمة: يعتبر تأثير ابتلاع المواد المسببة للتآكل على المريء قضية صحية مهمة على الرغم من برامج التعليم المكثفة والشاملة لتفاديها حيث تبدأ إصابة الغشاء المخاطي للمرئ في غضون دقائق من تناول مادة كاوية. ولقد ثبت أن مستخلص الكفير يتميز بخصائص مضادة للبكتيريا وتعديل المناعة.

الغرض من العمل: دراسة تأثير الدور الوقائي المحتمل للكفير على إصابة بنية المرئ بعد ابتلاع الصودا الكاوية.

المواد والطرق: تم استخدام خمسة وعشرين من ذكور الجرذان البيضاء في هذه الدراسة. تم تقسيم الجرذان بصورة عشوائية إلى: المجموعة الضابطة، تم تقسيمها إلى جزئين (أ) لم يتلق أي علاج، (ب) تلقى واحد مل من الكفير مرة واحدة يومياً لمدة سبعة أيام، مجموعة التهاب المريء التآكلي (تلقى الجرذان واحد مل من الصودا الكاوية ٥ ٪ عن طريق الفم وتم التضحية بالجرذان بعد يوم واحد)، مجموعة الشفاء العفوي (تلقى الجرذان ١ مل من الصودا الكاوية ٥ ٪ عن طريق الفم وتركزت لمدة سبعة أيام) والمجموعة المعالجة بالكفير (تلقى الجرذان ١ مل من الصودا الكاوية ٥ ٪ عن طريق الفم ، تم تغذيتها بواحد مل من الكفير مرة واحدة يومياً لمدة سبعة أيام). تم استخراج المرئ ومعالجته إستعداداً للفحص المجهرى الضوئى (باستخدام صبغة الهيماتوكسيلين والأيوسين و صبغة الميسون ثلاثي اللون) و المجهر الإلكتروني الماسح. تم إجراء التحليل الإحصائي لبعض القياسات.

النتائج: أظهر فحص المريء بعد إعطاء هيدروكسيد الصوديوم فقداناً في طبقات الغشاء المخاطي، وترققاً، وانفصالاً في طبقات المريء. فيما يتعلق بمجموعة الشفاء التلقائي ، لوحظ الحفاظ على جميع الطبقات، مع طبقات مخاطية قصيرة و ترقق الطبقات المخاطية ، أما المجموعة المعالجة بالكفير، فبدت طبيعياً فيما عدا بعض التغيرات الطفيفة مع استعادة طبقة الكيراتين متصلة.

الخلاصة: مما سبق نستنتج أن الكفير أثبت فعالية في حماية الغشاء المخاطي للمريء نتيجة ابتلاع المواد المسببة للتآكل