OPEN ACCESS
Advances in Animal and Veterinary Sciences

Research Article

Therapeutic Efficacy of Pantoprazole on Surgical Gastric Ulcer Healing in a Rabbit Model

Rafid M. Naeem, Alaa A. Ibrahim, Mohammed M. Jassim*, Mohammed R. Abduljaleel

Department of Surgery and Obstetrics, College of Veterinary Medicine, University of Basrah, Basrah, Iraq.

Abstract | Following the occurrence of a gastric mucosal ulcer, it is important to protect the underlying mucosal layers from damage by gastric acid. The present study aimed to histologically evaluate the effect of a proton pump inhibitor (pantoprazole) on acute gastric ulcers in a rabbit model. Twelve female rabbits, with body weights ranging from 1.5 to 2.0 kg, were used for this study. The animals were divided into two groups: one group treated with antibiotics plus pantoprazole, and a second group treated with antibiotics only (control). Under general anesthesia and complete aseptic conditions, the stomach was exteriorized from the abdominal cavity through a linear laparotomy. A circular ulcer, 2 mm in diameter, was created in the mucous membrane of all groups using thumb forceps and scissors. The stomach wall and then the abdominal wall were closed in the usual manner. Stomach ulcer status was monitored at 3, 7 and 15 days post-surgery. Clinically, the control group showed more regression signs (in movement and appetite) gradually in the late period of the study. Macroscopically, 3 days post-ulceration, the control group showed a black, marked lesion at the ulcer site; however, the treated group showed erythematous ulcers with a smaller diameter and an elevated, edematous border. In the control group on day 7, the ulcer was erythematous and edematous, with edema in the surrounding mucosa. By day 15, there was an elevated, edematous, white-colored lesion with diffuse erythematous areas in the mucosa surrounding the ulcers. However, in the treated group after 7 and 15 days, the ulcer lesion had apparently disappeared, with complete epithelialization of the mucosal surface. Microscopically, the treated group showed greater improvements in tissue regeneration and epithelial reconstruction compared to the control group. Pantoprazole can potentially treat gastric ulcers by successfully providing partial mucosal protection; it reduces gastric acid secretion, which lowers the aggressive factors in the lumen, allowing the body's natural healing processes (epithelial migration, regeneration) to occur more effectively.

Keywords | Pantoprazole, Proton pump inhibitor, Rabbit, Stomach, Ulcer

Received | February 01, 2026; **Accepted** | March 11, 2026; **Published** | April 24, 2026
***Correspondence** | Mohammed M. Jassim, Department of Surgery and Obstetrics, College of Veterinary Medicine, University of Basrah, Basrah- Iraq; **Email:** Mohammed.Majid@uobasrah.edu.iq
Citation | Naeem RM, Ibrahim AA, Jassim MM, Abduljaleel MR (2026). Therapeutic efficacy of pantoprazole on surgical gastric ulcer healing in a rabbit model. Adv. Anim. Vet. Sci., 14(5):895-905.
DOI | https://dx.doi.org/10.17582/journal.aavs/2026/14.5.895.905
ISSN (Online) | 2307-8316

INTRODUCTION

A gastric ulcer is defined as an injury that penetrates the submucosal layer and is linked to a fibrotic healing process, where scar development with fibrous protein production is more common than wound contraction. The resulting scar is frequently resorbed if the severity and duration of the gastrointestinal insult are reduced. Permanent scar development may arise from a substantial fibrotic reaction in long-term damage (Oncel and Basson, 2022). The gastric ulcer is a mucosal defect that penetrates the muscularis mucosa. However, the terms "gastric ulceration" and "gastroduodenal ulceration" actually refer to a clinical condition characterized by anatomically confined

cracks in the surface of the gastrointestinal mucosa, with a likely multifactorial origin that varies from case to case. Because acid pepsin bathes these ulcers, stomach or proximal duodenal ulcers are also known as peptic ulcers (Parrah *et al*., 2013).

The normal mucosa of the stomach is constantly renewing. Normal cytoprotective systems in the stomach shield the mucosa against autodigestion by digestive enzymes and gastric acid. Epithelial migration without proliferation quickly repairs superficial epithelial damage (Liu *et al*., 2023). Many gastrointestinal disorders may be caused by increase acid secretion and have negative effect on gastric mucosa lead to gastric ulcers (Ndidi, 2020), these disorders need the inhibition of stomach acid output. Proton Pump Inhibitors (PPIs) are very effective medications used to reduce stomach acid and treat significant erosive and non-erosive gastrointestinal diseases in animals (Yavuz and Arslan, 2017).

Long-term usage of NSAIDs and corticosteroids can be linked to GI ulcers, which are a dangerous side effect of several clinically significant disease conditions in companion animals. As a result, gastro-protectants are frequently given to companion animals. In spite of this, not much is known about these medications in veterinary patients. To further assess, the effectiveness and proper application of gastro-protectants in animals, more veterinary research is required (Henderson and Webster, 2006).

Glucocorticoids have been linked to a higher chance of gastric hemorrhage and enteritis; the exact mechanism, however, is unknown. Theories include decreased mucus production or mucosal cell renewal rate or changes in gastric mucus composition, possibly from disruption of normal gastric protective mechanisms mediated by prostaglandin (Tseng *et al*., 2015).

The protocols for the treatment of gastric ulcers depend on the underlying cause, severity of the lesions, and the animal's overall health. Medical treatment of patients with gastric ulceration is preferred if the underlying cause can be corrected or controlled, blood loss is not life threatening, and the ulcer does not appear to be in danger of immediate perforation. The goal of medical treatment is to increase mucosal defense systems and reduce stomach acidity. Patients with stomach ulcers have been treated with histamine (H2)-receptor antagonists, proton pump inhibitors, sucralfate, antacids, misoprostol, and bismuth subsalicylate. Surgical exploration and resection are recommended when medicinal therapy is not an option due to the severity of the ulcer. The animal should receive a mix of medications intended to preserve the stomach mucosa following surgical treatment (Demitrack *et al*., 2012; Patel *et al*., 2018; Cornell, 2012).

After PPIs were first used in clinical settings to treat acid-related illnesses, their use increased dramatically and steadily. Today, PPIs are among the most commonly prescribed medications worldwide, with many afflicted patients receiving PPI treatment for several years (Kinoshita *et al*., 2018).

Omeprazole and Pantoprazole are substituted benzimidazoles that covalently bind the hydrogen ion (H+)-potassium ion (K+) ATPase enzyme, blocking its activity and thereby blocking secretion of hydrogen ions into the gastric lumen. Proton pump inhibitors are absorbed in an alkaline environment; thus, their uptake is within the proximal duodenum. Proton pump inhibitors are metabolized primarily by the hepatic cytochrome P-450 system. Only pantoprazole is accessible as an injectable medication, despite the fact that all proton pump inhibitors may be used orally. To guarantee that peak serum concentration and maximal proton pump secretion activity coincide, these medicines should be given one hour prior to a meal. Near-complete inhibition of gastric acid secretion requires 3 to 5 days of therapy. Concerns with regard to bacterial overgrowth and decreased absorption of specific compounds have been raised when proton pump inhibitors are used for more than 30 days. Currently, these concerns have not been substantiated in the veterinary literature (Aguilera-Castro et al., 2016; Cornell, 2012).

Although a number of PPI-related side effects have been documented, their clinical significance is still unclear since the data presented in that research is insufficiently strong the majority are based on retrospective observational studies, and the stated hazard ratios are low. PPIs should only be given to patients who would have a significant therapeutic benefit, and high-quality prospective studies should continue to look at any negative effects (Kinoshita *et al*., 2018).

The study's objective was to investigate histologically how proton pump inhibitors (pantoprazole) affected a surgically induced gastric ulcer in a rabbit model.

MATERIALS AND METHODS

Animals

Twelve female rabbits, weighing 1.5-2.0 kg, were utilized in the present study. The animals were maintained in separate cages. They had free access to food and water during a seven-day acclimation period. Animals were deprived of food, but had free access to water, for five hours before surgery (Ibrahim, *et al*., 2025). After induction of gastric ulcers, rabbits were randomly divided into two groups: group one was ulcerated and treated with antibiotics only (control), and group two was treated with antibiotics plus

the proton pump inhibitor (pantoprazole) post-gastric ulceration (treated group).

Mucosal ulcer induction: Under general anesthesia induced by a mixture of xylazine and ketamine (Abduljalel *et al*., 2025), the stomach was exposed from the abdominal cavity through a midline celiotomy (Figures 1, 2). After performing a gastrotomy, a mucosal ulcer (2 mm) was created by using curved Mayo scissors and thumb forceps to excise the gastric mucosa (Figure 3). The stomach wall was closed with Schmieden and Cushing suturing patterns using 3-0 monofilament synthetic suture (polyglyconate) (Figures 4, 5). The abdominal incision was closed with usual manner (Figure 6). Systemic antibiotic was administered for seven consecutive days post operatively (Yamamoto *et al*., 2017).

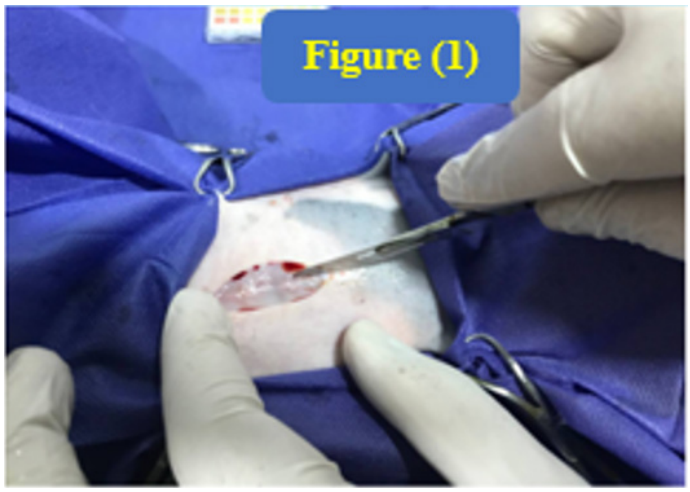

Figure 1: Abdominal midline incision.

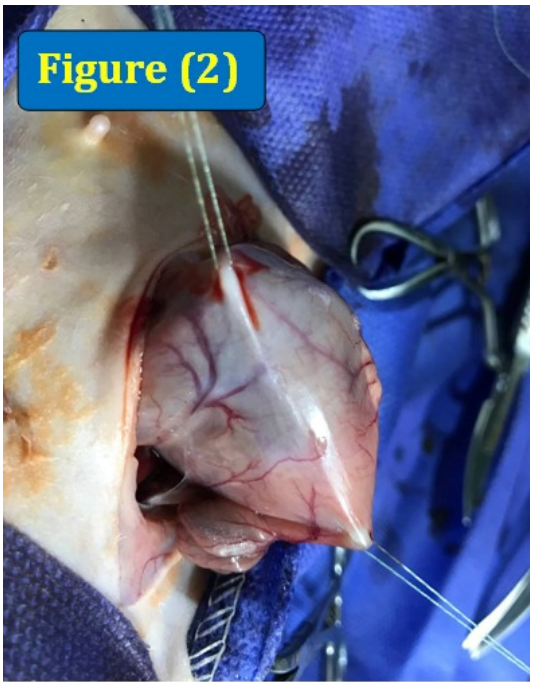

Figure 2: The stomach was exposed through a mid-line celiotomy.

Figure 3: Gastric ulcer was performed after doing gastrotomy.

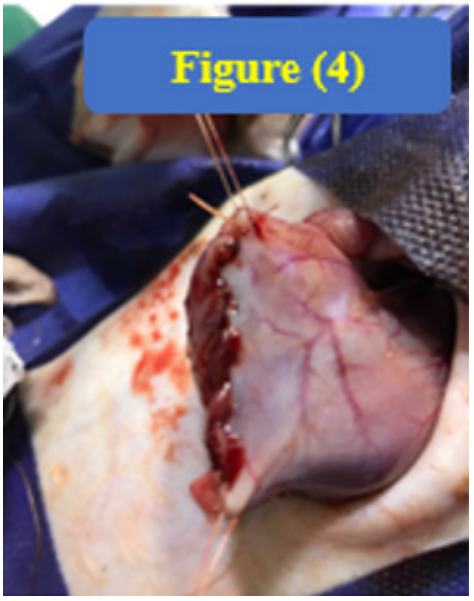

Figure 4: First row of suturing (schmeden) was done by monofilament suture.

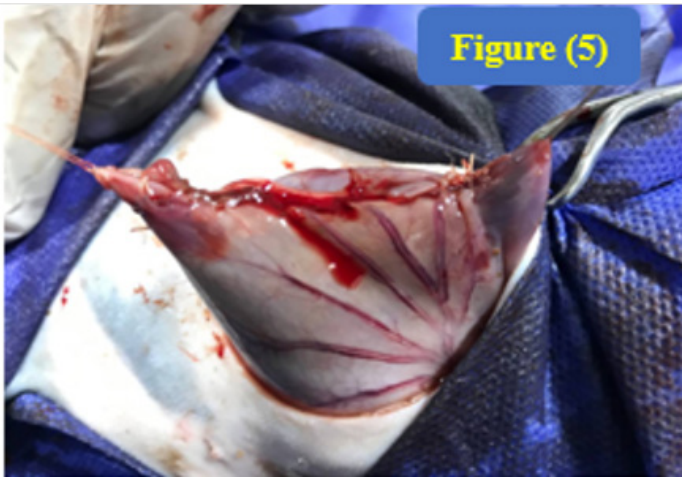

Figure 5: Cushing suturing patterns was performed as a second row.

The pharmaceutical product (proton pump inhibitor) used in the experiment to treat gastric ulcers contained pantoprazole (Strides Pharma, India) and was available as a tablet. Each tablet contained 20 mg, which was dissolved in 10 ml normal saline to obtain a concentration of 2 mg/ml of pantoprazole. The animals in the treated group were orally administered 2 ml (4 mg) of pantoprazole for seven consecutive days (Babitha *et al*., 2009).

Samples of the stomach ulcer were obtained at three different time points after 3, 7, and 15 days when the animals were humanely sacrificed. After being excised and preserved in 10% neutral buffered formalin, these samples were transported to the University of Basrah's Veterinary College laboratory for histopathological analysis. The histological sections were stained using hematoxylin and eosin stain (Mohsin *et al*., 2025). In addition, gastric specimens were evaluated using a scoring system that included grades to assess gastric mucosal injury and neutrophil infiltration (Table 1).

Table 1: Histolopathological scoring system (Liu *et al*., 2016).

State of pathology	Score No.	
Mucosal injury	0	Intact
	1	The epithelial lamina desquamation
	2	The superficial lamina propria desquamation or 1/3 gastric glands reduction
	3	The middle lamina propria desquamation or 2/3 gastric glands reduction
	4	lower lamina propria desquamation or >2/3 gastric glands reduction, even submucosal exposure
Infiltrations of Leuko-cytes	0	Absent
	1	2 to10/HPF
	2	11 to 20/HPF
	3	21to 30/HPF
	4	>31/HPF
hemorrhage	0	Absent
	1	<10% of the total area/LPF
	2	11% to 20% of the total area/LPF
	3	21% to 30% of the total area/LPF
	4	> 30%

HPF: high power field; LPF: low power field.

RESULTS

Clinical examination

On the first day postoperatively, the experimental animals suffered from a loss of appetite and reduced movement. Gradually, they returned to normal on the third and fourth days. However, after a few days, the health status of the animals in the control group began to decline in terms of appetite and movement.

Statistical analysis

The pathological scoring results were presented as mean values ± standard errors. Data were analyzed using the Independent Samples t-test and One-Way ANOVA with multiple comparison tests, employing a statistical software program (SPSS for Windows, version 22, USA). Significant differences were considered acceptable at ($P \leq 0.05$) (Liang *et al*. 2019; Choudhary, 2018).

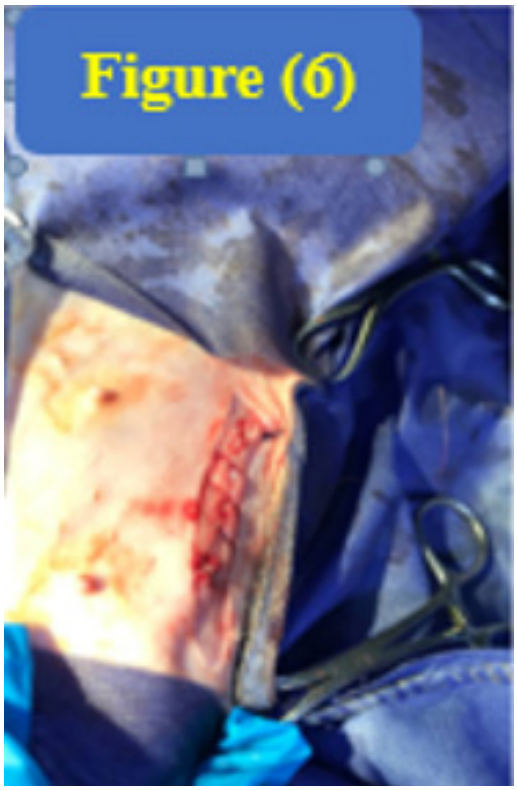

Figure 6: The abdominal wall was closed with a simple continuous pattern.

Gross pathological examination

Control group

Three days post-surgery, there was black, marked lesion at the site of the ulcer. On the 7th day, the ulcer was erythematous and edematous, with edema in the surrounding mucosa. On the 15th day, there was an elevated, edematous, white-colored lesion with a diffuse erythematous area in the mucosa around the ulcers (Figure 7, 8 and 9).

Treated group

Three days post-ulceration, the ulcer was erythematous, smaller in diameter, and had an elevated, edematous border. After 7 and 15 days post-surgery, the ulcer had disappeared, with complete epithelialization of the mucosal surface (Figures 10, 11).

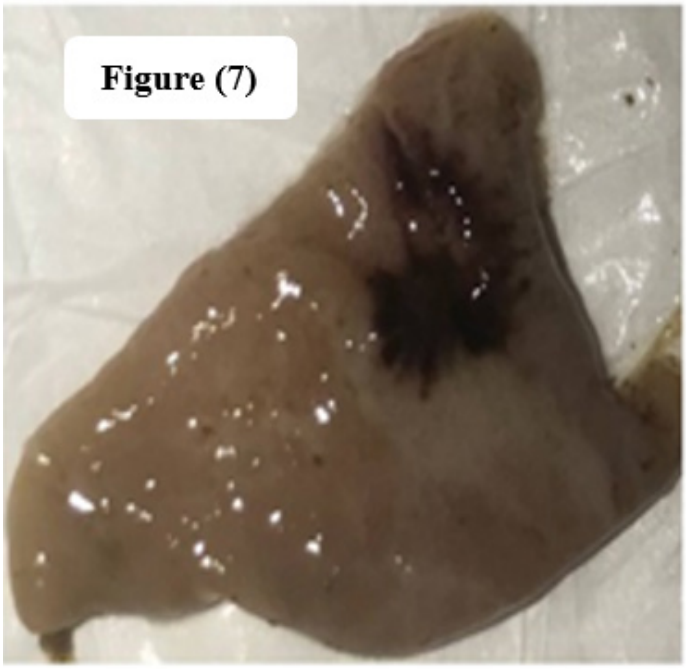

Figure 7: A black in color, demarcated lesion at the site of the ulcer.

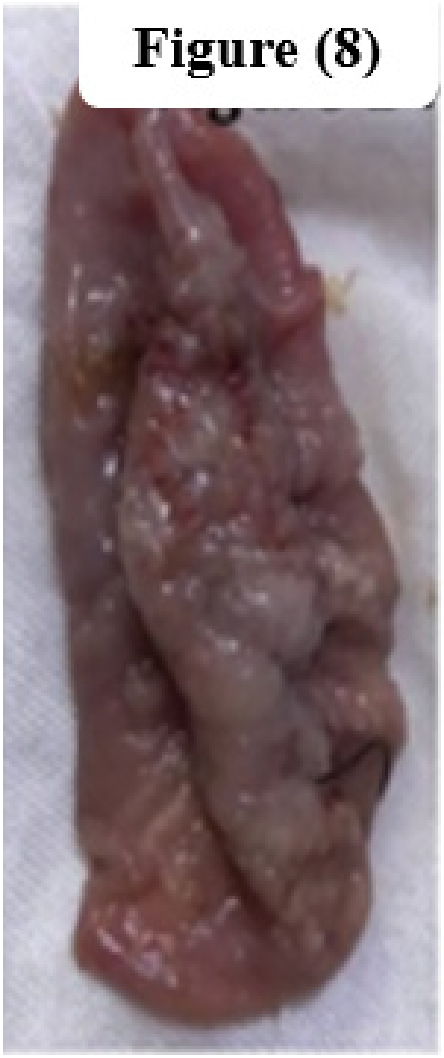

Figure 8: The site of ulcer was erythematous and edematous with edema in the surrounding mucosa.

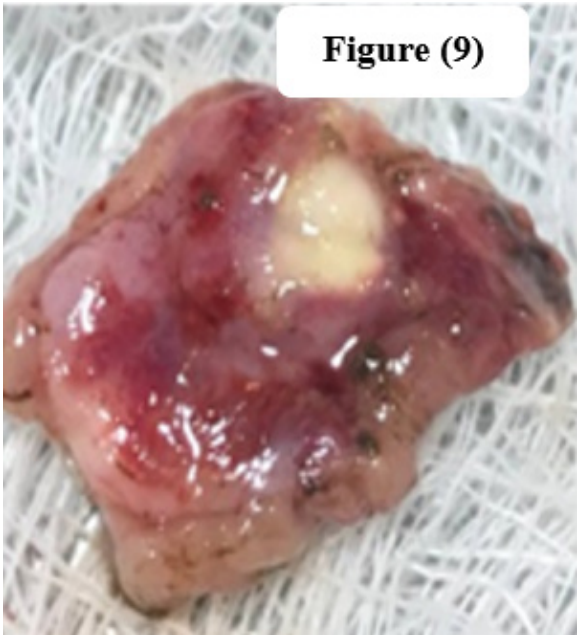

Figure 9: Elevated edematous white color lesion with diffuse erythematous lesions in mucosa.

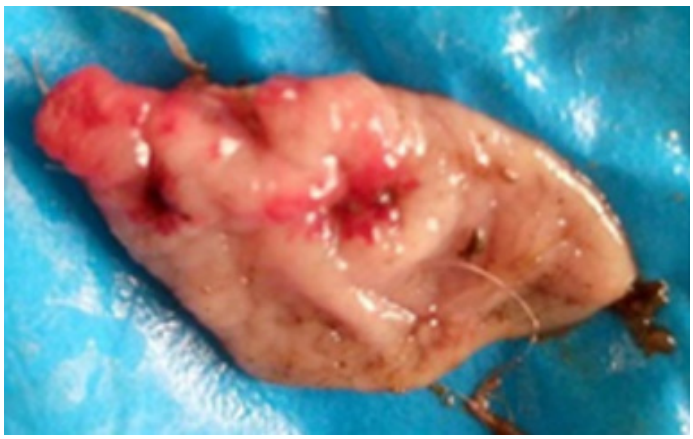

Figure 10: Erythematous ulcer and with elevated edematous border.

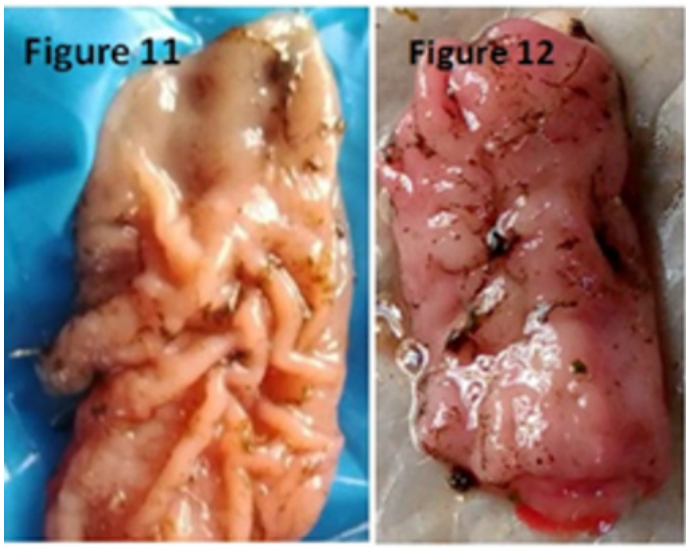

Figure 11 and 12: The ulcers disappeared with complete epithelialization of the mucosal surface.

Histopathological examination

Three days post-surgery, the control group showed mild necrosis and mild infiltration of inflammatory cells (Figure 13A, 17). Furthermore, most of the gastric mucosa, including the gastric foveolae, gastric pits, gastric glands, parietal cells, and chief cells, appeared normal and intact. However, the treated group from the same period revealed a severe peptic ulcer characterized by severe necrosis and damage to the gastric mucosa, with signs of congestion and severe infiltration of inflammatory cells (Figures 13B, 16, 17, 18).

Seven days post-surgery, gastric ulcers in the control group showed the following changes: Severe infiltration of inflammatory cells and accumulation of granulation tissue. The infiltrated inflammatory cells include plasma cells and lymphocytes as well as accumulation of fibroblasts. The damage to the majority of the stomach mucosa, including gastric foveolae, gastric pits, gastric glands, parietal cells and chief cells, was noted in the control group 7 days following the onset of stomach ulcers. In addition, significant buildup of fibrous tissue and a massive infiltration of inflammatory cells were observed (Figures 14A, 16, 17). In contrast, the changes in the pantoprazole-treated group became less severe. The severe changes that appeared 3 days after ulcer induction began to fade. Instead of severe necrosis of the gastric mucosa, a focal area of necrosis was observed, with a regenerative area separated from the necrotic area by granulation tissue. Some infiltration of inflammatory cells was noted, and some cystic shaped gastric glands were evident (Figures 14B, 16, 17).

At the 15-day time point after gastric ulcer induction, histological evaluations of the control group showed severe necrosis, thick granulation tissue, and infiltration of inflammatory cells inside the gastric glands and in the sub-epithelial layer. Dilated, cystically-shaped gastric glands were observed, with infiltrated inflammatory cells, mostly lymphocytes, present inside them (Figures 15A, 16, 17). While, the histopathological changes of the treated group after 15 days after ulcer induction showed that the subepithelial mucosa displays cystic dilation of few of the gastric glands, presence of focal area of inflammation, and presence of regenerated gastric glands (Figures 15B, 16, 17).

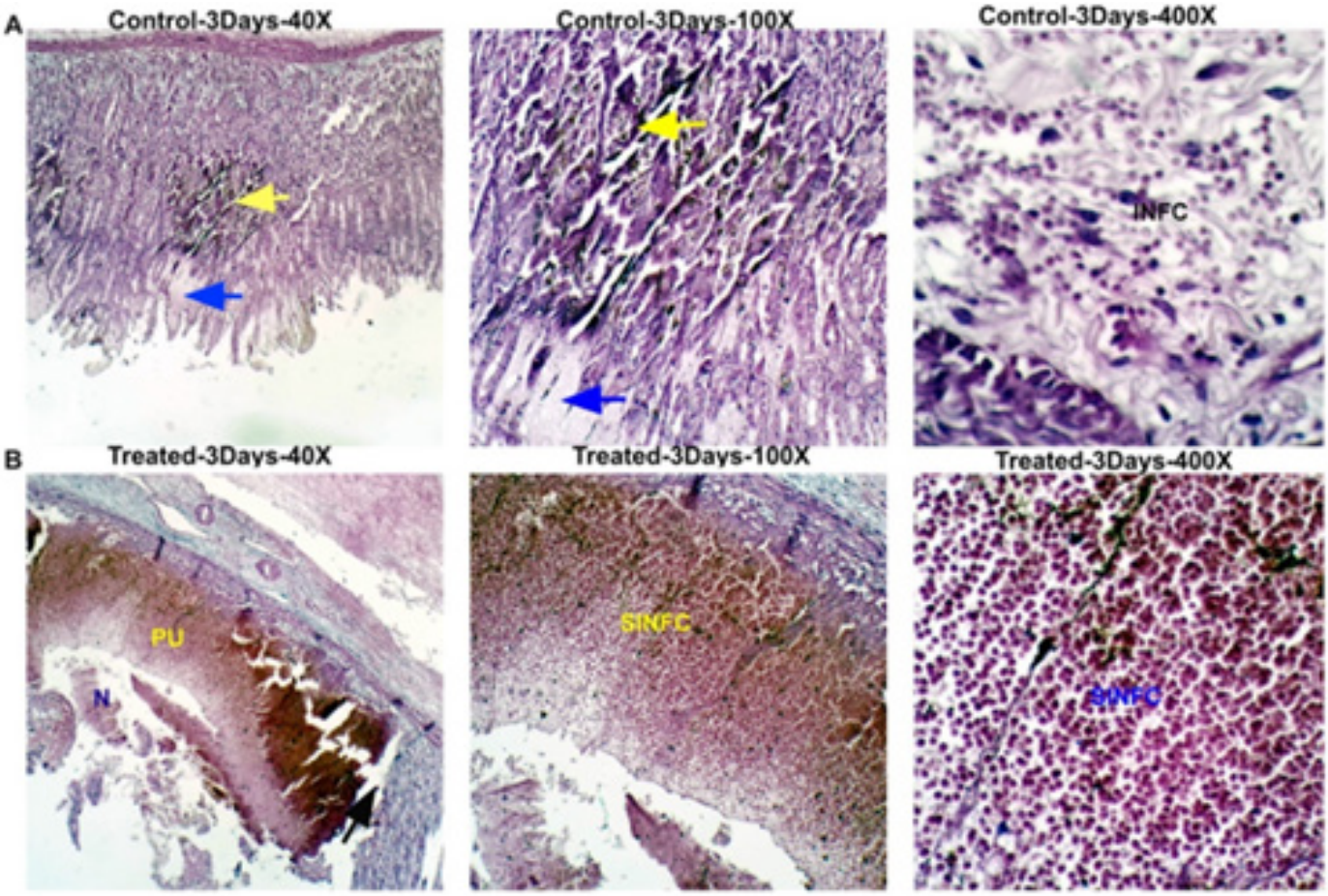

Figure 13: 3days post surgery, section of gastric ulcer. (A) Control group: showed mild necrosis (blue arrows) and infiltration of inflammatory cells (yellow arrows, INFC). (B) treated group: revealed a severe peptic ulcer (PU) characterized by severe necrosis (N), severe infiltration of the inflammatory cells (INFC). H&E, 40X; 100X, 400X.

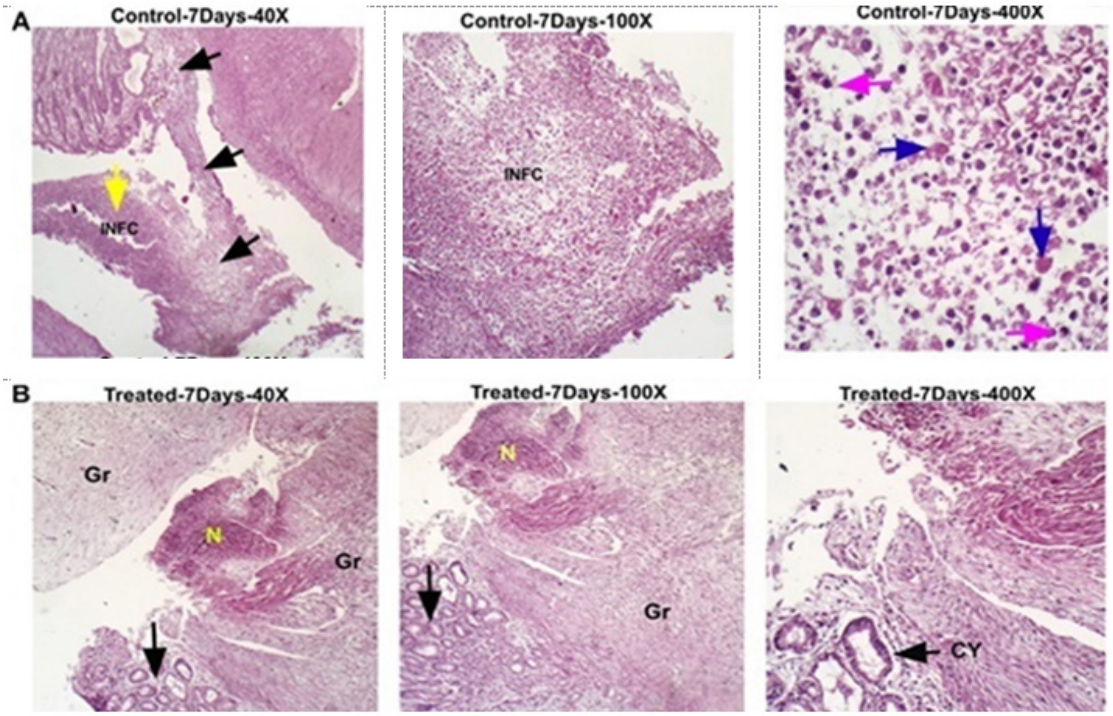

Figure 14: 7 days post surgery, section of gastric ulcer. (A) Control group: showed severe infiltration of inflammatory cells (INFC), accumulation of the granulation tissue (black arrows). The infiltrated inflammatory cells include plasma cells (blue arrows) and lymphocytes (pink arrows) as well as accumulation of fibroblasts (red arrows). (B) Treated group It shows the presence of two regions, the first region is a focal area of necrosis (N), and the second region is a group of regenerative gastric glands (black arrows) separated by granulation tissue (Gr). Some of the gastric glands are cystic in shape contain inflammatory cells inside (CY). H&E, 40X, 100X, 400X.

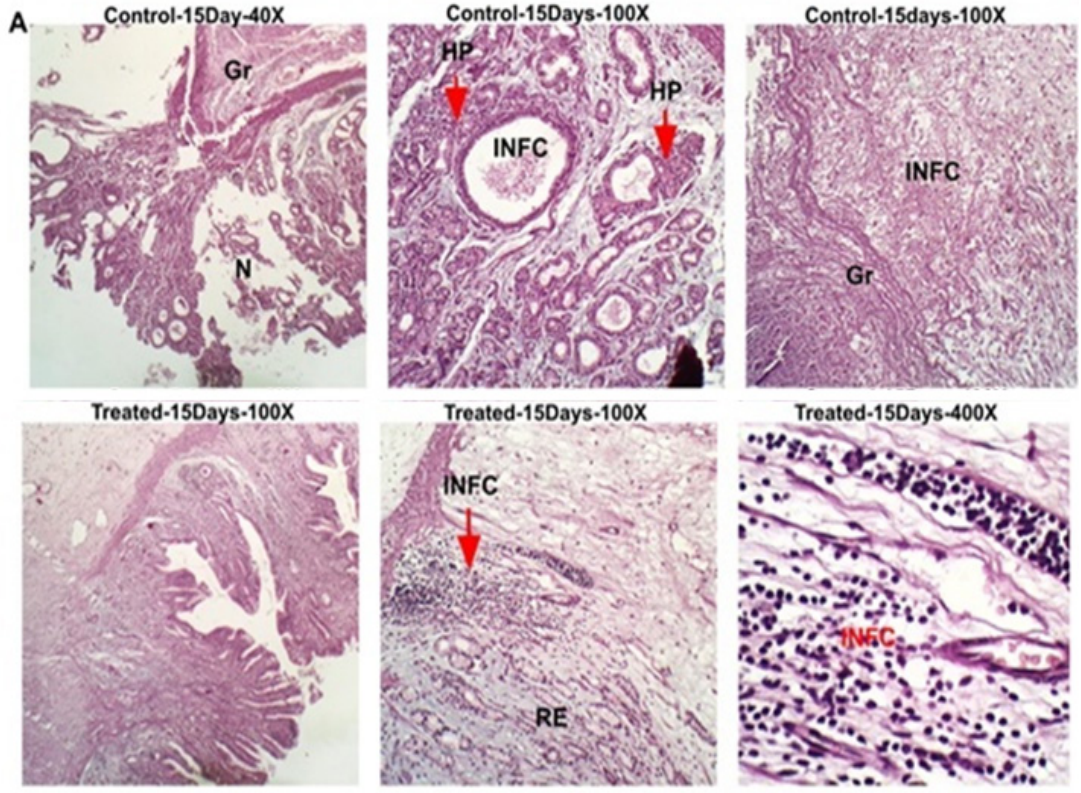

Figure 15: 15 days post inducing gastric ulcer. (A) Control group showed sever necrosis (N), thick granulation tissue (Gr), infiltration of inflammatory cells inside the gastric glands and in the subepithelial layer (INFC). (B) treated group 15 days after ulcer induction showed that the subepithelial mucosa displays cystic dilation of few of the gastric glands (black arrows), presence of focal area of inflammation (red arrow, INF), and presence of regenerated gastric glands. H& E, 40X, 100X, 400X.

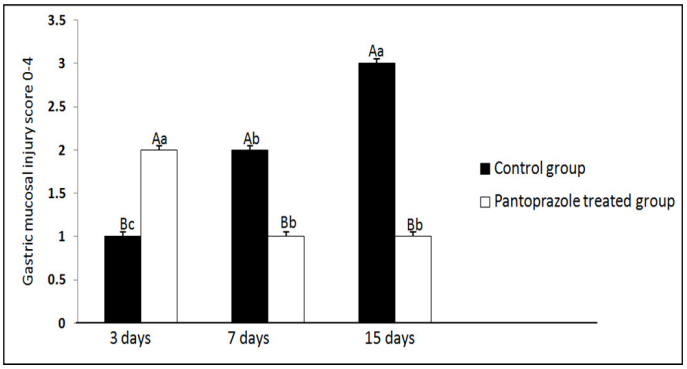

Figure 16: Pathological state of gastric mucosal ulcer showed mucosal injury scores for control group and pantoprazole treated group after 3, 7, 15 days of ulcers induction. The values were showed as means and standard errors. AB Different letters among groups indicates significant differences (P<0.05). abc Different letters within group indicates significant differences (P<0.05).

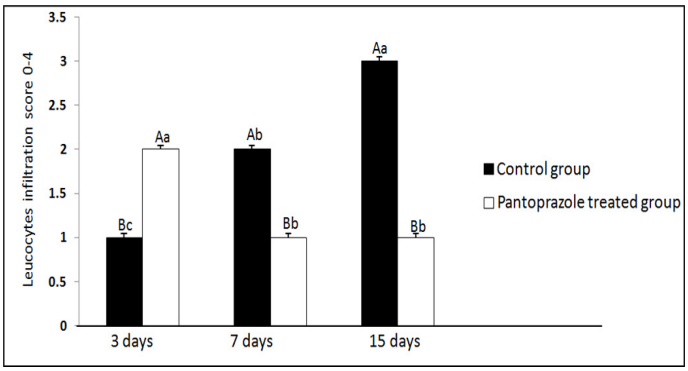

Figure 17: Pathological state of gastric mucosal ulcer showed leukocytes infiltration scores for control group and pantoprazole treated group after 3, 7, 15 days of ulcers induction. The values were showed as means and standard errors. AB Different letters among groups indicates significant differences (P<0.05). abc Different letters within group indicates significant differences (P<0.05).

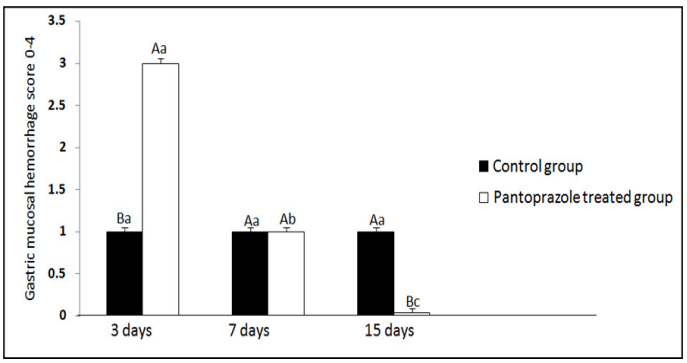

Figure 18: Pathological state of gastric mucosal ulcer showed hemorrhage scores for control group and pantoprazole treated group after 3, 7, 15 days of ulcers induction. The values were showed as means and standard errors. AB Different letters among groups indicates significant differences (P<0.05). abc Different letters within group indicates significant differences (P<0.05).

DISCUSSION

The very acidic luminal contents are shielded from the stomach epithelium by the gastric mucosal barrier. The stomach mucosa's cellular layers are sealed by tight junctions, which prevent luminal contents from leaking into or around these cells. The surface of the epithelium is covered in a thick, bicarbonate-rich mucous coating. The strong blood flow to this region swiftly eliminates the tiny quantity of stomach acid that diffuses into epithelial cells. Drugs that reduce acid production can preserve injured GI mucosa, but many illnesses and medications have the potential to disturb the delicate balance between the harsh luminal contents and the GI protective barrier (Demitrack *et al.*, 2012).

The aim of the study was to evaluate the effect of pantoprazole (PPI) after surgically inducing a gastric mucosal ulcer, assessed clinically, grossly, and histopathologically.

On the first postoperative day, the experimental animals suffered from loss of appetite and less movement. Gradually, they returned to normal on the third and fourth days. However, the health status of the animals of control group gradually began to decline. These results demonstrate the protective effect of the proton pump inhibitor on the ulcerated gastric mucosa in treated animals and the progression of the unprotected gastric ulcer in the control group. Proton Pump Inhibitors are highly effective medications used to treat significant erosive and non-erosive gastrointestinal problems in animals as well as to control stomach acid. It relieves clinical signs and heals mucosal damage, and it offers better intragastric pH management over a 24-hour period (McQuaid, 2004).

After 3 days post-surgery in control group, macroscopically, there was a black in color, demarcated lesion at the site of the ulcer while in treated group, ulcers were erythematous and less diameter with elevated edematous border. These results demonstrate the gastroprotective action of the PPI in improving gastric wound healing and the migration of mucosal epithelial cells, which helps decrease the ulcer diameter. Omeprazole and pantoprazole usage led to an elevated stomach pH (>3) after just two days of dosing in a recent canine study. Despite having a brief 1.5-hour serum half-life, PPIs prolong acid inhibition because they permanently deactivate the proton pump, and it takes 36 to 96 hours for new H+/K+ ATPase pump molecules to be synthesized (Foushee, 2000).

At the 7th day, control group showed the ulcer was erythematous and edematous with edema in the surrounding mucosa while at the 15thday, there was elevated edematous white in color lesion with diffuse erythematous

lesions in the mucosa around the ulcers. However, after 7, and15 days, the ulcer was disappeared with complete epithelialization of the mucosal surface in treated group. This result showed that inflammatory, erosive, and eventually ulcerative lesions may occur when the stomach mucosal barrier is disrupted. Gastroprotectants are used to heal ulcers and prevent mucosal damage because they enhance mucosal protective mechanisms (Henderson and Webster, 2006).

PPIs are helpful in reducing GI ulcer hemorrhage. One important element in the management of bleeding ulcers is elevated intra-gastric pH. By disrupting the coagulation system, fibrinogen polymerization, and platelet aggregation, both HCl and pepsin impair coagulation (Gisbert *et al*., 2001).

After 3 days post-surgery, the control group showed mild necrosis and mild infiltration of inflammatory cells. Furthermore, most of the gastric mucosa including gastric foveolae, gastric pits, gastric glands, parietal cells and chief cells are normal and intact. While the treated group revealed severe peptic ulcer and characterized by severe necrosis and damage of gastric mucosa with signs of congestion, severe infiltration of the inflammatory cells, ulceration. The tissue repair phases of the wound-healing process can be inhibited by chronic inflammation (a prolonged inflammatory stage); however, acute inflammation appears early in an improved healing phase (Gao *et al*., 2024). It hypothesized that the control group's ulcer was immediately covered by a protective clot/crust (the "black" lesion seen grossly), while the PPI group, with higher pH, might have had a more "clean" but exposed ulcer bed that appears more reactive histologically.

These results disagree with those of Peng *et al.* (2017), who obtained 75 gastric antral mucosa samples from adult patients who had undergone upper gastrointestinal endoscopy and reported gastrointestinal symptoms. Prior to the stomach biopsy, each patient's history was documented, including smoking, alcohol usage, and the length of time they had been on PPIs. The samples were assessed using histology. They discovered that PPIs reduced neutrophil infiltration of the gastric mucosa, an effect that may be connected to mucosal atrophy.

Seven days after gastric ulcer induction, the control group exhibited the following changes: severe infiltration of inflammatory cells and accumulation of granulation tissue. The infiltrated inflammatory cells include plasma cells and lymphocytes as well as accumulation of fibroblasts. The damage to the majority of the stomach mucosa, including gastric foveolae, gastric pits, gastric glands, parietal cells, and chief cells was noted. In addition, significant buildup of fibrous tissue and a massive infiltration of inflammatory cells were observed. In contrast, the changes in the treatment group became less severe. The severe changes that appeared 3 days after ulcer induction began to fade. Instead of severe necrosis of the gastric mucosa, a focal area of necrosis was observed, with a regenerative area separated from the necrotic area by granulation tissue. Some infiltration of inflammatory cells was noted, and some cystic shaped gastric glands were evident.

Proton-pump inhibitors permanently attach to hydrogen-potassium ATPase molecules, which results in a dose-dependent and long-lasting suppression of stomach acid output. The inhibition lasts long after the medication is removed from the plasma because acid secretion can only start again after fresh hydrogen-potassium ATPase molecules are produced. Proton-pump inhibitors speed up ulcer healing because of this complete and long-lasting inhibition (Simadibrata *et al*., 2022).

At the 15-day time point after gastric ulcer induction, the control group showed severe necrosis, thick granulation tissue, and infiltration of inflammatory cells inside the gastric glands and in the subepithelial layer, dilated cystic shaped gastric glands were observed with presence of infiltrated inflammatory cells inside mostly lymphocytes. While, the histopathological changes of the treated group showed that the subepithelial mucosa displays cystic dilation of few of the gastric glands, presence of focal area of inflammation, and presence of regenerated gastric glands, these evident come in agree with (Olivarez *et al*., 2023). They discovered that PPI usage leads to better intragastric pH management, making it more successful in treating and preventing ulcers.

CONCLUSION

Pantoprazole, a proton pump inhibitor, is a very effective medication used to reduce stomach acid and treat significant gastrointestinal problems in animals. This study suggests that pantoprazole can potentially treat gastric ulcers by successfully providing partial mucosal protection, thereby enhancing epithelial migration and mucosal regeneration.

ACKNOWLEDGEMENT

We want to thank head of department of surgery and obstetric, Dean of Veterinary Medicine and Basrah university's president for their support in college laboratory.

NOVELTY STATEMENT

This study provides a novel evaluation of Pantoprazole's therapeutic impact on surgically induced gastric ulcer in a rabbit model, moving beyond traditional chemical-

induced ulcer studies. It demonstrates that Pantoprazole actively reduces the gastric mucosal healing time at the surgical site of ulcer induction by suppression of gastric acid secretion and providing the suitable environment for healing. Therefore, it collaborates with gastric ulcers healing. By utilizing a rabbit model, which more closely mirrors human gastric physiology, this research offers original insights into the prophylactic role of PPIs in post-operative gastric recovery.

AUTHOR'S CONTRIBUTION

All authors equally contributed.

Ethical approval

The Animal Care and Use Committee/College of Veterinary Medicine, University of Basrah, Iraq, followed ethical guidelines and all relevant national and international legislation when designing and conducting this study. (Approval number: 119/37/2026).

Generative AI and AI assisted technology statement

The authors declare that no generative AI and AI assisted technology was used in the creation of this manuscript.

Conflict of interest

The authors have declared no conflict of interest

REFERENCES

Abduljaleel MR, Abdulrazaq AW, Jassim MM, Abbas MF, Alfaris AA, Naeem RM, Ibrahim AA, Saleh WMM, Alrashid IMH, Alrafas HR (2025). Understanding the synergistic impact of atropine with xylazine and ketamine on recovery time, heart rate and respiratory rate in male rabbits. Assiut. Vet. Med. J., 71(187): 237-246. https://doi.org/10.21608/avmj.2025.341413.1505

Aguilera-Castro L, Martín-de-Argila-dePrados C, Albillos-Martínez A (2016). Practical considerations in the management of proton-pump inhibitors. Rev Esp Enferm Dig.,108(3):145-153.DOI:10.17235/reed.2015.3812/2015

Babitha S, Ramesh B, Singh J (2009). A study on the drug-drug interaction of pantoprazole with glipizide in rabbits. Pharmacology online, 2: 667-672. https://pharmacologyonline.silae.it/files/archives/2009/vol2/068.Babita.pdf

Choudhary R (2018). Application of "independent t-test" by using SPSS for conducting physical education researches. Hypothesis, 20(92): 99

Cornell K (2012). Stomach, CHAP. 91. Tobias, K. M. Veterinary Surgery Small Animal. Volum 1. Saunders, an imprint of Elsevier Inc.

Demitrack ES, Aihara E, Kenny S, Varro A, Montrose MH (2012). Inhibitors of acid secretion can benefit gastric wound repair independent of luminal pH effects on the site of damage. Gut, 61(6): 804-811. https://doi.org/10.1136/gutjnl-2011-300420

Foushee LL (2000). Omeprazole. Compend. Contin. Educ. Pract. Vet., 22: 746–749.

Gao M, Guo H, Dong X, Wang Z, Yang Z, Shang Q, Wang Q (2024). Regulation of inflammation during wound healing: The function of mesenchymal stem cells and strategies for therapeutic enhancement. Front. Pharmacol., 15: 1345779. https://doi.org/10.3389/fphar.2024.1345779

Gisbert JP, Gonzalez L, Calvet X, Roque M, Gabriel R, Pajares J (2001). Proton pump inhibitors versus H2-antagonists: A meta-analysis of their efficacy in treating bleeding peptic ulcer. Aliment. Pharmacol. Therapeut., 15(7): 917-926. https://doi.org/10.1046/j.1365-2036.2001.01012.x

Henderson AK, Webster CR (2006). The use of gastroprotectants in treating gastric ulceration in dogs. IN-DEPTH LOOK, 358-370. https://vetfolio-vetstreet.s3.amazonaws.com/mmah/3f/f6e30633754ecbb578feafd56ce2b5/filePV_28_05_358.pdf

Ibrahim A A, Naeem RM, Abduljaleel MR, Jassim MM, Abdulrrazaq AW, Khaleefah IA, Hashim AM (2025). Effect of platelet-rich fibrin (PRF) from different sources on avulsion wound healing in a rabbit model. J. Anim. Health Prod., 13(4): 1244-1251. https://doi.org/10.17582/journal.jahp/2025/13.4.1244.1251

Kinoshita Y, Ishimura N, Ishihara S (2018). Advantages and disadvantages of long-term proton pump inhibitor use. J. Neurogastroenterol. Motil., 24(2): 182-196. https://doi.org/10.5056/jnm18001

Liang G, Fu W, Wang K (2019). Analysis of t-test misuses and SPSS operations in medical research papers. Burns Trauma, 7 (31): 1-5. https://doi.org/10.1186/s41038-019-0170-3

Liu J, Wang F, Luo H, Liu A, Li K, Li C, Jiang Y (2016). Protective effect of butyrate against ethanol-induced gastric ulcers in mice by promoting the anti-inflammatory, anti-oxidant and mucosal defense mechanisms. Int. Immunopharmacol., 30: 179-187. https://doi.org/10.1016/j.intimp.2015.11.018

Liu M, Liu Q, Zou Q, Li J, Chu Z, Xiang J, Chen W, Miao Z, Bin Wang B (2023). The composition and roles of gastric stem cells in epithelial homeostasis, regeneration, and tumorigenesis. Cell. Oncol., 46(4): 867-883. https://doi.org/10.1007/s13402-023-00802-z

McQuaid KR (2004). Drugs used in the treatment of gastrointestinal disease, in Katzung BG (ed): Basic and Clinical Pharmacology, ed 9. New York, McGrawHill, pp. 1034–1044.

Mohsin TA, Abduljaleel MR, Radhi AJ, Abbas MF, Alrashid IMH, Khudhair ZW (2025). Histopathological effects of streptomycin treatment on macrophages in lymph nodes, spleen, liver and kidneys of rats. Adv. Anim. Vet. Sci., 13(6): 1337-1345. https://doi.org/10.17582/journal.aavs/2025/13.6.1337.1345

Ndidi K (2020). Gastric ulcers; causes, symptoms, treatment, and prevention. J. Wetenskap Health, 1(1): 1-4. https://doi.org/10.48173/jwh.v1i1.8

Olivarez JD, Mulon PY, Ebner LS, Cremerius H, Cantrell C, Rahn R, Soto-Gonzalez W, Bergman J, Cox S, Mochel JP, Kreuder AJ, Smith JS (2023). Pharmacokinetic and pharmacodynamic properties of pantoprazole in calves. Front. Vet. Sci., 9: 110146. 13 pages. https://doi.org/10.3389/fvets.2022.1101461

Oncel S, Basson MD (2022). Gut homeostasis, injury, and healing: New therapeutic targets. World J. Gastroenterol., 28(17): 1725. https://doi.org/10.3748/wjg.v28.i17.1725

Parrah JD, Moulvi BA, Gazi Makhdoomi MA, Athar Dar HS, Mir AQ (2013). Gastric ulceration in dog: A review. Vet. World, pp. 449-454. https://doi.org/10.5455/vetworld.2013.449-454

Patel PK, Patel SK, Dixit SK Rathore RS (2018). Gastritis and peptic ulcer diseases in dogs: A review. Int. J. Curr. Microbial. App. Sci., 7(3): 2475-2501. https://doi.org/10.20546/ijcmas.2018.703.288

Peng Y, Huang L, Ho H, Chang C, Lee S, Cheng C, Wen M, Yeh H, Ho S (2017). The effect of proton pump inhibitors on the gastric mucosal microenvironment. Adv. Clin. Exp. Med., 26(1): 39–43. https://doi.org/10.17219/acem/33116

Simadibrata DM, Syam AF, Lee YY (2022). A comparison of efficacy and safety of potassiumcompetitive acid blocker and proton pump inhibitor in gastric acid related diseases: A systematic review and metaanalysis. J. Gastroenterol. Hepatol., 37(12): 2217-2228. https://doi.org/10.1111/jgh.16017

Tseng CL, Chen YT, Huang CJ, Luo JC, Peng YL, Huang DF, Hou MC, Lin HC, Lee FY (2015). Short-term use of glucocorticoids and risk of peptic ulcer bleeding: A nationwide population-based case-crossover study. Aliment Pharmacol. Ther., 42: 599–606. https://doi.org/10.1111/apt.13298

Yamamoto T, Satoi S, Yanagimoto H, Yamaki S, Hirooka S, Michiura T, Inoue K, Matsui Y (2017). Optimal duration of prophylactic antibiotics administration in pancreaticoduodenectomy. Surg. Gastroenterol., 22(4): 334-341. https://doi.org/10.21614/sgo-22-4-334

Yavuz G, Arslan HH (2017). Proton pump inhibitors in veterinary medicine. Am. J. Anim. Vet. Sci., 12(3): 132.138. https://doi.org/10.3844/ajavsp.2017.132.138