Original Research Article

Diagnosis and Therapeutic Management of Chronic Gastritis in Dogs Using Triple Therapy

Abstract

This study was conducted for a period of six months to investigate the diagnosis and therapeutic management of chronic gastritis in dogs using triple therapy. Chronic gastritis, characterized by persistent vomiting and gastric mucosal inflammation, possess a significant challenge in veterinary practice. Chronic gastritis was diagnosed on the basis of history, clinical signs, haemato-biochemical parameters, ultrasonography and endoscopy. Endoscopy was performed in 30 dogs out of which 14 dogs were found positive for chronic gastritis.The parameters like age, sex and breed of each dog having chronic gastritis were recorded to study the distribution of condition. The age-wise distribution of chronic gastritis was higher in 1-3 years of age. The breed wise highest distribution of chronic gastritis was recorded in the Retriever breed of dogs. The gender-wise distribution was observed higher in males. For therapeutic study, a total 12 confirmed cases of chronic gastritis were selected and randomly divided into 2 groups as G1 and G2, each group comprised of six dogs. However, six apparently healthy dogs were included as a healthy control group (G3). Group G1 received the Amoxycillin with sulbactam, Metronidazole and Pantoprazole while group G2 received Ofloxacin, Ornidazole and Esomeprazole. Endoscopy was done on day 0 and day 10 to record changes in mucosa of oesophagus and stomach. The results showed a significant improvement in both groups, with Group G1 achieving an 83.33% recovery rate compared to 66.66% in Group G2. G1 demonstrated greater reductions in vomiting frequency and endoscopic lesions, alongside better normalization of haemato-biochemical parameters.

Keywords: Chronic gastritis, Endoscopy, Triple therapy, Dogs

Introduction

The term “gastritis” was first introduced by Georg Ernst Stahl to describe the inflammation of inner lining of the stomach. Gastritis is specifically characterized as the inflammation of gastric mucosa [1]. Gastritis occurs as a result of disruption of the gastric mucosal barrier leading to increased permeability, mucosal erosions and in some cases ulcerations. The clinical manifestations in canine gastritis include vomiting, hematemesis, melena, retching, belching, hypersalivation, abdominal distention, abdominal pain and loss of body weight [2]. Gastritis is a relatively common condition and can manifest in either acute or chronic forms. Acute gastritis in canines is characterized by sudden onset of vomiting (a clinical

Commented [AKHTER1]: Type of study?

Commented [AKHTER2]: Should not begin with this sentence

Commented [AKHTER3]: In this study, chronic gastritis was…

Commented [AKHTER4]: How you can tell it was chronic based on endoscopy?

Commented [AKHTER5]: Make your observations simple

Commented [AKHTER6]: G1 treated with?, demonstrated…

Commented [AKHTER7]: Reframe the abstract in a better and concise way

hallmark) and infiltration of polymorphonuclear (PMN) cells in the mucosa of the antrum and body, resulting in gastric inflammation and mucosal injury [3]. On the other hand, intermittent vomiting lasting more than one to two weeks is clinically defined as chronic gastritis [4].

The primary causes of chronic gastritis in dogs commonly include ingestion of foreign body, dietary indiscretion, renal dysfunction, hepato-biliary diseases, inflammatory bowel disease, toxin ingestion, occult parasitic infections and *Helicobacter* spp. infection [4][5]. Chronic gastritis is categorized based on the predominant cellular infiltrates, including eosinophilic, lymphoplasmacytic, granulomatous or lymphoid follicular types. It is also classified based on architectural abnormalities such as atrophy, hypertrophy, fibrosis, edema, ulceration or metaplasia, as well as by severity, ranging from mild to moderate or severe [6] [7]. Persistent vomiting is the cardinal clinical sign of chronic gastritis [8]. Aside from the most common manifestation of persistent vomiting, other signs such as weight loss, loss of appetite and hematemesis also observed [9]. Chronic gastritis can be diagnosed on the basis of clinical signs, laboratory findings and imaging techniques such as radiography, ultrasonography and endoscopy. An accurate diagnosis of gastritis is possible through gastroscopy [10][11].

The primary treatment strategies for acid-related disorders include fluid therapy, proton pump inhibitors (PPIs), H2 blockers, cytoprotective agents, antiemetics and antioxidants therapy (ascorbic acid, N-acetyl cysteine, vitamin E [12]. Triple therapy is a highly effective approach for bacterial (Helicobacter) infections, particularly in cases of chronic gastritis, due to the bacteria's rapid development of drug resistance. This regimen typically comprises two antibiotics, such as amoxicillin, clarithromycin, or metronidazole, alongside an acid-reducing agent like omeprazole (a proton pump inhibitor) or an H2 receptor antagonist [13]. The antibiotics act synergistically to eliminate the bacteria, while the acid-reducing agent lowers gastric acid secretion, creating an environment less favourable for bacterial survival and enhancing the efficacy of antibiotics. This combination not only improves treatment success rates but also promotes mucosal healing and minimizes the risk of complications such as ulcers and gastric cancer.

2. Materials and methods

The proposed study was carried out for six months *i.e.* from May 2024 to October 2024 at Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Nanaji Deshmukh Veterinary Science University (N.D.V.S.U), Jabalpur, Madhya Pradesh (M.P.). For this study, dogs presented to the Veterinary Clinical Complex, College of Veterinary Science & Animal Husbandry, NDVSU, Jabalpur (M.P.), with history of different clinical signs, *i.e.*, inappetence, vomiting, hematemesis, weight loss, retching, etc., were thoroughly examined for the presence of chronic gastritis.The complete history of the patient, including age, gender, breed, duration of illness and related history was recorded. The diagnosis of chronic gastritis was made based on history, clinical signs, hematobiochemical parameters, ultrasonography and endoscopy.

Commented [AKHTER8]: Use simple word

Commented [AKHTER9]: Apart from persistant vomiting..

Commented [AKHTER10]: ?
Reframe

Commented [AKHTER11]: What acid reducing agents?
Seems meaningless

Commented [AKHTER12]: What is ulcer, cancer, gastrc proliferation, hypertrophy hyperplasia?
Use terms precisely

Commented [AKHTER13]: delete

Commented [AKHTER14]: delete

Commented [AKHTER15]: different?

Commented [AKHTER16]: Replace this abb

Commented [AKHTER17]: Doesn't make sense. Revise it again

Commented [AKHTER18]: Which related history?

Commented [AKHTER19]: Wheather only chronic cases you have encounter throughout the study?

Figure 01: Clinical signs (Vomiting) in chronic gastritis affected dogs

Commented [AKHTER20]: Describe the figure legend in precise way

2.1 Clinical examination

Each dog was clinically examined for following parameters on day 0 (pre-treatment) and day 10 (post-treatment): Temperature (°F), Pulse rate (beats/minute) and Respiratory rate (breaths/minute). Rectal temperature was recorded using clinical thermometer. Pulse rate was recorded by palpating the femoral artery for 1 minute.Similarly, the respiration rate was recorded by observing the thoracic or chest movements over the same time interval.

Commented [AKHTER21]: These all things need not to be discussed. It's known

2.2 Sample collection

A specimen of approximately 3 ml of blood was aseptically drawn from each dog's saphenous or cephalic vein. 1 ml of blood was extracted into a vial containing EDTA and submitted for standard haematology testing, while the remaining 2 ml was collected into clot activator vials. The serum was extracted following centrifugation (3000 rpm for 5 minutes) and stored at -20°C for biochemical analysis. Haematological parameters (Haemoglobin, PCV, TLC and DLC) were estimated with IDEXX ProCyte Dx ™ automatic haematology analyser on day 0 (pre-treatment) and day 10 (post-treatment) following standard protocols. Serum biochemical parameters (ALT, AST, ALP, Creatinine and BUN) were estimated using CHEM-5 plus semiautomatic analyser with a readymade kit on day 0 (pre-treatment) and day 10 (post-treatment), following standard protocols.

Commented [AKHTER22]: Make it simple. like Blood from cephalic or sephenous vein was collected in vacutainer….

Commented [AKHTER23]: Make it concise

2.3 Diagnostic imaging

Abdominal Ultrasonography

Abdominal ultrasonography was performed by using Philips HD7 XE Ultrasound machine with 3-12 MHz linear array transducer in all the suspected cases of chronic gastritis on day 0 and day 10 to record the changes in gastric mucosal wall thickening. All the animals were thoroughly shaved and cleaned properly. The site caudal to the 10th rib up to the level of pubis

Commented [AKHTER24]: From day 0 to 10, you have recorded the changes in gastric mucosa? And what were the significant changes observed during study? As You have provided only two USG imgs, what about other cases and changes observed wrt groups you mentioned?

and on either side of midline up to the level of transverse processes of the vertebrae was prepared aseptically.

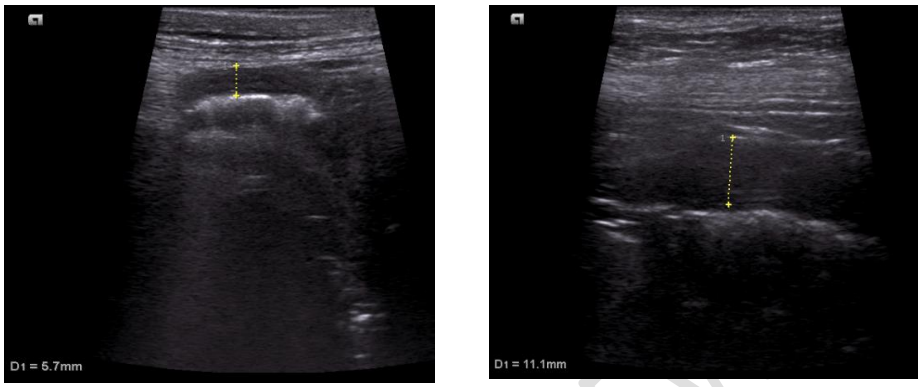

Figure 02: Ultrasonographic image showing gastric wall thickness measuring (a) 5.7 mm, (b) 11.1 mm indicative of (chronic gastritis)

Commented [AKHTER25]: Which one is A and B in figure. Add the imgs with significant changes you observed while performing USG.

Endoscopy

A gastroscopic examination was conducted on day 0 and day 10, using a Karl Storz endoscope (Germany), with a diameter of 10.1 mm and a working length of 3000 mm.To prepare the dogs for the procedure, dogs were fasted by withholding food for 12-24 hours and water for 4-6 hours before the endoscopic procedure to prevent suction channel blockage and ensure better mucosal visualization. Sedation was induced with Atropine Sulphate (0.02-0.04 mg/kg IM), Xylazine Hydrochloride (1-1.5 mg/kg IM) and Diazepam (1-2 mg/kg IV), followed by general anaesthesia using Ketamine Hydrochloride (5-10 mg/kg IM), with maintenance also using Ketamine. During the procedure dogs were positioned in left lateral or dorsal recumbency with the head and neck extended for oesophagoscopy and gastroduodenoscopy, ensuring better visualization of the stomach curvature during gastroscopy.

Commented [AKHTER26]: Have you used the range of drug doses or particular dose?

2.4 Therapeutic regimen

A total of 12 dogs with chronic gastritis were selected for therapeutic study and randomly divided into two treatment groups, namely G1 and G2. Each group comprised six dogs. Apart from these six apparently healthy dogs were taken as healthy control group *i.e.* group G3 (Table 01).

Commented [AKHTER27]: Designated as

Table 01: Therapeutic regimen for chronic gastritis in dogs

Group	Number of animals	Drugs	Dose rate	Route	Duration

G1	06	Amoxycillin+sulbactam Metronidazole Pantoprazole	@ 10mg/kg @ 20mg/kg @ 1mg/kg	IV IV IV	BID for 5 days BID for 5 days OD for 5 days
G2	06	Ofloxacin Ornidazole Esomeprazole	@ 5mg/kg @ 20mg/kg @ 1mg/kg	IV IV IV	BID for 5 days BID for 5 days OD for 5 days
G3	06	Apparently healthy control			

Therapy was continued by oral route for the next 5 days. Sucralfate was given along with the therapy @ 0.5-1 gram, orally, TID for 10 days. Symptomatic and supportive therapy was provided based on the clinical condition of the dog.

Commented [AKHTER28]: To all?

Therapeutic response was evaluated based on the improvement in clinical and haemato-biochemical parameters. Ultrasonography of selected dogs was done on day 0 to observe gastric mucosal wall thickening and followed up on day 10 to assess the alterations in mucosal wall thickening.Similarly, gastroscopy was performed on day 0 to identify gastric lesions and was followed up on day 10 to evaluate alterations in the lesions and to evaluate the efficacy of treatment.

Commented [AKHTER29]: Where is the data or images?

2.5 Statistical analysis

The recorded data was analyzed as per the standard procedures outlined by [14]. The recorded data was analyzed by applying one-way ANOVA and mean comparisons were made by Duncan's multiple range test and paired t-test was applied for comparison within group between interval as per the standard procedure IBM SPSS computer software version 25.0.

Commented [AKHTER30]: Statistical analysis was performed

Commented [AKHTER31]: Which interval?

Commented [AKHTER32]: ?

Commented [AKHTER33]: Reframe

Results and discussion

Occurrence of chronic gastritis in dogs

A total of 30 dogs exhibiting clinical signs of chronic gastritis were screened for the presence of chronic gastritis. Endoscopy was performed in all 30 dogs for the confirmation of chronic gastritis, out of which 14 dogs were found positive for endoscopic lesions pertaining to chronic gastritis. Therefore, the occurrence of chronic gastritis among the suspected cases were 46.66% (Table 02).

Table 02: Occurrence of chronic gastritis in dogs

Particulars	No. screened	No. affected	Occurrence (%)
Dogs suspected for chronic gastritis (Endoscopic evaluation)	30	14	46.66

The occurrence of chronic gastritis in this study aligns with the findings of Krstic *et al.*[15] and Shabestari *et al*. [16]. In contrast, Kalundia[17] reported a lower prevalence of chronic gastritis compared to acute gastritis. The high occurrence of chronic gastritis in dogs in VCC, Jabalpur is might be due to poor dietary practices, including feeding low-quality or spoiled

Commented [AKHTER34]: delete

food and parasitic infections common in the warm, humid climate. Additionally, the significant stray dog population, often consuming contaminated food and water, contributes to the condition. The long-term use of NSAIDs and corticosteroids in high dose, along with stress from environmental factors, further worsen the condition.

Commented [AKHTER35]: Reference?

Overall distribution of chronic gastritis in dogs

To assess thedistribution of chronic gastritis, all 14 dogs diagnosed with chronic gastritis were distributed into various categories of age, breed and gender as mentioned in table 03. Age wise distribution of chronic gastritis was higher in age group of 1 to 3 years *i.e*. 35.71% (05/14). Breed wise distribution was recorded highest in Retrievers, accounting for 35.71%. While the gender wise distribution was higher in males (64.29%) in comparison to females (35.71%).

Commented [AKHTER36]: Interpret it in a more precise way

Table 03: Overall distribution of chronic gastritis in dogs

Age wise distribution		
Age groups (Years)	**No. positive (n=14)**	**Distribution (%)**
Up to 1	03	21.43
1 to 3	05	35.71
3 to 6	04	28.57
Above 6	02	14.29
Breed wise distribution		
Breed	**No. positive (n=14)**	**Distribution (%)**
German Shepherd	03	21.43
Retriever	05	35.71
Indian spitz	01	07.14
Non-Descript	03	21.43
Others (French Mastiff, Boxer, Great Dane, Dobermann etc.)	02	14.29
Gender wise distribution		
Sex	**No. positive (n=14)**	**Distribution (%)**
Male	09	64.29
Female	05	35.71

The results were consistent with those of Bhat *et al.*[18] and Faucher *et al*. [19]. In contrast, Seim-Wiske *et al*. [20] reported chronic gastritis more in middle-aged dogs (4 to 8 years). However, the occurrence of gastric foreign bodies was higher among dogs in 2-4 years of age (80%), which was in accordance with Boag *et al*. [21], Gianella *et al*. [22], Kalundia[17] and Poggiani*et al*. [23]. Chronic gastritis is more common in dogs aged 1-3 years due to factors like dietary indiscretion, prolonged irritant exposure, infections (e.g., *Helicobacter spp.*) and stress. Long-term medication use (e.g., NSAIDs) and unresolved acute gastritis also contribute. This might be due to more activity than adults and their inquiring nature. They may unnecessarily try to eat or feed things around them which often leads to the ingestion of foreign objects or infections, as well as weaning stress [24].

Commented [AKHTER37]: Doesn't make sense

Commented [AKHTER38]: Revise it

The findings of breed wise distribution were aligned with Kaur [25], who observed similar trends in Retrievers, further supporting the idea that breed preferences and lifestyle factors influence health conditions in pets. Sagar [26] also reported the highest occurrence in Labrador Retrievers (26.19%), followed by Rottweilers (21.42%) and German Shepherd (14.28%). This may be due to the popularity of Retrievers breeds among pet owners in Jabalpur area. The high percentage of chronic gastritis in Retrievers (35.71%) could be attributed to their frequent exposure to items like toys, bones and clothing, which increases the risk of gastrointestinal issues, including choking or the ingestion of foreign bodies.

The gender-wise distribution of chronic gastritis in dogs was higher in males compared to females, which may be attributed to a greater owner preference for male dogs. The findings of our study well collaborate with Anju *et al*. [27], Kalundia[17], Bhat *et al*. [18], Sagar [26] and Verma *et al*. [28]. Additionally, the population of sexually intact males may be higher than that of sexually intact females.

Commented [AKHTER39]: Is it dietary factor?

Commented [AKHTER40]: Check it and write the specific factor, direct or indirect cause of chronic gastric ulcer which aligns with your study. Mention it clearly

Commented [AKHTER41]: What does that signify?

Clinical parameters

Temperature (°F), pulse rate (beats/min) and respiration rate (breaths/min) of all 12 dogs in the treatment groups were recorded on day 0 and day 10, with comparisons made to the healthy control group. All the mean values of body temperature, pulse rate and respiration rate in different categories were within the normal physiological range and no significant variation was observed in clinical parameters between different groups at different intervals(Table 04).

Table 04: Mean clinical parameters of dogs affected with chronic gastritis in different treatment groups at different intervals

Parameter	Group	Day 0	Day 10
Temperature(°F)	G1	102.38±0.24	101.82±0.19
	G2	101.82±0.37	101.72±0.22
	G3	101.68±0.21	101.93±0.22
Pulse rate(beats/min)	G1	86.33±2.33	88.33±3.81
	G2	90.33±2.16	86.00±3.06
	G3	84.67±2.51	86.67±2.62
Respiration rate(breaths/min)	G1	36.00±1.73	38.17±1.60
	G2	37.00±1.53	36.33±2.28
	G3	38.00±1.46	35.67±1.74

The findings of study supported by Chandra [29] and Sagar [26], they also reported non-significant variation in rectal temperature, pulse rate and respiration rate in dogs with chronic gastritis. However, the respiration rates were at higher end of normal range, this might be due to presence of non-complicated chronic gastritis (without infection or systemic inflammation). Whereas, dehydration or electrolyte imbalances develop, compensatory slightly increased respiration rate that may occur as the body attempts to restore normal blood pH levels and

oxygenation. In contrast, Shaheen *et al*. [30] reported slight increase in temperature and pulse (104±1.24°F and 160 beats per minute, respectively).

Commented [AKHTER42]: This need to be rechecked and reframed

Commented [AKHTER43]: Add the basic reason for inc. in vitals and correlate with the studies.

Haematological Parameters

Haematological parameters *i.e*. haemoglobin (g/dl), packed cell volume (%), total leukocyte count (10^3/µl), differential leukocyte count (%) in all 12 dogs of the treatment groups were recorded on day 0 and day 10 and compared with the healthy control group.

The mean haemoglobin, PCV and TLC values were significantly higher in G1 and G2 group on day 0 as compared to healthy control group (G3) which were significantly reduced on day 10 (post-treatment) in G1 compared to G2(Table 05).

Commented [AKHTER44]: Make it one concise para

Table 05: Mean haematological parameters of dogs affected with chronic gastritis in different treatment groups at different intervals

Parameter	Group	Day 0	Day 10
Hb (g/dl)	G1	16.92^{Aa}±0.55	13.73^{Bb}±0.67
	G2	16.63^{Aa}±0.58	15.73^{Ba}±0.49
	G3	13.43^{Ab}±0.31	13.12^{Ab}±0.31
PCV (%)	G1	50.75^{Aa}±1.65	39.57^{Bb}±2.60
	G2	49.55^{Aa}±1.74	45.35^{Ba}±1.62
	G3	38.63^{Bb}±0.98	36.90^{Bb}±0.77
TLC (10^3/µl)	G1	19.97^{Aa}±2.47	12.07^{Bb}±0.67
	G2	20.22^{Aa}±1.35	14.47^{Ba}±0.63
	G3	11.80^{Ab}±0.42	12.28^{Ab}±0.26

Mean values with different superscripts between groups (lower case) and between days (upper case) differ significantly ($p \leq 0.05$)

Commented [AKHTER45]: Is this superscript or what?

The haemoglobin concentration is a critical parameter for evaluating the general health status and oxygen-carrying capacity of blood in animals. The findings of our study were corelates with Bhat *et al.* [18], Novianae*t al*. [31], Maheshwarappae*t al*. [2], Patel *et al*. [32], Khanduri *et al*. [33]. Change in haematocrit value could be attributed to dehydration caused by vomiting, which leads to fluid loss and subsequent haemoconcentration, commonly observed in gastritis [34]. However, the results were in contrary with Tyagi *et al*. [35], Arora *et al*. [36] and Monika *et al*.[37]. This might be due to compensatory physiological mechanisms or pathological changes associated with the chronic gastritis that affect gastric mucosa, potentially impairing iron absorption because of gastric acid secretion.Similar findings were recorded by Novianae*t al*. [31], Arora *et al*. [36] and Kamble *et al*. [38]. The intestinal form of ALP plays a role in nutrient absorption, particularly by breaking down certain compounds in the gut. This might be due to gastrointestinal causes such as chronic gastritis would generally be associated with liver or bile duct involvement. The increase of normal ALP concentration indicated damage of hepatocyte that was related to leakage of enzymes from cytoplasm of hepatocytes [31].

Commented [AKHTER46]: delete

Commented [AKHTER47]: Revise it

Commented [AKHTER48]: ALP was increased in your cases or what?

The increased PCV (%) in affected dogs indicated dehydration in comparison to healthy dogs and these observations simulated with the findings of Ramprabhu*et al.* [39], Kamalpreet *et al.* [40]), Agnihotri *et al.* [41], Novianae*t al*. [31] and Monika *et al*. [37] revealed significant increase in PCV value in dogs affected with vomiting. Contrarily, Arora *et al*. [36] and Mouhamed *et al*. [42] reported the reduced PCV in diseased dogs as comparison to healthy control. The mean value of TLC was observed to be higher in dogs suffering with chronic gastritis in comparison to healthy control group as found in our study. Similar findings were also reported by Suresh [43], Shah *et al*. [44], Mohanta *et al.* [45] and Khanduri *et al*. [33]. Ramprabhu*et al*. [39] recorded increase in mean value of TLC in dogs affected with vomiting due to haemorrhagic gastroenteritis. However, Maheshwarappa*et al*.[2] and Mouhamed *et al*.[42] founds the higher value of TLC in dogs affected with chronic gastritis. This leucocytosis might be due to secondary bacterial infection in the gastric mucosa of affected dogs.

Commented [AKHTER49]: In which case recorded

Commented [AKHTER50]: Bacterial infection in gastric mucosa?
Recheck

Differential leukocyte count (DLC)

Differential leukocyte count (DLC) in all 12 dogs of the treatment groups were recorded on day 0 and day 10 and compared with the healthy control group. The neutrophils, eosinophils and monocytes were significantly higher in both the groups in comparison to healthy control group. However, significant reduction in count was observed on day 10, post-treatment. However, the mean lymphocyte count was significantly lower in G1 and G2 group on day 0 as compared to healthy control group (G3) which were significantly increased on day 10 after treatment in G1 and G2 but the values were in normal physiological range and there were no significant variations were recorded in basophil count (Table 06).

Table 06: Mean differential leukocyte count of dogs affected with chronic gastritis in different treatment groups at different intervals

Parameter	Group	Day 0	Day 10
Neutrophils (%)	G1	78.17^{Aa}±1.33	70.33^{Bb}±0.61
	G2	78.00^{Aa}±0.82	73.00^{Ba}±0.45
	G3	69.67^{Bb}±1.20	69.67^{Bb}±1.28
Lymphocyte (%)	G1	12.67^{Bb}±1.33	23.50^{Aa}±1.07
	G2	13.50^{Bb}±1.34	18.17^{Ab}±0.48
	G3	25.17^{Aa}±1.40	24.67^{Aa}±1.28
Basophils (%)	G1	0.00 ±0.00	0.33 ±0.21
	G2	0.00 ±0.00	0.00 ±0.00
	G3	0.17 ±0.17	0.00 ±0.00
Eosinophils (%)	G1	4.67^{Aa}±0.67	2.83^{Bb}±0.60
	G2	4.17^{Aa}±0.75	4.33^{Aa}±0.49
	G3	2.00^{Ab}±0.45	2.50^{Ab}±0.34
Monocytes (%)	G1	4.50^{Aa}±0.56	3.00^{Bb}±0.57
	G2	4.33^{Aa}±0.33	4.50^{Aa}±0.22
	G3	3.00^{Ab}±0.26	3.17^{Ab}±0.40

Mean values with different superscripts between groups (lower case) and between days (upper case) differ significantly ($p \leq 0.05$)

The findings of the present study were agreements with Bhat *et al*. [18], Maheshwarappa*et al*. [2] and Patel *et al*.[32].Marked neutrophilia and a left shift was recorded in dogs affected with gastritis[46]. Contrarily, Stanton and Bright [47] reported the normal leukogram in dogs with gastric diseases. Monocytosis observed in dogs with chronic gastritis indicates a sustained inflammatory response characterized by the recruitment of monocytes to the gastric mucosa, where they differentiate into macrophages, playing a role in tissue repair and cytokine release. This haematological alteration is commonly associated with chronic inflammation, stress-induced bone marrow activation and antigenic stimulation frequently observed in gastrointestinal disorders. The findings of this study regarding monocyte counts were contrary to those reported by Suresh [43] and Mohanta *et al*. [45]. Eosinophils are key immune cells involved in the response to allergens and parasitic infections, as well as in modulating inflammation and tissue damage in chronic diseases [48]. The findings were in agreements of Maheshwarappa*et al*. [2] and Patel *et al*. [32]. The increase in the mean value of eosinophil might be due to inflammation of gastric mucosa or due to secondary parasitic infestation like *Ancylostoma*as opined by [43].

Commented [AKHTER51]: Which finding?

Commented [AKHTER52]: ?

Biochemical Parameters

Biochemical parameters of 12 dogs under therapeutic study were estimated on day 0 (pre-treatment) and day 10 (post-treatment).

Hepatic Biomarkers

In hepatic parameters ALT, AST and ALP were estimated on day 0 and day 10. There was no significant difference recorded in mean serum alanine aminotransferase (ALT), all the mean values in different groups and at different intervals remained within the physiological range. Whereas the AST and ALP levels were significantly decreased from day 0 to day 10 post treatment.

Commented [AKHTER53]: Combine it into one precise para

Table 07: Mean hepatic biomarkers of dogs affected with chronic gastritis in different treatment groups at different intervals

Group	Day 0	Day 10
Serum alanine aminotransferase (U/L)		
G1	70.17±3.53	71.50±2.55
G2	69.00±3.30	72.83±2.39
G3	69.50±2.06	70.33±2.12
Serum aspartate aminotransferase (U/L)		
G1	131.67^{Aa}±10.09	53.83^{Bb}±4.49
G2	132.67^{Aa}±7.23	77.33^{Ba}±3.00
G3	51.83^{Ab}±3.16	50.67^{Ab}±3.03

Serum alkaline phosphatase (U/L)		
G1	202.55^{Aa}±12.99	92.37^{Bb}±4.40
G2	204.42^{Aa}±19.88	111.62^{Ba}±9.15
G3	94.35^{Ab}±2.66	92.67^{Ab}±2.07

Mean values with different superscripts between groups (lower case) and between days (upper case) differ significantly ($p \leq 0.05$)

The findings of this study were consistent with those of Monika *et al*. [37] and Patel *et al*. [32]. In contrast, Bhat *et al*. [18] and Khanduri *et al*. [33] reported an elevation in both ALT and AST enzyme activities. Increase in the ALT was also observed by Stanton and Bright [47] and this may be due to drug induced hepatopathy. Whereas, non-significant statistical observed in ALT and AST was observed by [32]. This discrepancy may be due to the association between chronic gastritis and hepatic function. However, the medications which were administered in our study to treat chronic gastritis did not affect the hepatocytes, post-treatment.The results of present study were according with Arora *et al*. [36] and Mouhamed *et al*. [42]. However, elevated ALT and AST activity were recorded by Hendrix [49], Cooper and Webster [50] and Suresh [43] in dogs with vomition. Large amounts of AST are present in red blood cells, liver, heart, muscle tissue, pancreas and kidneys, so the destruction of any of these tissues results in the release of large amounts of this enzyme into the blood, in addition to dehydration and the passage of microbes, endotoxins via portal circulation, precipitating reactive hepatopathy. The increase in AST value could be due to dehydration [51] [52]. Similar findings were recorded by Novianaet *al*. [31], Arora *et al*. [36] and Kamble *et al.* [38]. The intestinal form of ALP plays a role in nutrient absorption, particularly by breaking down certain compounds in the gut. This might be due to gastrointestinal causes such as chronic gastritis would generally be associated with liver or bile duct involvement. The increase of normal ALP concentration indicated damage of hepatocyte that was related to leakage of enzymes from cytoplasm of hepatocytes [31].

Commented [AKHTER54]: In what case?

Commented [AKHTER55]: ?

Commented [AKHTER56]: How you can say?

Commented [AKHTER57]: Which result , which finding you want to mention that aligns with your study?

Commented [AKHTER58]: Repeating the same thing

Commented [AKHTER59]: Revise it carefully

Commented [AKHTER60]: Revise it all, write in a precise way. Your main findings aligning or in contrast with other

Renal Biomarkers

Mean creatinine and mean BUN level were estimated on day 0 and on day 10. Analysis of data revealed there were no significant difference was recorded in creatinine value between groups at different interval although mean BUN level wassignificantly higher in G1 and G2 group on day 0 as compared to healthy control group (G3) which were significantly reduced on day 10 after treatment (Table 06).

Commented [AKHTER61]: From beginning you already mentioned it. Why to repeat the same thing

Table 08: Mean creatinine and mean BUN value of dogs affected with chronic gastritis in different treatment groups at different intervals

Group	Day 0	Day 10
Mean Creatinine (mg/dl)		
G1	1.48±0.22	1.03±0.11
G2	1.20±0.07	1.08±0.11
G3	1.02±0.13	1.00±0.10

Mean BUN (mg/dl)		
G1	24.98^{Aab}±3.25	16.10^{Bb}±1.47
G2	31.73^{Aa}±4.20	30.27^{Ba}±4.06
G3	17.82^{Ab}±2.21	17.85^{Ab}±1.33

The present findings of this study were corresponding to Guzelbektes*et al.* [53] and Patel *et al*. [32]. Chronic gastritis in dogs is not directly linked with creatinine levels [53]. However, Murali [54] and Shah *et al*. [44] reported elevated serum creatinine levels in dogs represented with vomition due to gastroenteritis. Creatinine is a waste product primarily eliminated by the kidneys and alteration in normal physiological range, typically indicates impaired kidney function. Although creatinine elevation is not a direct consequence of chronic gastritis, it could occur due to secondary effects, particularly related to dehydration or pre-existing renal dysfunction [55]. The results of present study well supported by Bhat *et al*. [18], Monika *et al*. [37] and Khanduri *et al*. [33]. The elevated blood urea nitrogen levels indicate pre-renal uraemia, likely resulting from a reduced glomerular filtration rate caused by haemoconcentration in dogs experiencing vomiting due to gastroenteritis [56]. The present findings are also in accordance with Murali [54], Suresh [43] and Shah *et al*. [44]. The increase in BUN level could be due to dehydration or depleted body fluids leading to decreased renal perfusion resulting in physiological oliguria, which further impairs the excretion of waste products and uremic toxins from the body resulting in high BUN values.

Commented [AKHTER62]: ?

Therapeutic Response Evaluation

The efficacy of various therapeutic agents was assessed in distinct groups, each comprising six dogs diagnosed with chronic gastritis. The evaluation was based on the resolution of clinical signs, particularly vomiting, which was the most common sign among the affected dogs. Additionally, improvements in endoscopic lesions were monitored on day 10 and compared to the baseline endoscopic findings recorded on day 0 (Table 09).

Table 09:Evaluation of response to therapy in different treatment

Parameters / Groups	Absence of vomiting	Improvement in gastritis (endoscopic lesions)	Recovery (%)
G1	5/6 (83.33%)	5/6 (83.33%)	83.33%
G2	4/6 (66.66%)	4/6 (66.66%)	66.66%

The results of therapeutic response evaluation were in agreement with Simpson [57], Parrah*et al.* [10] and Tolbert and Gould [58]. Patel *et al.* [11] treated gastric *Helicobactor*infection with "Triple therapy" consisting amoxycillin, metronidazole and bismuth subcitrate. Manzoor and Rasool [59] therapeutically intervened with pantoprazole and amoxicillin. Whereas, Shaheen *et al.* [30] and Chanda *et al.* [60] treated gastrointestinal

infections with combination of ofloxacin and ornidazole.Triple therapy is a highly effective approach for bacterial infections, particularly in cases of chronic gastritis, due to the bacteria's rapid development of drug resistance. This regimen typically comprises two antibiotics, such as amoxicillin, clarithromycin, or metronidazole, alongside an acid-reducing agent like omeprazole (a proton pump inhibitor) or an H2 receptor antagonist. The antibiotics act synergistically to eliminate the bacteria, while the acid-reducing agent lowers gastric acid secretion, creating an environment less favourable for bacterial survival and enhancing the efficacy of antibiotics. This combination not only improves treatment success rates but also promotes mucosal healing and minimizes the risk of complications such as ulcers and gastric cancer. However, the choice of antibiotics must consider regional resistance patterns, as resistance to drugs like clarithromycin is increasingly common, making susceptibility testing vital for effective therapy[61].

Commented [AKHTER63]: Bacteria's is not even a word?

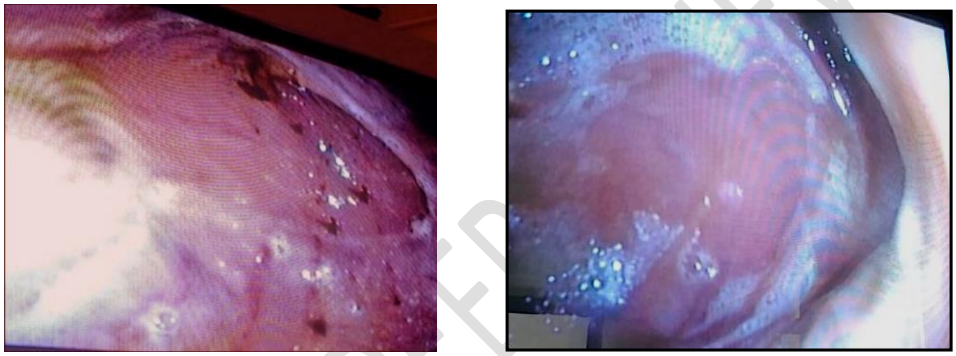

Fig 03: Endoscopic evaluation of gastric mucosa in Group 1 (a) Day 0 (b) Day 10

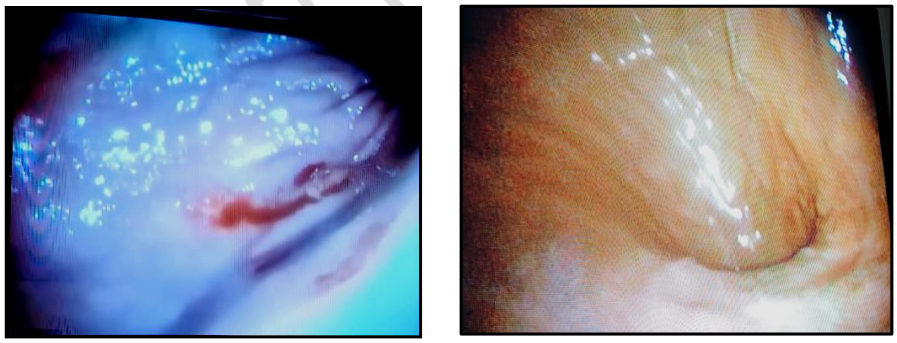

Fig 04: Endoscopic evaluation of gastric mucosa in Group 2 (a) Day 0 (b) Day 10

The combination of amoxicillin-sulbactam and metronidazole is considered more effective than ofloxacin and ornidazole for managing chronic gastritis in dogs due to their

superior antimicrobial spectrum, lower resistance rates and better safety profile compared to ofloxacin and ornidazole. This combination includes amoxicillin, a broad-spectrum penicillin that inhibits bacterial cell wall synthesis and sulbactam, a β-lactamase inhibitor that prevents bacterial resistance to amoxicillin. Together, they effectively target gram-positive and gram-negative bacteria [62]. Metronidazole disrupts the DNA of anaerobic bacteria and certain protozoa. Ofloxacin is a fluoroquinolone antibiotic with a broad spectrum of activity against aerobic Gram-negative and Gram-positive bacteria, but limited efficacy against anaerobes. Its primary mechanism of action involves inhibiting bacterial DNA gyrase, an enzyme crucial for DNA replication [63].

Pantoprazole is frequently preferred for long-term use due to its more favourable safety profile and reduced risk of drug interactions, as it is metabolized primarily via sulfation rather than the CYP enzymes. Additionally, pantoprazole binds more extensively in the proton transport pathway compared to omeprazole, resulting in a longer duration of acid suppression [64].

The groups responded well to the respective treatment regimens which was manifested with the disappearance of clinical signs and returning of the haemato-biochemical parameters to the normal physiological range. However, in group G1, 5 out 6 were fully recovered followed by group G2 where 4 out of 6 were fully recovered. Overall, the treatment regimen of amoxycillin with sulbactam, metronidazole and pantoprazole demonstrated the most significant recovery in dogs with chronic gastritis, as evidenced by both clinical and diagnostic parameters.

Commented [AKHTER64]: Revise and make it more clear by first mentioning recovery rate in groups that signs or hematobiochem normalization….

Conclusion

This research effectively established that chronic gastritis in canines can be managed with a regimen combining antibiotics and acid suppressants. Administration of amoxicillin-sulbactam, metronidazole and pantoprazole resulted in superior clinical outcomes, including enhanced mucosal healing and minimal adverse effects, compared to the combination of ofloxacin, ornidazole and esomeprazole. Endoscopic assessments indicated marked improvement in gastric mucosal integrity, consistent with the observed normalization of haematobiochemical parameters. Statistically significant recovery was noted in both treatment groups, with Group G1 exhibiting an 83.33% recovery rate versus 66.66% in Group G2. Group G1 demonstrated a more substantial reduction in vomiting episodes and endoscopic lesions, along with improved haematobiochemical normalization.

Future scope

Further studies would assess the effectiveness of alternative antibiotics and proton pump inhibitors in the treatment of chronic gastritis. A deeper investigation into the potential involvement of Helicobacter species in chronic gastritis in dogs could help guide more targeted therapies, such as the use of specific anti-Helicobacter agents.

Commented [AKHTER65]: italics

Commented [AKHTER66]: What was the main reason for chr. Gastritis in your study?.

Commented [AKHTER67]: This is not the future scope.

References

1. Rugge, M., Pennelli, G., Pilozzi, E., Fassan, M., Ingravallo, G., Russo, V.M. and Mario, F.D. (2011). Gastritis: The histology report. *Digestive and liver Diseases*, **43**(4): 373-384.
2. Maheshwarappa, Y.P. (2020). Study on effect of pantoprazole and ranitidine in canine gastritis. *Journal of Entomology and Zoology Studies*, **8**(1): 754-757.
3. Cote, Ettinger, S.J. and Feldman, E.C. (2024). Feline myocardial disease. *In:* Ettinger's Textbook of Veterinary Internal Medicine, Vol 2, 9th Edn., Elsevier, Canada, pp 1671-1675.
4. Amorim, I., Taulescu, M.A., Day, M.J., Catoi, C., Reis, C.A., Carneiro, F. and Gartner, F. (2016). Canine gastric pathology: A review. *Journal of Comparative Pathology*, **154**(1): 9-37.
5. Berghoff, N. and Steiner, J.M. (2011). Laboratory tests for the diagnosis and management of chronic canine and feline enteropathies. *Veterinary Clinics of North America- Small Animal Practice*, **41**(2): 311–328.
6. Day, M.J., Bilzer, T., Mansell, J., Wilcock, B., Hall, E.J., Jergens, A., Minami, T., Willard, M. and Washabau, R. (2008). Histopathological standards for the diagnosis of gastrointestinal inflammation in endoscopy biopsy samples from the dog and cat: A report from the World Small Animal Veterinary Association Gastrointestinal Standardization Group. *Journal of Comparative Pathology*, **138**(1): 1-43.
7. Wilcock, B. (2013). Histopathology. In*: Canine and Feline Gastroenterology*, Washabau, R.J. and Day, M.J. (eds.), Elsevier Saunders, St. Louis, pp 333-385.
8. Koster, L.S. (2018). Chronic gastritis. *In*: Chronic Disease Management for Small Animals. Gram, W.D., Milner, R.J. and Lobetti, R. (ed.). Wiley Blackwell, USA, pp 193-199.
9. Colakoglu, E.C., Borku, K., Haydardedeoglu, A.E., Alihosseini, H., Senel, O.O., Yumuşak, N. and Ugurlu, L. (2017). Correlation between endoscopic and histopathological findings in dogs with chronic gastritis. *Journal of Veterinary Research*, **61**(3): 351-355.
10. Parrah, J.D., Moulvi, B.A., Gazi, M.A., Makhdoomi, D.M., Athar, H., Dar, S. and Mir, A.Q. (2013). Gastric ulceration in dog: A review. *Veterinary World*, **6**(7): 449-454.
11. Patel, P.K., Patel, S.K., Dixit, S.K. and Rathore, R.S. (2018). Gastritis and peptic ulcer diseases in dogs: A review. *International Journal of Current Microbiology and Applied Sciences*, **7**(3): 2475-2501.
12. Dixit, S.K., Patel, P.K., Yadav, S., Patel, S.K. and Verma, N. (2022). Vomiting in dogs– A review. *IntasPolivet*, **23**: 99-104.
13. Birchard, S.J. and Sherding, R.G. (2005). Saunders Manual of Small Animal Practice-E-Book. Elsevier Health Sciences.
14. Snedecor, G.W. and Cochran, W.G. (1994). Statistical Method, 8th Edn., The Lowa State University Press, Ames, United States of America.
15. Krstic, V., Marinković, D., Vakanjac, S., Lalošević, D. and Aleksić-Kovačević, S. (2006). *Helicobacter* Spp. in gastroscopic biopsies in dogs. *Acta veterinaria*, **56**(4): 305-314.
16. Shabestari, A.S., Mohammadi, M., Jamshidi, S., Sasani, F., Bahadori, A. and Oghalaie, A. (2008). Assessment of chronic gastritis in pet dogs and its relation with *Helicobacter*-like organisms. *Pakistan Journal of Biological Sciences*, **11**(11): 1443-1448.
17. Kalundia, A. (2012). Studies on advanced diagnostic techniques and therapeutic management of chronic gastritis in dogs. M.V.Sc thesis. Sri Venkateswara Veterinary University, Tirupati andhra Pradesh.
18. Bhat, A.A., Wadhwa, D.R., Mandial, R.K., Sharma, A., Katoch, A. and Sharma, P. (2015). Clinico-biochemical alterations and therapeutic management of canine gastroenteritis. *Journal of Animal Research*, **5**(1): 149-153.
19. Faucher, M.R., Biourge, V., German, A.J. and Freiche, V. (2020). Comparison of clinical, endoscopic and histologic features between dogs with chronic gastritis with and without lymphofollicular hyperplasia. *Journal of the American Veterinary Medical Association*, **256**(8): 906-913.
20. Seim-Wikse, T., Skancke, E., Nodtvedt, A., Jorundsson, E., Grotmol, T., Kristensen, A.T. and Bjornvad, C.R. (2019). Comparison of body condition score and other minimally invasive biomarkers between dogs with gastric carcinoma and dogs with chronic gastritis. *Journal of the American Veterinary Medical Association*, **254**(2): 226-235.
21. Boag, A.K., Coe, R.J., Martinez, T.A. and Hughes, D. (2005). Acid-base and electrolyte abnormalities in dogs with gastrointestinal foreign bodies. *Journal of Veterinary Internal Medicine*, **19**(6): 816–821.
22. Gianella, P., Pfammatter, N.S. and Burgener, I.A. (2009). Oesophageal and gastric endoscopic foreign body removal: complications and follow-up of 102 dogs. *Journal of Small Animal Practice*, **50**(12): 649–654.

23. Poggiani, F.M., Duarte, R.P.C., Santana, M.I.S. and Galera, P.D. (2020). Endoscopic removal of foreign body in upper gastrointestinal tract in dogs: success rate and complications. *Acta Scientiae Veterinariae*, **48**: 1735.
24. Macartney, L., McCandlish, I.A.P. and Thomson, H. (1984). Canine Parvovirus enteritis 1: Clinical, haematological and pathological features of experimental infection. *The Veterinary Record*, **115**(9): 201-210.
25. Kaur, J. (2012). Diagnostic approaches to gastrointestinal tract disorders and their surgical management in canines. M.V.Sc. thesis. Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab.
26. Sagar, R.S. (2016). Clinico-biochemical and diagnostic imaging studies on gastric disorders in dogs. Doctoral dissertation, College of Veterinary and Animal Sciences, Pookode, Wayanad.
27. Anju, S., Vijayakumar, G., Srinivasan, S.R. and Raj, G.D. (2008). Study of *Helicobacter* infection in dogs with chronic gastritis. *Indian Veterinary Journal*, **85**(10): 1123.
28. Verma, R., Jawre, S., Shahi, A., Vaish, R., Singh, R., Das, B. and Chandrapuria, V.P. (2020). Interventional gastroscopy in dogs. *Indian Journal of Veterinary Surgery*, **41**(1): 47-49.
29. Chandra, D.S. (2016). Advanced diagnostic modalities and surgical management of upper gastrointestinal tract disorders in dogs. M.V.Sc. thesis. Veterinary Surgery and Radiology, Nanaji Deshmukh Veterinary Science University, Jabalpur, Madhya Pradesh.
30. Shaheen, M., Zaman, R. and Muhee, A. (2010). Clinical, endoscopic and therapeutic studies on canine gastritis. *IntasPolivet*, **11**(2): 293-296.
31. Noviana, D., Afidatunnisa, K., Syafikriatillah, A.R., Ulum, M.F., Gunanti and Zaenab, S. (2017). Diagnostic imaging and endoscopy of the Schnauzer dog with upper gastrointestinal tract disorders. *Journal KedokteranHewan*, **11**(1): 1-5.
32. Patel, P.K., Dixit, S.K., Kumar, A., Saxena, A.C., Kumar, N.V. and Bhatt. S. (2020). Study of alteration in hemato-biochemical indices in canine gastritis. *Haryana Veterinarian*,**59**(SI): 87- 89.
33. Khanduri, R., Raval, S.K., Rao, N. and Dave, K. (2021). Hemato-biochemical alterations in dogs with vomition. *Indian Journal of Veterinary Science and Biotechnology*, **17**(1): 52-55.
34. Lidbury, J.A., Suchodolski, J.S. and Steiner, J.M. (2009). Gastric histopathologic abnormalities in dogs: 67 cases (2002-2007). *Journal of American Veterinary Medical Association*, **234**(9): 1147-1153.
35. Tyagi, S.P., Varshney, A.C. and Kumar, A. (2010). Prophylactic efficacy of Seabuckthorn oil vis-a-vis other gastroprotective agents against gastric ulcerations and erosions.In *Proceedings of the 34th Annual Congress and International Symposium on Newer Concepts on Surgical Techniques for Farm and Companion Animal Practice*, pp 60. College of Veterinary Sciences, Pondicherry, India.
36. Arora, R., Tyagi, A., Shekhar, S., Rajora, V.S. and Arora, N. (2018). Hemato-biochemical alterations in gastroenteritis affected dogs. *Journal of Entomology and Zoology Studies*, **6**(5): 972-974.
37. Monika, S.K., Rana, A., Dimri, U. and Yadav, S. (2018). A study on serum gastrin-17 and cortisol levels in canine gastritis. *Journal of Pharmacognosy and Phytochemistry*, **7**(1): 1601-1604.
38. Kamble, K.D. (2021). Treatment of chronic gastritis in an adult Labrador working dog: A case study. *International Journal of Veterinary Sciences and Animal Husbandry*, **6**(3): 22-24.
39. Ramprabhu, R., Prathaban, S., Nambi, A.P. and Dhanapalan, P. (2002). Haemorrhagic gastroenteritis in dogs: a clinical profile. *Indian Veterinary Journal*, **79**(4): 374-376.
40. Kamalpreet Kaur, K.K., Sharma, S., Sandhu, K.S. and Sharma, D.R. (2005). Haemorrhagic gastroenteritis in dogs. *Indian Journal of Veterinary Medicine*, **25**(2): 72-75.
41. Agnihotri, D., Singh, Y., Maan, S., Jain, V.K., Kumar, A., Sindhu, N. and Anita, A. (2017). Molecular detection and clinico-haematological study of viral gastroenteritis in dogs. *Haryana Veterinarian*, **56**(1): 72-76.
42. Mouhamed, N.M., Ali, A.O., Ibrahim, K. and Mahmoud, A.E. (2023). Leverage of gastroenteritis on blood and biochemical profile in dogs. *Suez CanalVeterinary Medical Journal*, **28**(1): 45-54.
43. Suresh, K. (2009). Advanced studies on differential diagnosis and therapeutic management of vomiting in dogs. Doctoral thesis, Sri Venkateswara Veterinary University, Tirupati andhra Pradesh, India.
44. Shah, S.A., Sood, N.K., Wani, N., Gupta, K. and Singh, A. (2013). Haemato-biochemical changes in canine Parvo viral infection. *Indian Journal of Veterinary Pathology*, **37**(2): 131-133.
45. Mohanta, L., Mohanty, A.K. and Sahoo, N. (2018). Impact of haemorrhagic gastroenteritis on canine total leucocyte count and differential leucocyte count. *International Journal of Development Research*, **8**(6): 21056-21058.
46. Fitzgerald, E., Barfield, D., Lee, K.C.L. and Lamb, C.R. (2017). Clinical findings and results of diagnostic imaging in 82 dogs with gastrointestinal ulceration. *Journal of Small Animal Practice*, **58**(4): 211-218.

47. Stanton, M.E. and Bright, R.M. (1989). Gastroduodenal ulceration in dogs. *Journal of Veterinary Internal Medicine*, **3**(4): 238-244.
48. Weiss, D.J. and Wardrop, K.J. (2010). Eosinophils and their disorders. *In:* John wiley and sons. Schalm's Veterinary Hematology, 6th Edn., Wiley-Blackwell Publishing, pp 281.
49. Hendrix, A.D. (2004). Diagnosis of chronic active hepatitis in a Miniature Schnauzer. *In*: Canine and Feline. A Waverly Company, Philadelphia, pp 1156-1157.
50. Cooper, J. and Webster, C.R. (2006). Acute liver failure. *Compendium on Continuing Education for the Practicing Veterinarian*, **28**(7): 498-515.
51. Kaneko, J.J., Harvey, J.W. and Bruss, M.L. (2008). Clinical Biochemistry of Domestic Animals, 6th Edn., Academic Press, New York.
52. Shima, F.K., Apaa, T.T., Tion, M.T., Atuna, S.T. and Omobowale, T.O. (2022). Clincopathological findings in some cases of canine gastroenteritis in Nigeria. *Journal of Animal Research and Veterinary Science*, **6**: 33.
53. Guzelbektes, H., Coskun, A., Ortatatli, M., & Sen, I. (2008). Serum gastrin in dogs with acute or chronic gastritis and positive or negative for Helicobacter sp in the stomach. *BULLETIN OF THE VETERINARY INSTITUTE IN PULAWY*, *52*(3).
54. Murali, K.M. (2003). Studies on renal failure and its clinical management in canines. Doctoral thesis, SriVenkateswara Veterinary University Tirupatiandhra Pradesh, India.
55. Polzin, D.J. (2005). Chronic kidney disease. *In*: Ettinger, S.J. and Feldman, E.C. (ed.). Textbook of Veterinary Internal Medicine, 6th Edn., Elsevier Saunders, pp 1991–2000.
56. Coles, E.H. (1986). Veterinary Clinical Pathology. 4th Edn., W.B. Saunders Company, Philadelphia, pp 80-96.
57. Simpson, K.W. (2006). Chronic gastritis in companion animals. *World Small Animal Veterinary Association World Congress Proceedings*, 392-395.
58. Tolbert, K. and Gould, E. (2020). Gastritis and gastric ulceration in dogs and cats. *Clinical Small Animal Internal Medicine*: 547-555.
59. Manzoor, I. and Rasool, A. (2024). Comprehensive evaluation and management of chronic gastritis in a dog: A case report. *Journal of Veterinary Animal Science*, **55**(3): 673.
60. Chanda, S., Pradhan, N.R. and Lodh, C. (2014). Acute bacterial enteritis in pups and it's treatment. *Indian Journal of Canine Practice*, **6**(2): 123.
61. Birchard, S.J. and Sherding, R.G. (1994). Saunders Manual of Small Animal Practice. Universidade Federal de Minas Gerais, Belo Horizonte, Brazil.
62. Soutric, J., Bantar, C., Caruso, N., Heguilen, R., Casellas Jr, J.M., Casellas, J.M. and Rodriguez, M. (2006). Review of pharmacokinetic, pharmacodynamic and clinical studies with a modern combination of amoxicillin/sulbactam. *Chemotherapy***, 52**(4): 200-204.
63. Todd, P.A. and Faulds, D. (1991). Ofloxacin: a reappraisal of its antimicrobial activity, pharmacology and therapeutic use. *Drugs*, **42**: 825-876.
64. Sachs, G. and Shin, J.M. (2004). The basis of differentiation of PPIs. *Drugs of Today (Barcelona, Spain: 1998)*, **40**: 9-14.