Original Research Article

DIAGNOSIS AND THERAPEUTIC MANAGEMENT OF CHRONIC GASTRITIS IN DOGS USING TRIPLE THERAPY

ABSTRACT

Chronic gastritis, characterized by persistent vomiting and gastric mucosal inflammation, possess a significant challenge in veterinary practice. This six-month study aimed to diagnose and evaluate the therapeutic management of chronic gastritis in dogs using triple therapy. In this study chronic gastritis was diagnosed on the basis of history, clinical signs, haemato-biochemical parameters and endoscopy. Among the 30 dogs examined via endoscopy, 14 were confirmed positive based on visible gastric mucosal alterations. The parameters like age, sex and breed of each dog having chronic gastritis were recorded to study the distribution of condition. The highest cases of chronic gastritis were seen in dogs aged 1-3 years. Retrievers had the most cases among breeds and males were affected more than females. For therapeutic study, 12 confirmed cases of chronic gastritis were selected and randomly divided into 2 groups as G1 and G2, each group comprising of six dogs. However, six apparently healthy dogs were included as a healthy control group (G3). Group G1 received the Amoxicillin with sulbactam, Metronidazole and Pantoprazole while group G2 received Ofloxacin, Ornidazole and Esomeprazole. Endoscopic evaluations on day 0 and day 10 showed significant improvement in both groups, with G1 achieving an 83.33% recovery rate compared to 66.66% in G2. G1 treated with Amoxicillin with sulbactam, Metronidazole and Pantoprazole 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 the 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 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, inappropriate eating habits, 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]. Apart from the most common manifestation of persistent vomiting, other signs such as weight loss, loss of appetite and hematemesis are 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 rapid development of bacterial 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 antacids lower 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 gastric ulcers and neoplastic changes.

2. MATERIALS AND METHODS

The proposed study was carried out for six months 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 with history of clinical signs such as inappetence, vomiting, hematemesis, weight loss, retching, etc., were thoroughly examined. The complete history of the patient, including age, gender, breed, duration of illness, was recorded. The diagnosis of chronic gastritis was made based on history, clinical signs, hemato-biochemical parameters and endoscopy.


Fig. 1: Vomiting in chronic gastritis affected dogs

2.1 Clinical examination

Each dog was clinically examined for the following parameters on day 0 (pre-treatment) and day 10 (post-treatment): Temperature (°F), Pulse rate (beats/minute) and Respiratory rate (breaths/minute).

2.2 Sample collection

A specimen of approximately 3 ml of blood was aseptically drawn from each dog's saphenous or cephalic vein into a vacutainer. The serum was extracted by centrifugation (3000 rpm for 5 minutes) and stored at -20°C for biochemical analysis. Hematological parameters (hemoglobin, PCV, TLC, and DLC) were measured using the IDEXX ProCyte Dx™ analyzer on days 0 (pre-treatment) and 10 (post-treatment). Serum biochemical parameters (ALT, AST, ALP, creatinine, and BUN) were assessed with a CHEM-5 plus semi-automatic analyzer using a ready-made kit on the same days, following standard protocols.

2.3 Diagnostic imaging

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.

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 a healthy control group designated as group G3 (Table 1).

Table 1: Therapeutic regimen for chronic gastritis in dogs

Group	Number of animals	Drugs	Dose rate	Route	Duration
G1	06	Amoxicillin+ sulbactam	@ 10mg/kg	IV	BID for 5 days
		Metronidazole	@ 20mg/kg	IV	BID for 5 days
		Pantoprazole	@ 1mg/kg	IV	OD for 5 days
G2	06	Ofloxacin	@ 5mg/kg	IV	BID for 5 days
		Ornidazole	@ 20mg/kg	IV	BID for 5 days
		Esomeprazole	@ 1mg/kg	IV	OD for 5 days
G3	06	Apparently healthy control			

Therapy was continued by oral route for the next 5 days. Sucralfate was given in confirmed cases 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.

Therapeutic response was evaluated based on the improvement in clinical and haemato-biochemical parameters. 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.

2.5 Statistical analysis

The statistical analysis was performed according to 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 groups between intervals (day 0 and day 10).

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 2).

Table 2: 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 might be due to poor dietary practices, including feeding low-quality or spoiled 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 doses, along with stress from environmental factors, further worsen the condition.

Overall distribution of chronic gastritis in dogs

To assess the distribution 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. The distribution of chronic gastritis was highest in dogs aged 1-3 years, with the highest cases in Retrievers and a higher distribution in males compared to females.

Table 3: 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
Nondescript	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 their tendency to unnecessarily eat or chew things around them, which often leads to the ingestion of foreign objects or infections, as well as the stress associated with weaning [24].

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 (dietary habits, feeding patterns) 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.

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. In our study the population of sexually intact males was higher than that of sexually intact females. 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].

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 4).

Table 4: 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 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) due to the body's physiological response to inflammation and infection.

Haematological Parameters

Haematological parameters (haemoglobin, PCV, TLC and 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 the G1 and G2 group on day 0 as compared to the healthy control group (G3) which were significantly reduced on day 10 (post-treatment) in G1 compared to G2 (Table 5).

Table 5: 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 ($p \leq 0.05$)

The findings of our study were correlates with Bhat *et al*. [18], Noviana *et al*. [31], Maheshwarappa *et 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 Noviana *et al*. [31], Arora *et al*. [36] and Kamble *et al.* [38].

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], Noviana *et 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 an 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] found 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.

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 the 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 6).

Table 6: 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 neutrophil count in 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 of this study regarding eosinophil count 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].

Biochemical Parameters

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

Hepatic Biomarkers

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.

Table 7: 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 regarding ALT and AST 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 in gastritis cases. Increase in the ALT was also observed by Stanton and Bright [47] and this may be due to drug induced hepatopathy. Whereas, non-significant statistics observed in ALT and AST were observed by Patel *et al.* [32]. This discrepancy may be due to the association between chronic gastritis and hepatic function. However, the medications administered in this study for the treatment of chronic gastritis had no adverse effects on hepatocytes, as evidenced by normal hepatic parameters post-treatment. The results of present study related to AST levels were according to 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 vomiting. 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 Noviana *et 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 to hepatocytes that was related to leakage of enzymes from cytoplasm of hepatocytes [31].

Renal Biomarkers

Analysis of data revealed there were no significant differences recorded in creatinine value between groups at different intervals although mean BUN level was significantly 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 6).

Table 8: 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

vomiting 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 the present study are 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, creatinine and BUN from the body resulting in high BUN values.

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 9).

Table 9: 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 amoxicillin, 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 rapid development of bacterial 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].

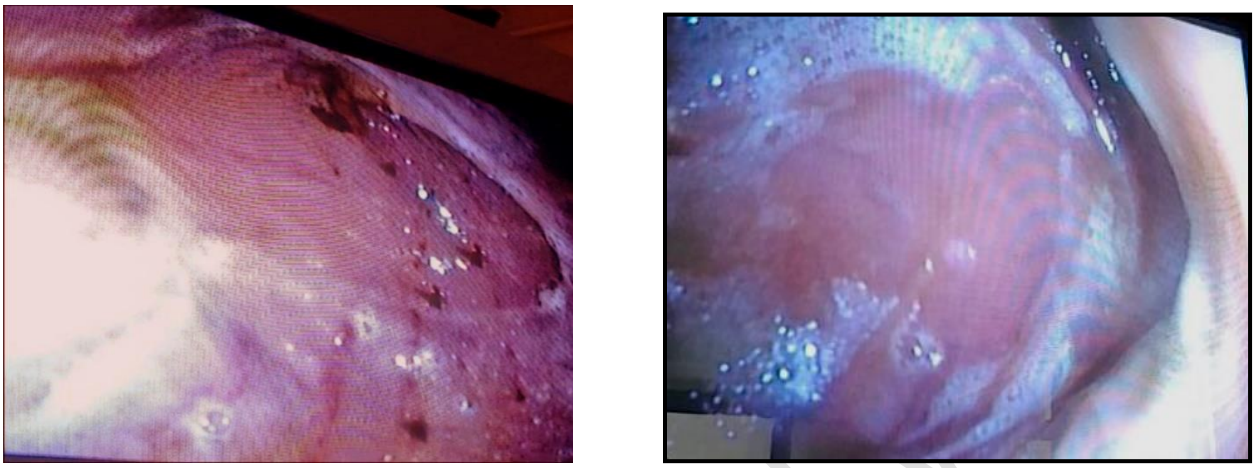

Fig 2: Endoscopic evaluation of gastric mucosa in Group 1 (a) Day 0 (b) Day 10

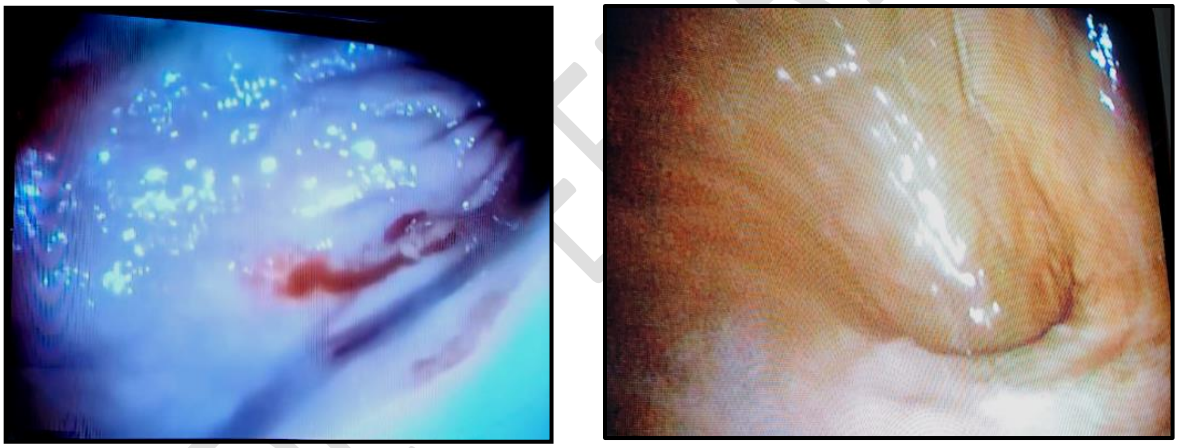

Fig 3: Endoscopic evaluation of gastric mucosa in Group 2 (a) Day 0 (b) Day 10

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 (especially vomiting) and returning of the haemato-biochemical parameters to the normal physiological range (Table 5,6,7,8). However, in group G1, 5 out of 6 were fully recovered followed by group G2 where 4 out of 6 were fully recovered. Overall, the treatment regimen of amoxicillin with sulbactam, metronidazole and pantoprazole demonstrated the most significant recovery in dogs with chronic gastritis, as evidenced by both clinical and diagnostic parameters.

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 haemato-biochemical 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 haemato-biochemical normalization.

FUTURE SCOPE

Further studies could assess alternative antibiotic combinations and proton pump inhibitors to determine the most effective treatment while minimizing bacterial resistance. 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. The integration of molecular diagnostics and biomarker-based tests could enhance early detection and treatment precision.

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

ACKNOWLEDGEMENT

The authors are thankful to the Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, NDVSU, Jabalpur, Madhya Pradesh, India for providing support to the research work.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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. *Intas Polivet*, **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. *Intas Polivet*, **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 Kedokteran Hewan*, **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 Canal Veterinary 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, Sri Venkateswara Veterinary University Tirupati andhra 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.