Chapter

An Update on Canine Duodenal Disorders

Mani Saravanan

Abstract

Duodenal disorders are difficult to identify clinically because of the non-specific symptoms. They may or may not be identifiable through routine radiographic, ultrasonographic, or laboratory evaluation methods, because many diseases of duodenum primarily involve the mucosal surface. Duodenal ulcers, inflammatory bowel disease (IBD), and small intestinal bacterial overgrowth (SIBO) are the duodenal disorders. Duodenoscopy is an efficient way of identifying the abnormalities of mucosal irregularities, distortion, ulceration, neoplasia, and inflammation, and it is also useful for obtaining mucosal biopsy and duodenal juice for routine histopathological and bacteriological studies. This paper will provide more detail about the advanced diagnostic methods and therapeutic measures for various duodenal disorders.

Keywords: duodenal ulcer, IBD, SIBO, duodenoscopy, malabsorption

1. Introduction

Gastrointestinal disorder is one of the commonly encountered problems in canine practice. Chronic disorders of small intestine of dogs are one among the major problems. Majority of the disorders can be successfully managed if diagnosed and treated earlier. If small intestine disorders are not treated appropriately, they may result in severe malabsorptive disease and death. Chronic form of intestinal disease usually disrupts the normal function of the small intestine and results in vomiting, diarrhea, weight loss, and reduced appetite [1]. Duodenal disorders like ulcers, inflammatory bowel disease (IBD), and small intestinal bacterial overgrowth (SIBO) play a major role in the small intestinal disorders [2]. Duodenal disorders are difficult to identify clinically because of the non-specific symptoms. They may or may not be identifiable through routine radiographic, ultrasonographic, or laboratory evaluation methods, because many diseases of duodenum primarily involve the mucosal surface [3]. Thus diagnosis and treatment of the duodenal disorders are challenging for the canine practitioners [4]. Duodenoscopy is an efficient way of identifying the abnormalities of proximal small intestinal mucosa including mucosal irregularities, distortion, ulceration, neoplasia, inflammation, and other mucosal disorders of the duodenum. Duodenosocopy is also useful for obtaining mucosal biopsy and duodenal juice for routine histopathological and bacteriological studies [5].

2. Incidence

Twet [6] listed out the common duodenal disorders as inflammatory bowel disease, lymphangiectasia, ulcer, foreign body, *Giardia*, parasites (Strongyloides, *Ascaris*), bacterial overgrowth, and neoplasia.

Duodenal disorders	Breed	Reference
Gastroduodenal ulceration and erosion (GDUE)	Mixed breeds—2 years to 17 years of age groups of dogs	[7]
Spontaneous gastroduodenal perforation	Rottweiler, Dobermann Pinscher, German Shepherd, and Labrador Retriever	[8]
Gastrointestinal ulceration with intervertebral disk prolapse	Dachshunds	[9]
Congenital form of duodenal diverticulum	Boxer	[10]
Gastrointestinal perforation by meloxicam toxicity	Rottweiler, Springer Spaniel, Siberian Husky, and Newfoundland	[11]
Inflammatory bowel disease (IBD)	German Shepherd, Boxers, and Yorkshire Terriers; Labrador Retrievers and Spitz and Dobermann Pinscher	[12, 13]
Chronic lymphocytic-plasmacytic enteritis	Basenji	[14]
Small intestinal bacterial overgrowth (SIBO)	German Shepherd Mongrel and Spitz	[15–17]
Lymphangiectasia	Labrador Retriever, Yorkshire Terriers, Dachshund, and German Shepherd dog	[18]
Neoplasia—duodenal gastrinoma	No breed specific	[19]
Giardia	No breed specific	[20, 21]
Duodenal foreign body • Bones (**Figure 5**) & Thread (**Figure 6**)	No breed specific commonly in puppies	[22, 23]
Duodenal worms • *Ancylostoma caninum* (**Figure 1**)	Spitz	[24]

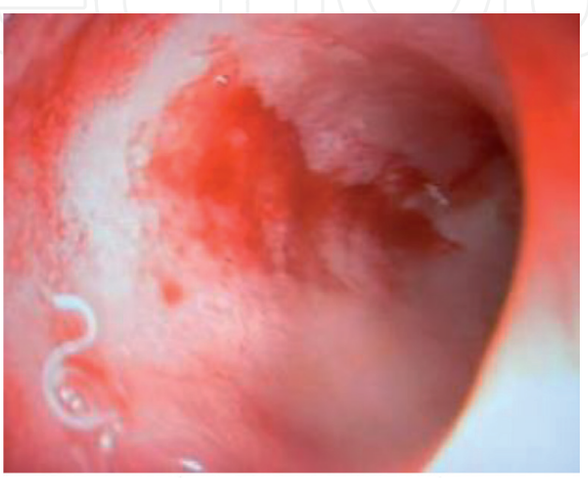

Figure 1.
Duodenoscopy: proximal duodenum—Ancylostoma caninum.

3. Pathogenesis

Neiger et al. [25] and Reed [26] opined that corticosteroids increased the secretion of free and total hydrochloric acid, diminished mucus secretion, and promoted bacterial colonization of peptic ulcers. Hinton et al. [8] reported that the common predisposing factors for gastroduodenal ulcer were nonsteroidal anti-inflammatory drug (NSAID) administration, corticosteroids, hepatic disease, major surgery, shock, decreased gastric circulation, gastric hyper secretions, and gastrointestinal neoplasia and predisposing factors for gastroduodenal perforation were IBD, non-neoplastic infiltrative gastrointestinal disease, *Dirofilaria immitis*, otitis externa, ulcerative dermatitis, dilated cardio myopathy, polycystic kidney, and thyroid illness. Various authors reported that nonsteroidal anti-inflammatory drug administration resulted in duodenal ulcer formation was primarily due to the inhibition of prostaglandin synthesis via inhibition of cyclooxygenase [26–29]. Roherer et al. [30] recorded that degenerative disk diseases along with corticosteroid administration induced ulceration.

Dossin and Henroteaux [12], Jergens [31], Pibot et al. [32], and Kobayashi et al. [33] described inflammatory bowel disease as a group of idiopathic, chronic, gastrointestinal tract disorders, characterized by infiltration of gastrointestinal tract mucosa by inflammatory cells like lymphocytes, plasma cells, and various forms of IBD including lymphocytic-plasmacytic, eosinophilic, and granulomatous enterocolitis.

Mucosal immune system might play an important role in the pathogenesis of small intestinal enteropathies like IBD and idiopathic SIBO. Increased numbers of CD_4 T cells, IgA, IgG, and plasma cells were noticed in duodenal mucosa of dogs with IBD [34]. Cave [2] stated that heating the amino acid, lysine, reacted with reducing sugars to form Maillard compounds that cannot be digested or absorbed in a usable form and served as substrate for luminal bacteria in the small intestine, leading to quantitative or qualitative changes in the flora.

Dossin and Henroteaux [12] opined that gastroduodenal ulceration, erosion, delayed gastric emptying, and postprandial discomfort could lead to IBD. Sancho et al. [14] recorded significantly high serum gastrin in chronic lymphocytic-plasmacytic enteritis (LPE) due to duodenogastric reflux and further reported that refluxes of duodenal contents like bile, pancreatic juice, and other duodenal secretions in the stomach damaged the gastric mucosal barriers leading to an antral hypomotility and gastric acid hypersecretion.

Simpson et al. [35] reported that exocrine pancreatic insufficiency also caused small intestinal bacterial overgrowth in dogs. Small intestinal bacterial overgrowth was defined as proliferation of abnormal numbers of bacteria in the upper small intestine. Common bacteria noticed in the proximal small intestine of dog included *Streptococcus faecalis*, other *Streptococcus species*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Staphylococcus xylosus*, other *Staphylococcus* spp., *Enterobacteriacea*, *Bacillus* sp., *Escherichia coli*, *Corynebacterium* sp., *Enterobacter cloacae*, *Pseudomonas* sp. and *Pasteurella multocida*, *Clostridium subterminale*, *Clostridium perfringens*, *Bifidobacterium* sp., *Eubacterium* sp., *Bacteroides fragilis*, *other Bacteroides* spp., *Peptostreptococcus tetradius*, *Peptostreptococcus* sp., *Propionibacterium acne*, and *Lactobacillus* sp. [60]. In small intestinal bacterial overgrowth, an increased numbers of luminal bacteria in the small intestine could damage the brush border of enterocytes directly or indirectly by attracting polymorphonucleocytes, deconjugating bile acids, producing hydroxylated fatty acid and alcohols, metabolizing dietary nutrients, and altering the absorptive function of the cells [36]. Rinkinen et al. [37] stated that IgA deficiency had been associated with increased susceptibility of small intestinal bacterial overgrowth and inflammatory bowel disease.

Intestinal lymphangiectasia was characterized by the abnormal dilatation of lymphatic vessels within the mucosa and submucosa, associated with exudation of protein-rich lymph, into the intestine resulting in lipid malabsorptions [32]. Primary or congenital lymphangiectasia was a result of insufficiency or aplasia of lymphatic vessels, and secondary or acquired lymphangiectasia was due to functional obstruction of the lymphatics, and it might be due to the right side heart failure, constrictive heart failure, or intestinal neoplasia and inflammation [37].

Bonfanti et al. [39] reported that adenocarcinoma, carcinoma, leiomyoma, leiomyosarcoma, fibrosarcoma, lymphoma, and extra medullary plasmacytoma were the most common gastrointestinal tumors in dogs.

German [40] reported that *Giardia* was a flagellate protozoon, transmitted by direct life cycle, and it predominantly inhabit the duodenum of dogs. *Giardia duodenalis* is the common cause of chronic diarrhea in dogs [41].

Richter [42] and Leib and Matz [43] recorded a variety of foreign bodies like fruit pits, rubber ball, cloth, metal objects, coins, sponges, hair ball, bottle caps, chew toys, marbles, and bones in duodenum.

Clinical signs	**IBD**	**Ulcers**	**SIBO**	**Lymphangiectasia**	***Giardia***	**Neoplasia**	**Parasites**	**Foreign body**
Vomiting	1	2	5	4	5	2	2	1
Diarrhea	2		1	1	1	3	1	2
Poor response to treatment	4							
Reduced appetite	5		7		3			5
Lethargy	6			5	4	4	3	
Previous steroids/NSAIDs		1						
Abdominal pain		3					4	4
Abdominal distension		6	6					6
Edema/ascites				3				
Weight loss	3		2	2		1		
Steatorrhea			4					
Smell in feces			3					
Melena		4				5		7
Hematemesis		5				6		
Dehydration					2			
Anemia	7	7				7		8
Potbelly				6			5	
Cough							6	
Constipation								3
References	[13, 33, 44, 45]	[7, 8]	[15–17]	[38]	[46, 47]	[48]	[49]	[22]

Table 1.
Common clinical signs of various duodenal disorders.

4. Diagnosis

4.1 History

Diagnosis of duodenal disorder is challenging one, because of the non-specific signs. It requires invasive and noninvasive diagnostic modalities, viz., X-ray, ultrasound, and duodenoscopy, to identify the lesion localization. To confirm the root cause of the duodenal disorder, histopathology, cytology, and culture examination of duodenal aspirates through routine duodenoscopy examination are performed.

4.2 Clinical signs

The clinical signs of various duodenal disorders were recorded by various authors in different periods which were presented in the **Table 1**. The signs of duodenal disorders were arranged serially according to the episode of the disorders from 1, 2,..... to 7 & 8.

4.3 Hematological changes

In dogs with prolonged gastrointestinal bleeding, iron deficiency could develop microcytic hypochromic anemia [50]. Normocytic normochromic anemia in dogs due to GI ulceration and contentious administration of NSAIDs [51].

Jergens et al. [52] observed leukocytosis and relative polycythemia in five dogs and non-regenerative anemia in one dog with gastroduodenal ulceration. Leib and Matz [43] recorded regenerative anemia and eosinophilia in *Ancylostoma*-infected dogs. Ridgway et al. [53] observed thrombocytopenia in dogs with IBD. Ristic and Stidworthy [54] reported severe iron-deficiency (microcytic hypochromic) anemia due to inflammatory bowel disease in the dogs. Dossin and Henroteaux [12] observed chronic anemia with typical feature of iron deficiency (microcytic hypochromic anemia) and thrombocytopenia in IBD-affected dogs.

4.4 Biochemical changes

Hall [55] suggested that panhypoproteinemia (hypoalbuminemia and hypoglobulinemia) was typical of protein-losing enteropathy (PLE) and also reported that hyperglobulinemia was noticed in intense inflammatory conditions. Hinton et al. [8] recorded hypoproteinemia and hypocalcemia in gastroduodenal ulcer dogs. Measurement of the serum concentration of unconjugated bile acid was considered as an index for bacterial activity in the small intestine [56]. Dossin and Henroteaux [12] reported that hypoproteinemia and hypoalbuminemia were observed in IBD. Craven et al. [57] recorded hypoalbuminemia, hypoproteinemia, increased serum folate, and decreased serum cobalamin concentration in canine inflammatory bowel disease. Potoenjak et al. [38] and Brooks [58] observed hypoproteinemia, hypocholesterolemia, and hypocalcemia in lymphangiectasia. Marks [59] recorded that decreased serum cobalamin and increased folate concentration in SIBO affected dogs. Johnston [60] stated that indirect diagnosis of SIBO was made by estimation of deconjugated bile acid, serum cobalamin, and folate concentrations. Rutgers et al. [15] reported serum folate and cobalamin were only present in 5% of dogs with duodenal juice culture proven SIBO. Hall [61] suggested that serum folate and cobalamin concentrations cannot be used to diagnose SIBO because of the poor sensitivity, although a low serum cobalamin still has value as an indication to treat. Allenspach et al. [62] reported hypoalbuminemia and hypoglobulinemia are the important indicators of protein-losing enteropathy in dogs, and in such

case serum albumin concentration should be measured because hypoalbuminemia has been shown to be a negative prognostic indicator in dogs with chronic enteropathies.

4.5 Fecal examination

Berghoff and Steiner [63] reported chronic signs of intestinal disease should be evaluated for endoparasitic infestation before detail diagnostic examination to rule out hookworms (*Ancylostoma* spp., *Uncinaria* spp.), roundworms (*Toxocara* spp.), and whipworms (*Trichuris vulpis*). Broussard [49] stated that hookworm and *Ascaris* were diagnosed by standard fecal flotation method. Sokolow et al. [64] reported that *Giardia* could be diagnosed by the combination of fecal examination and centrifugal flotation.

Cystic stage of *Giardia* was diagnosed with zinc sulfate centrifugal or simple flotation technique and trophozoite stage by direct smear and detection of trophozoites and cysts in feces with FAT technique [46, 55]. Direct microscopic examination of fresh fecal samples suspended in saline could detect *Giardia* trophozoite [40, 59]. Fecal direct immunofluorescence assays were considered to be a golden standard method of diagnosis of *Giardia* infection in dogs, with a reported sensitivity and specificity of more than 90% each [65, 66]. Qualitative enzyme immunoassays (e.g., ProSpecT Giardia Microplate assay) and a SNAP test (SNAP Giardia Test) are also used for *Giardia* infection [41].

4.6 Duodenoscopy

Donaldson et al. [67] and Lamb [68] stated that gastroduodenoscopy was an important tool for examination of a dog with chronic vomiting and diarrhea and also suggested that visualization of duodenum, aspiration of duodenal contents, and microscopic examination of mucosal samples could be useful in diagnosis. Simpson [69] stated that indications for duodenoscopy were small bowel diarrhea, protein-losing enteropathies, chronic vomiting, and melena. Zoran [3] and Willard [70] reported that gastroduodenoscopy was not only valuable in visualization of mucosal irregularities and obtaining biopsies, cytologic samples, or fluids from duodenum but also might reveal anatomical distortions or displacements occurring in those regions.

Hinton et al. [8] reported that endoscopic examination was the diagnostic tool for gastroduodenal perforation. Moore [71] stated that most of the foreign bodies lodged in proximal duodenum could be removed by using endoscopy. Boston et al. [72, 73] reported that the endoscopy was commonly used to evaluate gastroduodenal ulceration and erosions induced by nonsteroidal anti-inflammatory drugs and corticosteroids. Endoscopic examination of the upper gastrointestinal tract had been used increasingly as a diagnostic and therapeutic tool in small animal medicine, particularly in the diagnosis of chronic gastrointestinal diseases and in understanding their pathophysiology [74].

4.6.1 Endoscopic appearance of duodenum

Peyer's patches (lymph node) are noticed as flattened areas on the lateral aspect of ascending duodenum [48]. Richter [42] reported that the normal duodenum was pink, smooth, and uniform with velvet-like texture. Normal duodenum appeared slightly grainy or roughened because of the presence of mucosal villi and was reddish pink to yellow if bile was present [3]. In descending duodenum, the major duodenal papilla were noticed approximately 4–5 cm from pylorus on medial aspect

of duodenum, and minor duodenal papilla was located dorsal to the major papilla. Lymphoid follicle appeared as dished out area, often with white spots [70]. Bexfield [71] stated that the normal duodenum was more red in color than the stomach and had yellow tinge due to bile and also reported that the duodenal mucosa was more friable than that of the stomach and had a fine granular appearance due to the mass of intestinal villi.

4.6.2 Endoscopic appearance of duodenal disorders

Roth et al. [72, 73, 75] reported that increased granularity and friability associated with increased cellularity within the lamina propria were due to the inflammation or neoplasm of duodenum. Richter [42] described endoscopic appearance of duodenal ulcer as deep or shallow crater and well demarcated from the surrounding mucosa.

Forsyth et al. [76] described mucosal grading scale for ulceration study as follows: 0, no visible hemorrhage and erosions/ulcers; 1^+, 1–5 punctate erosions and hemorrhage; 2^+, 6–15 punctate erosions and hemorrhage; 3^+, 16–25 punctate erosions and hemorrhage; 4^+, ≥25 punctate erosions and hemorrhage and 1–5 invasive erosions; 5^+, ≥5 invasive erosions; and 6^+, ulcer of any size (**Figure 2**).

Yamasaki et al. [77] reported that endoscopic appearance of duodenum in lymphocytic-plasmacytic enteritis had mucosal friability and mucosal destruction and hemorrhage when the mucosa was gently touched with endoscope, which was not observed in normal duodenum of dog. Leib and Matz [43] reported that endoscopic appearance of duodenum in IBD-affected dogs was hyperemia, mucosal hemorrhage, ulceration, increased mucosal granularity, and friability. Lecoindre [78] observed a multifocal, pale, papular, and granular appearance of duodenum in lymphangiectasia in dogs. Miura et al. [79] and Snead [80] reported endoscopic appearance of duodenal neoplasia as ulceration, irregular mucosa, hyperemia, cobble stone or granular texture, and friability (**Figure 3**). Walker [81] recognized the diseased duodenum during duodenoscopy as reddened, ulcerated, thickened (**Figure 4**), and abnormally fibrosed or friable such that it bled abnormally following contact with the tip of the scope or after taking biopsy.

Freiche and Poncet [82] recorded increased granularity of mucosa and erythema of the proximal duodenum with highly visible Peyer's patches in brachycephalic breeds affected with upper airway and gastrointestinal syndrome. Sancho et al. [83] evaluated the endoscopic appearance of the descending duodenum in lymphocytic-plasmacytic enteritis and scored as follows: (1) intestinal lumen (0 = normal, 1 = narrowed due to

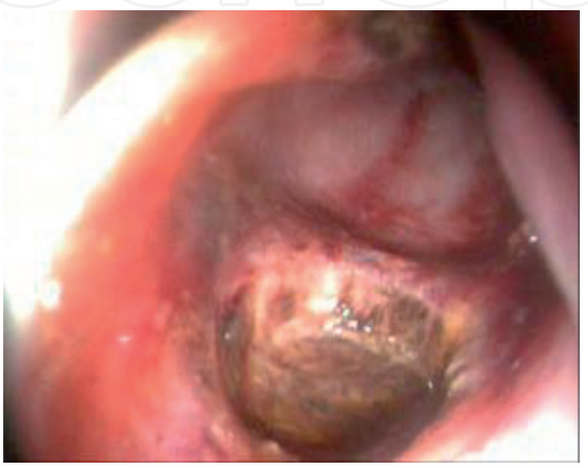

Figure 2.
Duodenoscopy: duodenal ulceration cum perforation.

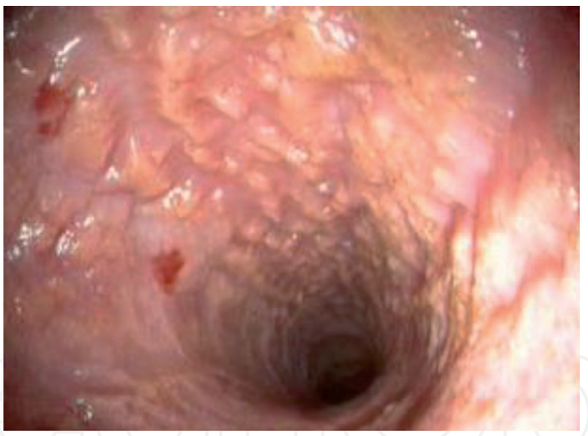

Figure 3.
Duodenoscopy: duodenal ulceration in plasmacytic enteritis.

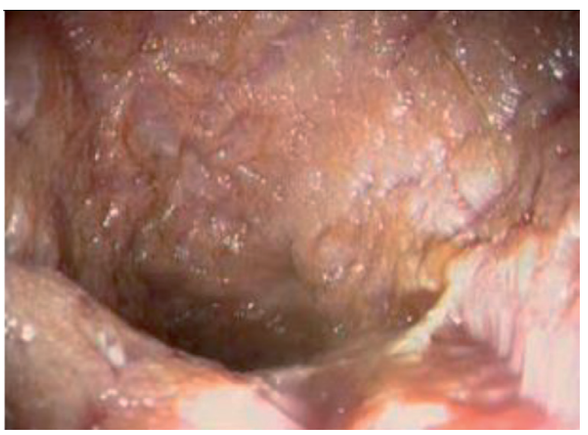

Figure 4.
Duodenoscopy: highly fragile duodenal mucosa in inflammatory bowel disease.

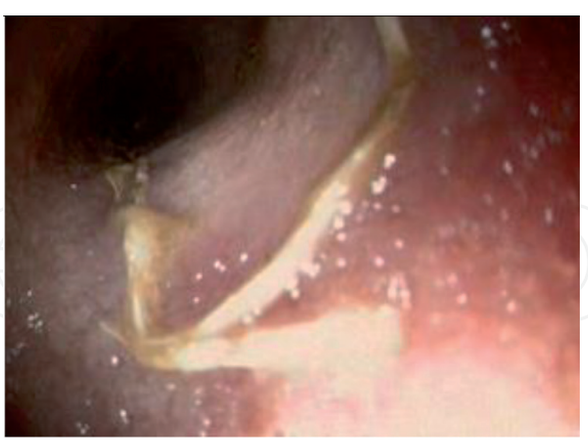

Figure 5.
Duodenoscopy: duodenal foreign body—thread in the proximal duodenum.

lack of elasticity), (2) mucosal erythema (0–3 depending on the degree of alteration), (3) mucosal granularity (0–3 depending on the degree of alteration), (4) irregular mucosa (0–3 depending on the degree of alteration), (5) friable mucosa (0–3 depending on the degree of alteration), and (6) erosions/ulcers (0–3 depending on the degree of alteration). And from this total score, the macroscopic and endoscopic duodenal lesions of the dogs were classified as normal duodenum (0 point), mild LPE (1–4, up to 25% of the maximum score), moderate LPE (5–8, up to 25–50% of the maximum score), and severe LPE (9–16, >50% of the maximum score).

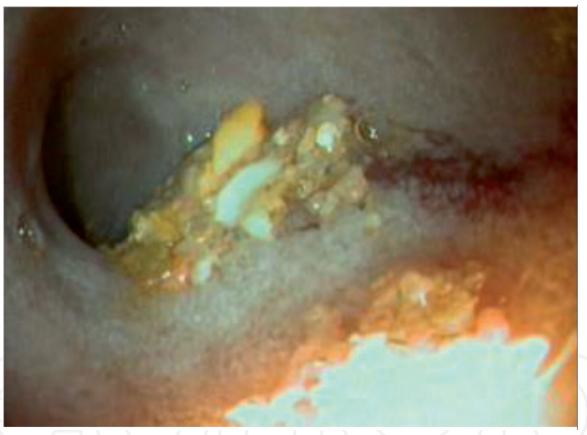

Figure 6.
Duodenoscopy: duodenal foreign body—stones and bone pieces in the proximal duodenum.

4.6.3 Endoscopy-aided biopsy of duodenum

Willard et al. [84] and Michael et al. [85] stated that the endoscopic biopsy of the duodenal mucosa had been an important tool in the diagnosis of small intestinal tract diseases of dogs. Obtaining excellent duodenal tissue samples was typically harder than obtaining samples from other portions of the gastrointestinal tract, and they also opined that it was easier to obtain high-quality tissue samples from the descending duodenum than ascending duodenum [86]. Jergens et al. [87] suggested that the multiple mucosal biopsy specimens were required for diagnosis of inflammatory bowel disease. Full-thickness biopsy of intestine was necessary to diagnose alimentary lymphoma, because lesions were usually deep seated and invaded the serosal layer [79].

The definitive diagnosis of IBD was obtained by histological examination of intestinal biopsies [12]. Walker [81] and Spillmann [88] reported that biopsy samples of ulcer condition should be obtained from the periphery of the lesion. Freiche and Poncet [82] observed lymphoplasmacytosis as a principle lesion from duodenal biopsies in brachycephalic dogs affected with upper airway and gastrointestinal syndrome. Bexfield [89] reported that biopsy could be performed by the use of flexible pinch biopsy forceps.

4.6.4 Endoscopy-aided brush cytology

Jergens et al. [90] and Zoran [3] observed that endoscopy-aided brush technique was most useful in detecting cellular infiltration in lamina propria and was considered as a reliable adjunct to histological examination of biopsy specimen in the diagnosis of GI diseases of dogs and cats.

4.6.5 Endoscopy-aided collection of duodenal juice

Pitts et al. [91], Roudebush and Delivorias [92], and Leib et al. [20] reported that aspirate of duodenal content revealed a *Giardia* trophozoite on microscopic examination of the sediments and it was more sensitive than zinc sulfate fecal flotation technique. Hall [55] stated that duodenal juice could be collected during duodenoscopy for diagnosis of SIBO.

4.7 Histopathology and brush cytology

Dow et al. [93] observed slightly blunted and thickened duodenal villi and increased infiltration of inflammatory cells in the dogs with duodenal ulcer (**Figure 7**). Willard

et al. [94] and Rutgers et al. [15] reported that histologic and intestinal mucosal cytologic examinations were not useful in detecting SIBO. Jergens et al. [52] and Yamasaki et al. [77] stated that duodenal mucosa in clinically normal dogs had low numbers of lymphocytes, histiocytes, plasma cells, and eosinophils. The severity of lymphangiectasia was graded by counting the number of dilated lacteals per low-power (10× objective) field. Ridgway et al. [53] observed increased mucosal granularity of duodenum, mucosal cellular infiltration, and architectural alteration in lymphocytic-plasmacytic enteritis (**Figure 8**).

Potoenjak et al. [38] reported histopathological appearances of lymphangiectasia, and there were dilated lacteals of small intestinal villi, lymphatics, focal infiltration of lamina propria with mononuclear cells particularly plasma cells, and mild edema of the lamina propria (**Figures 9**, **10**). Duodenal biopsy revealed severe fibrosis of the submucosa and infiltrate of small pockets and cards of round to polygonal cells with granular cytoplasm in duodenal gastrinomas [19].

Cytological examination of intestinal lymphosarcoma revealed large, atypical granular lymphocytes containing irregularly shaped, magenta cytoplasmic granules, and in histopathologic examination, diffuse infiltration of the lamina propria of duodenum by a population of large lymphocytes was observed [80]. Jergens [95], Sancho et al. [83], and Day et al. [96] suggested the scoring system

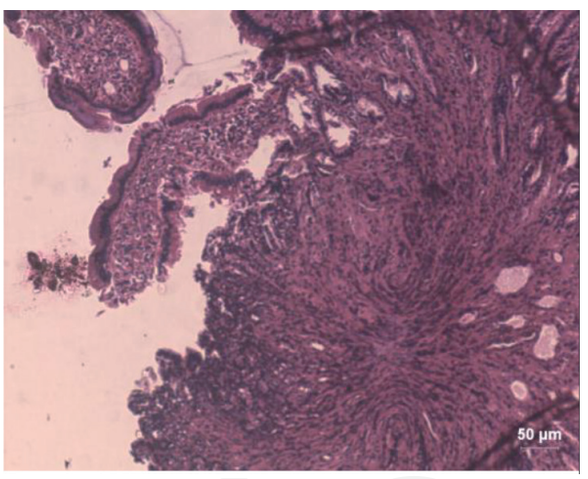

Figure 7.
Excessive loss of duodenal villus epithelium in duodenal ulcer (H&E 10 µm).

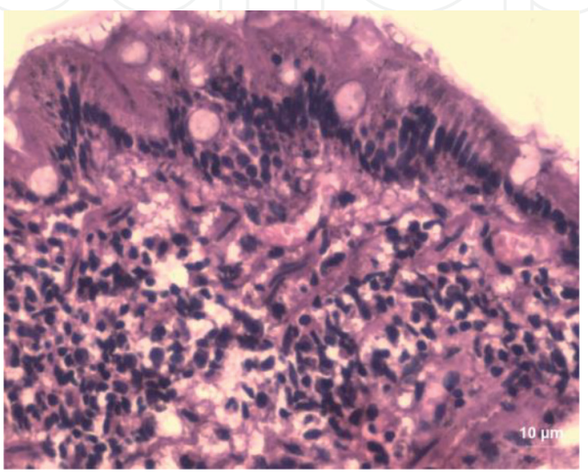

Figure 8.
Lymphoplasmacytic infiltration of lamina propria of duodenum in IBD (H&E 10 µm).

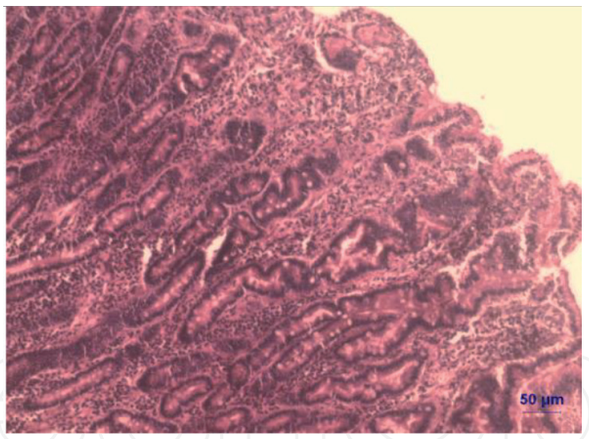

Figure 9.
Ulceration of villus epithelium with lymphoplasmacytic infiltration in IBD (H&E 50 µm).

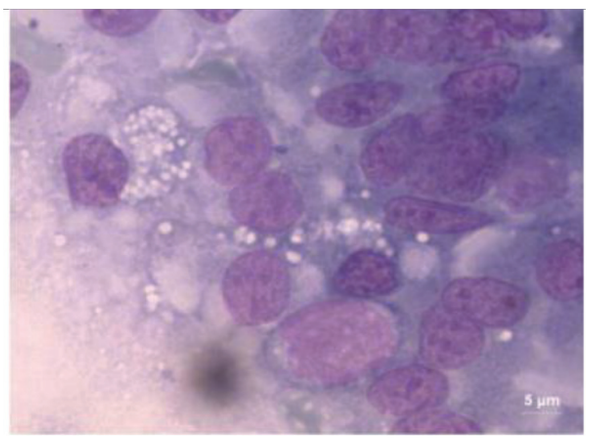

Figure 10.
Duodenal brush cytology—vacuolar degeneration of epithelium and MNC cells (5 µm).

for histopathology of duodenal biopsy samples as follows: intraepithelial lymphocytes, chronic inflammatory cell infiltration, edema, lymphocytic vessel dilation, glandular hyperplasia, and fibrosis scored between 0 and 3 according to the degree of alteration (0 = normal and 3 as the maximum alteration), and from the total score, the microscopic duodenal lesions of the dogs were classified as normal (0 point), mild (1–5, up to 25% of the maximum score), moderate (6–9, 25–50% of the maximum score), and severe (10–18, >50% of maximum score).

4.8 Duodenal juice culture

Small intestinal bacterial overgrowth was diagnosed by isolation of $\geq 10^5$ aerobic bacterial colony-forming units or $\geq 10^4$ anaerobic bacterial colony-forming units from duodenal/proximal jejunal fluid obtained from non-fed dogs [55]. Delles et al. [97] and Batt [16] reported that *Escherichia coli*, *Staphylococcus xylosus*, *Staphylococcus epidermidis*, *Corynebacterium* sp., and *Proteus mirabilis* were the most commonly isolated bacteria from intestinal fluid.

Willard et al. [94], Rutgers et al. [15], and Lecoindre [78] reported that small intestinal bacterial overgrowth was diagnosed by quantitative bacterial culture of duodenal juice samples obtained endoscopically. Johnston [60], German et al. [98], and Marks [59] opined that culture of duodenal juice had been regarded as the gold standard for detecting elevated bacterial counts in SIBO. German et al. [99] stated that in SIBO of German Shepherd dog, the duodenal juice had reduced IgA concentration.

4.9 Ultrasound

Gastrointestinal ultrasonography has become a supporting noninvasive diagnostic tool for intestinal diseases and disorders in dogs. It is used to differentiate inflammation and neoplastic infiltrative disease of the intestine [100, 101]. Ultrasonography can be used to inspect the small intestinal wall thickness, dilatation, and peristalsis as well as for intraluminal, intramural, and extraluminal causes of obstruction [102]. Abdominal ultrasonography has become in many instances a part of the minimum database in conjunction with abdominal radiography for the assessment of intestinal disease [103]. The most common ultrasonographic features of intestinal abnormalities are thickening of the bowel wall, loss of its normal layer, and alterations in the contour of the mucosal and/or serosal surfaces [104]. Thickness, altered wall layering appears to be an important finding which differentiates inflammatory from neoplastic and granulomatous infiltration [105]. In dogs, ultrasonographic examination of the small intestine has been successfully used to identify gastrointestinal tumors [104]. Rudorf et al. [100] concluded in his study that ultrasonography should be used to rule out diseases such as intussusceptions and localized tumors in cases of chronic diarrhea.

Ultrasonographic changes were similar in lymphoplasmacytic enteritis and lymphoma of the small intestine, since the overlapping USG findings of inflammatory and neoplastic disease conditions of the intestinal disorders were only confirmed by histopathology examination, which is necessary for differentiating the diseases [106].

Smith et al. [107] reported that hyperechoic mucosal striations are associated with lacteal dilation and are frequently associated with mucosal inflammation and protein-losing enteropathy in dogs.

Gaschen et al. [101] concluded in his study that intestinal mucosal echogenicity could be used for detecting inflammatory bowel disease than bowel wall thickness with chronic diarrhea in dogs.

4.10 Radiographic examination

Gaschen [106] reported abdominal survey radiographic examination remains an important part of screening patients with vomiting and diarrhea, and it should be performed in conjunction with the abdominal ultrasonographic examination instances. Radiography of intestine could identify foreign bodies, nails, mass/neoplasia, and obstructions. Contrast radiography can be used as an adjunct in the investigation of GI disease. Barium studies can provide information on GI transit, obstructive lesions, intestinal mass lesions, or disrupted mucosal integrity.

5. Treatment

Therapeutic protocols for the gastrointestinal disorders are based on the acute or chronicity of the condition. Mostly duodenal disorders are observed with chronic condition because of the non-specific signs. Hence, therapy initiated with symptomatically and appropriated therapeutic regimen is followed after confirming the disease or disorders which are aided by duodenoscopy, histopathology, and bacteriology studies. Herewith some of the commonly using drugs for the various duodenal disorders are as follows.

Name of the drug	Dose	Condition	Reference
Azathioprine	Initially 2 mg/kg q24 h for 3 weeks and then 1–2 mg/kg q48 h up to 3 weeks	IBD	[108]
Misoprostol	2–5 μg/kg PO q8 h	GI protectants and cytoprotective agents	[109]
Fenbendazole	50 mg/kg PO q24 h for 3 days	Giardiasis	[110]
Pyrantel pamoate	5–10 mg/kg PO	*Toxocara* and *Ancylostoma* infection	[110]
Loperamide	0.1–0.2 mg/kg PO q8–12 h	Motility-modifying agents	[110]
Bismuth subsalicylate	1 ml/kg/day PO divided q8–12 h for 1–2 days	Anti-secretory drug	[109]
Metoclopramide	0.25–0.5 mg/kg PO, IM, or IV q8–24h, 1-2 mg/kg/day, constant IV infusion	Antiemetics	[109]
Ondansetron	0.1–0.2 mg/kg IV q8–24 h	Antiemetics	[109]
Prednisolone	1 mg/kg orally twice daily for 10 days, then 0.5 mg/kg orally twice daily for 10 days, then 0.5 mg/kg orally once daily for 10 days, and then 0.5 mg/kg orally every other day for 10 days	IBD	[111]
Metronidazole	15–25 mg/kg, PO, q12–24 h, for 5–7 days	Giardiasis	[112]
	10 mg/kg given orally every 8–12 h	SIBO/ARD	[61]
Oxytetracycline	10–20 mg/kg given orally every 8 h	SIBO/ARD	[61]
Omeprazole	0.7–2 mg/kg PO q12–24 h	Antacid/GI ulceration	[110]
Pantoprazole	1 mg/kg IV q24 h	Antacid/GI ulceration	[110]
Sucralfate	0.5–1 g per dog	GI protectants and cytoprotective agents	[109]
Ranitidine	1–2 mg/kg, PO or IV, q8–12 h	Antacid/GI ulceration	[110]
Tylosin	20–40 mg/kg PO q12 h	SIBO and ARD	[61]
Tinidazole	44 mg/kg, PO, q24 h for 6 days	Giardiasis	[112]

5.1 Dietary management

- Marks suggested feeding management is necessary for the management of most chronic small bowel disease of pets. Dogs with small bowel diarrhea can be managed by highly digestible, moderately fat-restricted, lactose-free, and gluten-free dietary supplement [111, 113].

- Milk- and lactose-contained diet should be avoided during the diarrhea, because during the diarrhea intestinal mucosal erosion lead reduction in the brush border enzyme especially lactose. Therefore fail to digest lactose leads bacterial degradation of sugar to VFA which causes osmotic diarrhea.

- Hypoallergenic diet refers to a diet that is generally free of additives and preservatives and contains a single, novel protein source that is highly digestible. Hypoallergenic diet is very much useful for the management canine IBD along with immunosuppressive therapy.

6. Conclusions

Duodenal disorders are the one among major gastrointestinal disturbances in dogs and it also interrelated to the other GI disturbances. Diagnosis of these disorders is a challenging one, due to their non-specific signs and hence, contrasts radiography and endoscopy which play a vital role in the diagnosis. Duodenoscopy is an efficient way of identifying the abnormalities of mucosal irregularities, distortion, ulceration, neoplasia, and inflammation of intestines, and it is very much useful for obtaining mucosal biopsy and duodenal juice for histopathological and bacteriological studies.

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Conflict of interest

No conflict of interest.

Author details

Mani Saravanan
Veterinary Clinical Complex, Veterinary College and Research Institute, Tamilnadu Veterinary and Animal Sciences University (TANUVAS), Thanjavur, India

*Address all correspondence to: sara82vet@yahoo.com

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