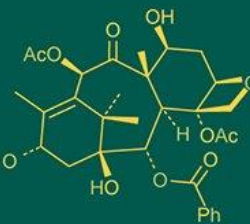
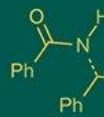
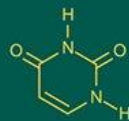


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Inflammatory bowel disease in dogs: An overview

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Abstract

Inflammatory bowel disease (IBD) is a chronic, relapsing inflammatory disorder of the canine gastrointestinal tract characterized by persistent vomiting, diarrhoea, weight loss and histological infiltration of inflammatory cells within the intestinal mucosa. The disease has a multifactorial etiology involving genetic susceptibility, immune dysregulation, intestinal dysbiosis, dietary factors and environmental influences. Diagnosis requires exclusion of other gastrointestinal disorders, assessment of clinical activity using indices such as CIBDAI and CCECAI, laboratory investigations, imaging, endoscopy and histopathological examination. Emerging non-invasive biomarkers, including perinuclear antineutrophil cytoplasmic antibodies (pANCA) and microRNAs, have shown promise for improving diagnostic accuracy and monitoring disease progression. Therapeutic management focuses on dietary modification using hydrolysed or novel-protein diets, immunosuppressive therapy, antibiotics when indicated and correction of nutritional deficiencies. Long-term disease control depends on individualized treatment and regular monitoring. Advances in understanding disease pathogenesis and biomarker development may enhance early diagnosis, therapeutic response assessment and overall clinical outcomes in canine inflammatory bowel disease.

Keywords: Canine inflammatory bowel disease (IBD), Intestinal dysbiosis, pANCA, MicroRNAs, Histopathology

Introduction

Inflammatory bowel disease (IBD) is a persistent and recurring condition affecting the gastrointestinal tract, distinguished by mucosal inflammation, notable for recurring instances of diarrhoea and vomiting and considered as the most common cause of chronic intestinal disease in dogs. Inflammatory bowel disease is also referred to as idiopathic, as it lacks a known underlying cause for intestinal inflammation. The various types of IBD in dogs primarily vary based on the types of cells found in the intestinal mucosa, including lymphocytes, plasma cells, eosinophils, macrophages, neutrophils or combinations of these cells. The pathogenesis of IBD involves a multifaceted interplay of genetic predisposition, environmental influences, immune response and dietary elements. An imbalance within the intestinal microbiota is identified as a major contributing factor in the development of IBD in dogs through the breakdown of immune tolerance to bacterial antigens within the intestinal lumen. This breakdown occurs due to disruption in the mucosal barrier, dysregulation of gut-associated lymphoid tissue, disturbance in the microbial flora or a combination of these factors. The development of early, non-invasive diagnostic methods, such as the use of various inflammatory biomarkers, plays an important role in the diagnosis of IBD. Among serum biomarkers, the detection of perinuclear antineutrophilic cytoplasmic antibodies (pANCAs) in dogs can be used as a helpful diagnostic tool to differentiate between dogs with IBD and those with other chronic gastrointestinal conditions. These are autoantibodies surrounding the granulocyte nucleus with a staining pattern and the primary target of these antibodies is myeloperoxidase, although they also attack other antigens like lactoferrin and elastase. Also, the evaluation of the expression of microRNAs (miRs) in the serum and colon tissue will offer a potential link between genetic susceptibility, environmental and immunological factors involved in the pathogenesis of IBD and may serve as a clinically useful biomarker for the diagnosis and treatment of canine IBD.

Inflammatory bowel disease comprises a group of chronic intestinal disorders characterized by infiltration of inflammatory cells into the intestinal mucosal lamina propria (Nelson *et al.*, 1988) [53]. In dogs, with chronic inflammatory enteropathies, approximately 50-66% exhibit

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food-responsive enteropathy, while 15-20% each have antibiotic-responsive enteropathy or idiopathic IBD (Allenspach *et al.*, 2016). Canine IBD is characterized by persistent or recurrent gastrointestinal signs such as anorexia, weight loss, vomiting and chronic diarrhoea, with histological evidence of intestinal inflammation. Diagnosis is based on compatible clinical signs, exclusion of other gastrointestinal and systemic diseases, failure to respond to dietary and antibiotic therapy, histopathological confirmation of chronic intestinal inflammation and response to immunosuppressive treatment (Lee *et al.*, 2021). Although its exact etiology remains unclear, IBD is thought to result from interactions among genetic susceptibility, immune dysregulation, dietary factors, environmental triggers, and intestinal microbiota (Simpson and Jergens, 2011). The most common forms are lymphocytic-plasmacytic and eosinophilic enteritis (Rousseau, 2005). Management focuses on long-term control through dietary modification, anti-inflammatory and immunosuppressive drugs, antibiotics, symptomatic therapy, and nutritional support when required (German *et al.*, 2003). Several breeds, including Basenjis, Soft-Coated Wheaten Terriers, Boxers, German Shepherds, Weimaraners, Rottweilers, Border Collies, French Bulldogs, Yorkshire Terriers, and Norwegian Lundehunds, show increased susceptibility to IBD, with genetic associations involving TLR4, TLR5, and NCF2 polymorphisms supporting a hereditary predisposition (Peiravan *et al.*, 2018 and Felix *et al.*, 2022). Canine IBD is primarily a disease of adult and middle-aged dogs, although cases have been reported in puppies as young as six months and in dogs only a few months old (Yogeshpriya *et al.*, 2017; Allenspach and Mochel, 2020). The highest prevalence has been observed in dogs between 5 and 10 years of age, with granulomatous colitis occurring predominantly in Boxer dogs younger than four years (Craven *et al.*, 2010; Hemida *et al.*, 2021). While some studies reported a higher incidence in male dogs, others found no significant sex predisposition, suggesting that gender may have a limited role in disease development (Calalang *et al.*, 2021; Hemida *et al.*, 2021). The etiology and pathogenesis of canine IBD are multifactorial, involving complex interactions among genetic susceptibility, dietary factors, intestinal microbiota, environmental influences and dysregulated immune responses (Hall and Batt, 1992; Sandborn and Yednock, 2003; Xavier and Podolsky, 2007) [3, 45, 64, 59, 28, 57, 24, 75, 2, 17, 34, 12, 34, 32, 63, 74]. Genetic predisposition has been demonstrated in breeds such as German Shepherds, Boxers and Rottweilers, with polymorphisms in genes including NOD2 and increased intestinal expression of TLR2 being associated with disease susceptibility and severity (Parker *et al.*, 2004; Glas *et al.*, 2010; Allenspach, 2011). Chronic intestinal inflammation is characterized by excessive infiltration of lymphocytes and plasma cells within the intestinal mucosa, and immune-mediated mechanisms are further supported by the response to immunomodulatory therapy and increased IgE-positive cells in affected dogs (Rychlik *et al.*, 2007; Cerquetella *et al.*, 2010). Infectious agents and adverse dietary reactions have also been proposed as contributing factors in disease pathogenesis (German *et al.*, 2003; Hall and Batt, 1992).

The pathogenesis of canine IBD is multifactorial, involving complex interactions among genetic susceptibility, intestinal microbiota, dietary factors and dysregulated immune responses (Simpson and Jergens, 2011; Jergens and Simpson, 2012). Genetic predisposition has been demonstrated by polymorphisms in innate immune genes, including complete linkage disequilibrium in German Shepherd dogs with IBD, as well as mutations affecting NOD2 and other toll-like receptors that impair mucosal immune function (Kathrani *et al.*, 2011a; Simpson and Jergens, 2011). Loss of tolerance to commensal microbiota, dietary antigens and endogenous antigens, resulting in inappropriate immune activation, is considered a central mechanism in disease development (Malewska *et al.*, 2011; Rychlik *et al.*, 2012). Additional contributing factors include alterations in gut microbial composition, mucus production, intestinal permeability, mucosal barrier regeneration, cellular autophagy and immune regulation (Maeda *et al.*, 2013). Dietary factors, particularly refined carbohydrate-rich diets, may promote dysbiosis, increased intestinal permeability and inflammation, thereby predisposing dogs to IBD (Martinez-Medina *et al.*, 2014). Intestinal dysbiosis, characterized by reduced microbial diversity, depletion of Gram-positive bacteria and expansion of Gram-negative populations, has been consistently associated with IBD and linked to aberrant expression of pattern-recognition receptors such as TLR4 and TLR5 in German Shepherd dogs (DeGruttola *et al.*, 2016). Clinical severity and therapeutic response in canine IBD are commonly assessed using the Canine Inflammatory Bowel Disease Activity Index (CIBDAI) and the Canine Chronic Enteropathy Clinical Activity Index (CCECAI) (Jergens *et al.*, 2003; Allenspach *et al.*, 2007). Both scoring systems classify disease severity as clinically insignificant (0-3), mild (4-5), moderate (6-8) and severe (≥ 9), and have been widely used to evaluate disease activity, treatment outcomes and intestinal inflammation in affected dogs (Allenspach *et al.*, 2007; Jergens and Simpson, 2012). The CIBDAI is considered a simple and reliable tool that incorporates major gastrointestinal signs and allows monitoring of disease progression and response to therapy (Rychlik *et al.*, 2012; Lee *et al.*, 2021). Canine IBD typically presents as chronic enteropathy with gastrointestinal signs persisting for at least three weeks, including vomiting, diarrhoea, abdominal pain, weight loss, altered appetite and changes in stool frequency and consistency (Dandrieux, 2016; Garraway *et al.*, 2018; Lee *et al.*, 2021). Clinical manifestations vary according to the affected gastrointestinal segment, with severe cases showing haematemesis, melena, haematochezia, anorexia and discomfort on rectal examination, particularly in eosinophilic inflammation and colitis (Wdowiak *et al.*, 2014). Some dogs may also develop protein-losing enteropathy resulting in hypoalbuminaemia, subcutaneous oedema, pleural effusion and ascites, while infiltrative intestinal diseases such as IBD are often associated with generalized gastrointestinal hypomotility (Garraway *et al.*, 2018; Jergens, 2020). CIBDAI and CCECAI therefore serve as valuable tools for the initial assessment and monitoring of disease severity in canine IBD (Bhavani *et al.*, 2021) [64, 38, 42, 64, 49, 62, 48, 52, 20, 40, 5, 5, 38, 62, 45, 19, 26, 45, 71, 26, 37, 10].

The diagnosis of canine idiopathic IBD is based on persistent gastrointestinal signs, exclusion of other gastrointestinal and systemic disorders, failure to respond to dietary, antibiotic

and antiparasitic therapies, histopathological confirmation of intestinal inflammation and clinical improvement following anti-inflammatory or immunosuppressive treatment (Willard *et al.*, 2002; Maeda *et al.*, 2013; Lee *et al.*, 2021). A systematic diagnostic approach includes elimination diets, antimicrobial trials, faecal examination to exclude parasitic infections and histological evaluation of gastrointestinal biopsies to confirm inflammation and differentiate IBD from alimentary lymphoma (Jergens and Simpson, 2012; Lee *et al.*, 2021). Clinicopathological abnormalities commonly observed in affected dogs include eosinophilia, anaemia, leucocytosis, thrombocytopenia, hypoproteinaemia, hypoalbuminaemia, hypoglobulinaemia, hypocholesterolaemia and increased serum activities of alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase (Craven *et al.*, 2004; Wdowiak *et al.*, 2014; Hotel and Tams, 2016). Reduced serum folate and cobalamin concentrations, indicative of impaired intestinal absorption, are also frequently reported (Feldman *et al.*, 2000). Hypoproteinaemia may result from reduced feed intake, malabsorption associated with villous atrophy or fibrosis, intestinal protein loss and increased intestinal permeability (Cabral *et al.*, 2001). These clinical, laboratory and histopathological findings collectively support the diagnosis and assessment of canine IBD. In canine IBD, haematological and biochemical alterations may reflect dehydration, inflammation, malabsorption and intestinal dysbiosis. Increased packed cell volume can occur secondary to severe dehydration caused by vomiting and diarrhoea (Bhargavi *et al.*, 2017), while affected dogs commonly exhibit anaemia, reduced haemoglobin and haematocrit values, and increased neutrophil, red blood cell and total white blood cell counts (El-Zahar *et al.*, 2021). Faecal cytology, including dry mount examination, may aid diagnosis by identifying inflammatory cell populations, with plasma cells being the predominant cytological finding in canine IBD (Frezoulis *et al.*, 2017; Benvenuti *et al.*, 2020). Hypocobalaminaemia is frequently reported in dogs with IBD and is attributed to intestinal dysbiosis, impaired absorption and reduced intrinsic factor availability (Heilmann *et al.*, 2018; Glanemann *et al.*, 2021; Hung *et al.*, 2023). Altered serum cobalamin and folate concentrations provide useful adjunctive diagnostic information, reflecting malabsorption, dysbiosis and the extent of intestinal damage, and may contribute to the development of macrocytic anaemia in affected dogs (El-Zahar *et al.*, 2021; Ullal *et al.*, 2023) [73, 48, 45, 38, 18, 71, 35, 23, 11, 8, 21, 25, 7, 33, 30, 36, 68].

Radiography is commonly employed in the evaluation of dogs with gastrointestinal disease and may reveal intestinal wall thickening, irregular mucosal patterns, gas accumulation or in severe cases, barium contrast abnormalities such as thumb-printing, although contrast studies are preferred for accurate assessment of bowel wall thickness (Raji, 2010; Allenspach, 2013; Hotel and Tams, 2016; Stieger-Vanegas, 2020). Additionally, radiographic examination is valuable in excluding differential diagnoses such as foreign bodies, neoplasia and structural gastrointestinal abnormalities that may mimic IBD (El-Zahar *et al.*, 2021). Ultrasonography is a valuable non-invasive imaging modality in the evaluation of canine IBD, allowing assessment of intestinal wall thickness,

layering, echogenicity, motility and regional lymph nodes (Linta *et al.*, 2021). A normal intestinal wall consists of distinct echogenic and hypoechoic layers, whereas inflammatory lesions may present with diffuse wall thickening, altered echogenicity and enlargement of intestinal layers, with intestinal wall thickening being considered one of the most important ultrasonographic indicators of inflammation (Spohr *et al.*, 1995). Administration of polyethylene glycol before ultrasonography improves intestinal distension and wall visualization, providing diagnostic efficacy comparable to contrast radiography for detecting intestinal lesions (Parente *et al.*, 2004). Ultrasonography is superior to radiography for evaluating intestinal wall thickness, mesenteric lymphadenopathy and the distribution of intestinal lesions, while also helping to exclude conditions such as intussusception, foreign bodies, obstructions, fungal lesions, localized tumours and other causes of chronic gastrointestinal disease (Ruau *et al.*, 2005; Bhavani *et al.*, 2021). However, ultrasonographic findings are not specific for IBD, as alterations in wall thickness, echogenicity and layering may occur in other intestinal disorders, particularly neoplasia, where abnormal wall layering is more frequently observed (Wdowiak *et al.*, 2014). Furthermore, some dogs with IBD may show normal ultrasonographic findings, and intestinal wall thickness alone lacks sufficient sensitivity and specificity for definitive diagnosis (Collins-Webb *et al.*, 2023). Nevertheless, ultrasonography remains an important diagnostic tool for differentiating inflammatory from neoplastic intestinal disease and guiding clinical management (Bhavani *et al.*, 2023) [35, 21, 46, 66, 55, 60, 10, 71, 16, 9, 39, 65, 43].

Colonoscopy and histopathology are important diagnostic tools for canine IBD. Endoscopic findings commonly include mucosal erythema, friability, increased granularity, erosions, ulcerations, white speckling and stenosis, reflecting varying degrees of intestinal mucosal inflammation (Jergens *et al.*, 2010b). Colonoscopy is more sensitive than ultrasonography for diagnosing colitis and assists in identifying the affected intestinal segments, assessing inflammatory activity and differentiating IBD from other causes of chronic enteropathy, although it has limited ability to evaluate deeper intestinal layers and colonic function (Bhavani *et al.*, 2023). Endoscopic lesions may be quantitatively assessed using scoring systems based on granularity, friability and erosions (Slovak *et al.*, 2015). Histopathological examination remains the gold standard for confirming IBD and typically reveals mucosal inflammation with villous blunting, epithelial injury, crypt hyperplasia or dilation, lacteal dilation, mucosal fibrosis and disruption of mucosal architecture (Kleinschmidt *et al.*, 2006). The inflammatory infiltrate is predominantly composed of lymphocytes and plasma cells, with occasional eosinophils, neutrophils and macrophages, supporting the classification of lymphocytic-plasmacytic enteritis as the most common histological form of canine IBD (Bhavani *et al.*, 2023). Histological severity is graded based on both inflammatory and morphological changes, ranging from mild to severe lesions, and provides definitive evidence of intestinal inflammation, complementing clinical indices such as the CIBDAI in assessing disease severity and progression (Bhavani *et al.*, 2023). Histopathological evaluation of endoscopic biopsy samples may additionally reveal epithelial

degeneration, crypt epithelial hyperplasia and mononuclear cell infiltration in the lamina propria, particularly in lymphocytic-plasmacytic colitis (Priya *et al.*, 2023).

The diagnosis of canine IBD can also be supported by non-invasive diagnostic methods, particularly the assessment of inflammatory biomarkers such as perinuclear anti-neutrophil cytoplasmic antibodies (pANCA) and microRNAs. Perinuclear antineutrophil cytoplasmic antibodies have been investigated as useful biomarkers in canine inflammatory bowel disease, with reported positivity ranging from 23% to 51% of affected dogs, indicating moderate diagnostic sensitivity (Allenspach *et al.*, 2004; Luckschander *et al.*, 2006). pANCA testing assists in differentiating IBD from other gastrointestinal disorders with similar clinical manifestations and provides evidence supporting an autoimmune component in disease pathogenesis. These autoantibodies, typically detected by indirect fluorescent antibody testing (IFAT), produce a characteristic perinuclear staining pattern and primarily target myeloperoxidase, although reactivity against other neutrophil antigens such as lactoferrin, elastase and pathogen-associated molecular patterns has also been reported (Weiner and Segelmark, 2016). In dogs with IBD, increased pANCA concentrations are believed to reflect ongoing intestinal inflammation and immune dysregulation, potentially triggered by dysbiosis and microbial antigens. Furthermore, serial measurement of pANCA levels may aid in monitoring disease progression and therapeutic response, with declining titres indicating clinical improvement and increasing levels suggesting disease exacerbation (Chen *et al.*, 2020). MicroRNAs (miRs) are small non-coding RNAs that regulate gene expression post-transcriptionally and play important roles in cellular proliferation, differentiation, immune regulation and disease pathogenesis (O'Brien *et al.*, 2018). Owing to their stability in exosomes, microvesicles, lipoproteins and RNA-protein complexes, miRs can be detected in tissues and a wide range of biological fluids including serum, plasma, urine, saliva and faeces, making them attractive diagnostic and prognostic biomarkers (Varvil and Dos Santos, 2023). Their expression is commonly quantified using reverse transcription real-time PCR, with approaches such as the S-Poly (T) primer method providing improved sensitivity and specificity and U6 small nuclear RNA frequently used as an endogenous reference gene (Konstantinidis *et al.*, 2020). Altered miR expression has been associated with inflammatory, autoimmune and other pathological conditions, including canine IBD. In dogs with IBD, significant upregulation of miR-16, miR-21, miR-122 and miR-147, along with downregulation of miR-185, miR-192 and miR-223, has been reported in colonic mucosa compared with healthy controls (Konstantinidis *et al.*, 2020). Increased expression of miR-16 and miR-147 has also been demonstrated in the colonic mucosa and serum of dogs with large intestinal IBD, suggesting their potential utility as non-invasive biomarkers for disease diagnosis and monitoring (Stiegeler *et al.*, 2021) [58, 4, 47, 72, 14, 54, 69, 44, 67].

The primary goals of canine inflammatory bowel disease treatment are to correct nutritional deficiencies, reduce intestinal inflammation, control dysbiosis and eliminate potential antigenic stimuli from the diet and intestinal microbiota (Marchesi *et al.*, 2017). Dietary management

forms the cornerstone of therapy, with hydrolysed protein or novel-protein diets proving effective, particularly in mild cases (Mandigers *et al.*, 2010). Dogs with moderate to severe IBD are commonly treated with a combination of dietary modification, prednisolone and metronidazole, whereas mild cases may respond to dietary therapy alone (Mandigers *et al.*, 2010). Corticosteroids remain the principal immunosuppressive agents, with prednisolone administered initially at immunosuppressive doses and gradually tapered to maintenance levels; additional agents such as azathioprine or chlorambucil may be used in refractory cases. Antibiotics, particularly metronidazole, may provide both antimicrobial and anti-inflammatory benefits, while prebiotics and probiotics can aid in modulating intestinal microflora (Marchesi *et al.*, 2017). Assessment of serum cobalamin and folate concentrations is recommended, and supplementation should be provided when deficiencies are identified, especially in dogs with chronic gastrointestinal disease (Kather *et al.*, 2020). In cases of large bowel inflammation, sulfasalazine may be employed as a therapeutic option because its active metabolite, mesalamine, is activated by colonic bacteria (Walker and McMahon, 2021). Treatment duration varies according to disease severity, and although long-term remission can often be achieved, complete cure is uncommon (Marchesi *et al.*, 2017). Dietary management is a fundamental component of canine inflammatory bowel disease therapy and is often the first-line treatment, particularly in mild cases (Gaschen and Merchant, 2011). An ideal diet for dogs with IBD should be nutritionally balanced, highly palatable and digestible, containing a novel intact or hydrolysed protein source, digestible carbohydrates, and low levels of gluten, lactose and fat. Novel-protein diets utilize a single protein source not previously consumed by the dog, whereas hydrolysed-protein diets contain proteins enzymatically broken down into small peptides that are less likely to trigger immune responses (Collier *et al.*, 2022). Dietary trials should be conducted exclusively for 8-12 weeks without introducing other food items to accurately assess response (Gaschen and Merchant, 2011). Mild cases of IBD may achieve remission through dietary management alone, whereas moderate to severe cases often require a combination of dietary modification and pharmacological therapy (Marchesi *et al.*, 2017). Evidence suggests that combining dietary intervention with medical treatment reduces disease relapse rates more effectively than pharmacotherapy alone, with hydrolysed-protein diets showing greater efficacy than novel-protein diets in preventing recurrence (Ambrosini *et al.*, 2020). Common novel protein sources used in hypoallergenic diets include white fish, venison, rabbit, duck, salmon, catfish and lamb (Collier *et al.*, 2022) [51, 50, 41, 70, 27, 15, 6].

Conclusion

Canine inflammatory bowel disease is a chronic, multifactorial enteropathy resulting from complex interactions among genetic predisposition, immune dysregulation, intestinal microbiota, dietary factors and environmental influences. The disease is characterized by persistent gastrointestinal signs and requires a systematic diagnostic approach involving clinical assessment, laboratory

investigations, imaging, endoscopy and histopathology. Although histopathological examination remains the gold standard for diagnosis, emerging non-invasive biomarkers such as pANCA and microRNAs show promise for improving early detection and disease monitoring. Effective management relies on individualized dietary modification, immunosuppressive therapy, control of dysbiosis and long-term monitoring to achieve sustained clinical remission and improve quality of life.

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