

Diet to be followed by Inflammatory Bowel Disease Patients

Mingzhu Zara^{*}

Department of Nephrology, University of Toronto, Toronto, Canada

DESCRIPTION

Inflammatory Bowel Disease (IBD) is divided into two primary categories; crohn's disease and Ulcerative Colitis (UC). A group of inflammatory conditions affecting the colon and small intestine make up IBD. Crohn's disease affects the mouth, oesophagus, stomach, and anus in addition to the small and large intestines, whereas ulcerative colitis mostly affects the colon and rectum. IBD can also affect dogs, and it is believed that the immune system, intestinal milieu, host genetics, and environmental factors interact to cause the condition. Humans with IBD who frequently require immunosuppressive therapy, many dogs respond to dietary changes alone. When dietary adjustments are insufficient, some dogs may additionally require therapy with immunosuppressant's or antibiotics. Intestinal biopsies are frequently carried out to determine what type of inflammation is present after other illnesses that can cause vomiting, diarrhoea, and abdominal pain in dogs have been ruled out (lymphoplasmacytic, eosinophilic, or granulomatous). Low blood cobalamin levels in dogs have been demonstrated to be a risk factor for unfavourable results [1].

IBD is primarily caused by loss of intestinal epithelial integrity. IBD colitis and related malignancy are influenced by innate immune system dysfunction brought on by aberrant signaling through immune receptors called Toll Like Receptors (TLRs), which activate an immune response to chemicals that are widely shared by numerous pathogens. Changes in the gut microbiota's makeup are a significant environmental element in the emergence of IBD. Inappropriate (uncontrolled) immune responses brought on by harmful changes in the intestinal microbiota cause injury to the intestinal epithelium. Breaks in this vital barrier, the intestinal epithelium, allow the microbiota to spread and trigger additional immunological reactions. Despite being a multifaceted disease, IBD is nevertheless partially fueled by an overactive immune response to the gut microbiota, which results in abnormalities in epithelial barrier function [2].

Numerous studies suggest that Deoxyribonucleic Acid (DNA) damage and oxidative stress are likely involved in the pathogenesis of IBD. Patients with IBD compared to control patients had considerably higher levels of oxidative DNA damage as determined by 8-OHdG levels, as patients with inflamed mucosa compared to non-inflamed mucosa [3].

Signs and symptoms

Despite the fact that crohn's and UC are quite different diseases, both can manifest with any of the following signs and symptoms: abdominal discomfort, diarrhoea, rectal bleeding, extreme internal cramps or muscle spasms in the pelvic region, and weight loss. Inflammatory bowel disease's most common extra intestinal consequence is anaemia.

Arthritis, pyoderma gangrenosum, primary sclerosing cholangitis, and non-thyroidal sickness syndrome are some of the associated symptoms or disorders. Associations with Bronchiolitis Obliterans Organizing Pneumonia (BOOP) and Deep Vein Thrombosis (DVT) have also been noted. The evaluation of inflammatory markers in faeces is typically followed by a colonoscopy and a biopsy of any abnormal lesions [4].

Causes

IBD is a complicated illness that develops when environmental and genetic variables interact, causing inflammatory reactions and immune responses in the intestines.

Diet

Food is a topic that IBD patients are particularly interested in, yet little is known about how diet affects the people. The best evidence-based diets for patients should include monitoring for the objective resolution of inflammation. Patients are encouraged to follow these diets.

According to a 2022 study, diets with higher intakes of fruits and vegetables, fewer processed meats and refined carbohydrates, and a preference for water over other liquids for hydration were linked to a lower risk of IBD active symptoms, though an increase in fruit and vegetable consumption did not alone lower the risk of crohn's disease symptoms [5].

Dietary habits are linked to an increased risk of ulcerative colitis. Particularly, those with the healthiest dietary pattern in their highest tertile had a 79% decreased incidence of ulcerative colitis.

Correspondence to: Mingzhu Zara, Department of Nephrology, University of Toronto, Toronto, Canada, E-mail: Mingzhuzara@yahoo.edu.ca Received: 02-Jan-2023, Manuscript No. JHGD-23-21592; Editor assigned: 06-Jan-2023, Pre QC No. JHGD-23-21592 (PQ); Reviewed: 20-Jan-2023, QC No. JHGD-23-21592; Revised: 27-Jan-2023, Manuscript No. JHGD-23-21592 (R); Published: 03-Feb-2023, DOI: 10.35248/2475-3181.23.9.228 Citation: Zara M (2023) Diet to be followed by Inflammatory Bowel Disease Patients. J Hepatol Gastroint Dis. 09: 228 Copyright: © 2023 Zara M. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. IBD patients who are sensitive to gluten are more likely to experience flare-ups. Patients with crohn's disease and ulcerative colitis exhibited gluten sensitivity at rates of 23.6% and 27.3%, respectively. An increased risk of inflammatory bowel disease and relapses may be linked to a diet heavy in protein, especially animal protein, and sugar. A change in the gut microbiome may be a factor in the development of inflammatory gut disorders because of microbial symbiosis and immunity.

The variety of commensal bacteria is observed to be 30–50% lower in IBD patients, with particular declines in bacillota. The fact that people with IBD are more likely to have received an antibiotic prescription in the two to five years preceding their diagnosis than people without the condition provides more proof of the significance of gut flora in the development of the condition. Environmental influences, such as concentrated milk fats (a frequent component of processed foods and sweets), or oral drugs like antibiotics and oral iron preparations, might affect the enteral flora. It was shown that the host epigenome of IBD patients with active inflammation was altered in a pro-inflammatory manner by the mucosal microbiota in the large intestine. Large multinational investigations, however, have not been able to pinpoint a single microbiological biomarker of IBD, indicating that it is not caused by a specific microbe [6].

Biopsies during colonoscopy are typically used to confirm the diagnosis. Since faecal calprotectin is sensitive but not specific for IBD, it is helpful as a first examination that may hint to the likelihood of IBD [7].

REFERENCES

- Kim KC, Koh YW, Chang HM, Kim TH, Yook JH, Kim BS, et al. Evaluation of HER2 protein expression in gastric carcinomas: comparative analysis of 1414 cases of whole-tissue sections and 595 cases of tissue microarrays. Ann Surg Oncol. 2011;18(10): 2833-2840.
- Baykara M, Benekli M, Ekinci O, Irkkan SC, Karaca H, Demirci U, et al. Clinical significance of HER2 overexpression in gastric and gastroesophageal junction cancers. J Gastrointest Surg. 2015;19(9): 1565-1571.
- 3. Jørgensen JT. Targeted HER2 treatment in advanced gastric cancer. Oncology. 2010;78(1):26-33.
- 4. Kurokawa Y, Matsuura N, Kimura Y, Adachi S, Fujita J, Imamura H, et al. Multicenter large-scale study of prognostic impact of HER2 expression in patients with resectable gastric cancer. Gastric Cancer. 2015;18:691-697.
- Park DI, Yun JW, Park JH, Oh SJ, Kim HJ, Cho YK, et al. HER-2/neu amplification is an independent prognostic factor in gastric cancer. Dig Dis Sci. 2006;51(8):1371-1379.
- Bang YJ, Van Cutsem E, Feyereislova A, Chung HC, Shen L, Sawaki A, et al. Trastuzumab in combination with chemotherapy *versus* chemotherapy alone for treatment of HER2-positive advanced gastric or gastro-oesophageal junction cancer (ToGA): a phase 3, openlabel, randomised controlled trial. Lancet. 2010;376(9742):687-697.
- Takehana T, Kunitomo K, Kono K, Kitahara F, Iizuka H, Matsumoto Y, et al. Status of c-erbB-2 in gastric adenocarcinoma: a comparative study of immunohistochemistry, fluorescence in situ hybridization and enzyme-linked immuno-sorbent assay. Int J Cancer. 2002;98(6):833-837.