

Nutrition in Patients with Inflammatory Bowel Diseases

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Contributor: Alice Scricciolo

Inflammatory bowel diseases (IBD) are a heterogeneous group of inflammatory disorders of the gastrointestinal (GI) tract, with a chronic or recurrent clinical course, characterized by phases of exacerbation and remission. The clinical presentation has a heterogeneous phenotypic spectrum, characterized by gastrointestinal and extraintestinal manifestations, with atypical or non-specific symptoms. Crohn's disease (CD) and ulcerative colitis (UC) are the most typical conditions in this spectrum, and they differ in anatomical location and type of lesions. The etiology is multifactorial, and to date, the prevailing hypothesis is that an abnormal mucosal immunological response is triggered against ubiquitous antigens, such as the resident bacterial flora, in genetically predisposed individuals. However, since the 1970s, in the pathogenesis of IBD diseases, there has been an increase in the number of diagnoses, especially in industrialized countries, thus highlighting the potential role of environmental factors, such as lifestyle and dietary habits, the interaction between diet and the susceptibility to gene variants, abnormal gut microbiota and altered immune response.

IBD

Mediterranean diet

gluten

lactose

FODMAP diet

nutrition

pediatric patient

1. Introduction

IBD significantly affects the quality of life of patients and their families, as lifelong modification of behavior, lifestyle and eating habits is required ^[1]. IBD management often consists of the use of immunomodulators and immunosuppressive drugs to control active flares and maintain a state of remission ^[2]. Dietary changes might be helpful in reducing symptoms, such as abdominal pain or diarrhea, and lessening the effects of IBD complications ^[3]; however, to date, the impact of diet on the disease is still highly debated in literature, and there are no dietary guidelines. This lack of clear information pushes patients to seek information from other sources and ultimately increases the risk of self-imposed dietary restrictions with important negative consequences on patients' health ^[4]. The dietary habits and intake of patients with IBD can also be based on personal experiences that may lead to the avoidance of certain foods or food groups to self-manage the disease.

2. Dietary Approaches in Adult IBD Patients

According to the ESPEN guidelines, there is no diet that can be generally recommended to promote remission in IBD patients with active disease ^[5]. A possible risk encountered in these patients is the development of nutritional

deficiencies, especially in micronutrients; for this reason, IBD patients should be checked frequently. In patients with severe and frequent diarrhea, body fluids and electrolytes should be monitored. When food intake is not indicated or insufficient, Oral Nutrition Supplements (ONS) are used to help prevent dehydration and replace electrolytes. Enteral Nutrition (EN) should be considered as supportive therapy if oral feeding is not sufficient, while EN is always preferred to Parental Nutrition (PN). PN is indicated when there is an obstructed bowel and is required in patients with short bowels. In adults, EN is less useful than corticosteroids in influencing remission of active CD [6]. However, in children, EN is the first-line treatment in the induction of remission. Corticosteroids are associated with severe side effects, and it is also important to meet the child's nutritional requirements for proper growth and health. During the remission phases of IBD, there is no specific diet. A meta-analysis has shown that probiotic treatment is effective in the active UC and remission phases, but no beneficial effect was found in the maintenance of remission in CD patients [7]. CD, mesenteric infarction, actinic enteritis, neoplasms, volvulus, and congenital anomalies are among the main causes of extensive resection of the small bowel, with or without a portion of the colon. As the jejunum is the main site of digestion and absorption of most nutrients, a surgical resection leads to the loss of an area of absorption, which significantly reduces its function. These surgical procedures result in short-bowel syndrome, a disorder characterized by an insufficient absorptive surface area in the intestine. This intestinal loss results in the malabsorption of fluids, electrolytes and other essential nutrients, severe diarrhea, dehydration, and progressive malnutrition. ESPEN guidelines recommend that short bowel syndrome (SBS) patients consume regular whole-food diets. SBS patients with a maintained colon should have a high intake of complex carbohydrates (60%) and a low intake of fat (20%) in order to increase overall absolute energy absorption [8][9]. Compared to long-chain triglycerides, a high content of medium-chain triglycerides is preferred in SBS patients with a preserved colon. In these patients, attention is paid to the potential deficiencies in essential fatty acids and fat-soluble vitamins [8]. However, the degree of evidence for these recommendations is low. It is always important that dietary counselling be conducted by a nutritionist, considering the patient's experience, in order to meet the body's energy and metabolic requirements and to prevent nutritional deficiencies.

3. Dietary Approach in Pediatric IBD Patients

Pediatric IBD is commonly associated with malnutrition and nutrient deficiencies [10].

Historically children and adolescents with IBD appear to have growth failure at disease presentation, especially in CD, and an association with being underweight and malnutrition has also been described. Roughly one third of newly diagnosed UC pediatric patients and half of newly diagnosed CD pediatric patients present with malnutrition [11]. There is likely multifactorial etiology that contributes to sub-optimal caloric intake including food avoidance, abdominal pain and malabsorption. Furthermore, it should be considered that a chronic inflammatory condition may cause a reduction in appetite via catabolic effects and hypothalamic weight regulation. This is particularly important since malnutrition is strictly related to poor prognosis in patients with IBD [12]. Thus, ongoing scientific literature supports the main role of nutrition and diet in children with IBD. According to a recent position paper published by the European Society of Gastroenterology, Hepatology and Nutrition (ESPGHAN) [13], a global nutritional assessment in children with IBD is a central step in the management of IBD patients, with the aim of tailoring

nutritional and dietary interventions. Indeed, children with IBD should be checked for qualitative and quantitative nutrient intake on a regular basis, through a weekly record. Starting from the past decade, the relationship between diet and IBD pathogenesis has been clearly supported by scientific evidence. Besides this, several nutritional strategies have evolved over the years and are currently considered therapeutic tools, with varying degrees of efficacy and support. Concerning pediatric IBD, multiple studies and meta-analyses have proven that EEN is as effective as steroid therapy in inducing remission in children with active CD [14].

In particular, EEN may induce remission in approximately 75–85% of children with mild-to-moderate CD [15], along with superior mucosal healing, a significant decrease in inflammatory biomarkers [16] and positive benefits to growth and overall nutritional status [17]. It is important to note that the efficacy of EEN in the induction of clinical remission in children with CD was further confirmed also when compared to biological therapy [18]. This nutritional therapy is based on the use of a complete liquid formula, administered either orally or via a feeding tube, which is given as the unique source of daily nutritional requirements for 6 to 8 weeks, whilst avoiding the intake of usual solid foods [19]. Due to its excellent safety profile and its equipotential to corticosteroids in inducing remission, the European Crohn's and Colitis Organization (ECCO) and the ESPGHAN recommend EEN as the first-line therapy for mild-to-moderate pediatric CD to induce remission, both in the first flare-up and during relapses of symptoms [20]. On the contrary, until today, there has been no evidence to support EEN as an effective therapy for active UC [21]. EEN is poorly tolerated, limiting its acceptability and clinical use [19]. Therefore, alternative and better-tolerated nutritional therapy strategies have been developed to overcome non-adherence. Aimed at pinpointing a more effective and longer-term dietary therapy, the next step has been to focus on the identification of potentially proinflammatory dietary components that may negatively affect the microbiome. From this perspective, an innovative approach, called the CD exclusion diet (CDED), was described for the first time in a case series of adults and children with CD by Sigall-Boneh et al. [22]. Patients following the CDED whole-food diet coupled with partial enteral nutrition (PEN) reached high rates of clinical response and remission [22]. The efficacy of CDED + PEN compared to EEN in inducing clinical remission in children with mild-to-moderate CD was recently shown in a multinational trial [23]. CDED was developed to exclude all industrially made packaged and processed foods that are rich in preservatives and emulsifiers, sugary drinks, dairy products, red meat and all processed meats, cereals containing gluten and gluten-free industrial products, ice cream and packaged desserts. This nutritional regimen consists of different phases with incremental varieties of food allowed and provides a consistent amount of high-quality protein and sugars. A somewhat different approach was recently developed by Svolos et al. [24], with an individualized food-based therapy (CD-TREAT). The latter consists in an ordinary solid food diet that aims to recreate as closely as possible the composition of EEN, excluding certain dietary components (i.e., gluten, lactose) and the combination of others (macronutrients, vitamins, minerals and fiber). The authors anticipated that CD-TREAT aims to mimic EEN's effects on the gut microbiome, metabolome, inflammation, and clinical outcomes. After a course of 8 weeks of CD-TREAT with five children with active CD in a pilot study, four of the five children responded to CD-TREAT, showing efficacy in inducing clinical remission [24]. Less prominent approaches include the low-FODMAP diet and the specific carbohydrate diet (SCD). However, at present, no consensus has been reached on their use in the pediatric population due to a lack of evidence. The development of novel dietary treatments has enabled a turnaround in the treatment of pediatric IBD, especially CD. Although promising, the data

need to be confirmed with future well-designed studies in order to unravel the full potential of nutritional and dietary therapies for IBD in the pediatric population.

4. IBD Patients' Approach to Diet

Diet can play a key role in the etiology and symptoms of IBD. Despite this, it has been observed that patients with IBD can be divided into two groups: some patients believe that intentional avoidance of certain foods can manage symptoms and improve the disease; others do not consider dietary patterns to be an additional treatment and observe improvement only with drug treatment [25]. Qualitative semi-structured interviews with patients of IBD about the psychosocial impact of food and perceptions, eating and drinking showed that some participants made a direct link between the presence or severity of their symptoms and the type of food. These patients are constantly trying to change their diet according to how they feel after eating a certain food, while others make no changes to their diet after an IBD diagnosis [26]. Whereas only few participants used diet as their first treatment for IBD, the majority believed there should be a combination of diet and drugs. Thus, the choice of dietary patterns of patients with IBD is not a static process but evolves with the disease and symptoms. Patients' dietary patterns are connected to their understanding of how food items influence their disease course. Dietary restrictions in many patients occur during flares and are stopped in remission. Some patients claim that they can only eat chicken and rice or liquid nutrition during a flare [2][27]. Several studies have been conducted to analyze the foods that are avoided by patients with IBD, summarized in **Table 1**, and the list of "bad" and "good" foods differed between patients. The bad foods are considered to be triggers that can exacerbate their IBD symptoms, such as diarrhea, pain, nausea and bloating. Bad foods that are typically avoided include high-fat and spicy foods, meat alternatives, fruit, vegetables, dairy products and milk, alcohol, coffee and fizzy or carbonated drinks [1][2][27][28][29]. The dietary patterns of patients with IBD are analyzed via a dietary questionnaire and interviews, both of which provide insight into intentional food avoidance.

Table 1. Main food items that are avoided in the IBD population according to some studies.

Food Items	Avoidance
Alcohol, salads and raw vegetables, and deep-fried foods	These foods are the most commonly eliminated during the acute phase [24][30]. Alcohol was avoided to prevent flares [27][23].
Capsaicin (spicy foods)	Up to 84.8% of the IBD population chooses to avoid spicy food to prevent disease relapse [2][28][24].
Fresh fruit, vegetables and fibres	Patients are likely to avoid fibers because they are worried about disease complications [1][2][28][29].
Milk and dairy products	Dairy products are described to be commonly excluded by patients and are the food group that is most typically avoided following a health professional's advice [2][27][28][24][30][31].

Food Items	Avoidance
Sweets	Sweets were associated with IBD symptom aggravation more in UC patients than in CD patients.
Meat alternatives (legumes, nuts, seeds and peanut butter)	These are the most eliminated food items in the sample of IBD patients [2][24].
Coffee and fizzy drinks	Studies report that patients exclude coffee due to the worsening of symptoms; lower coffee consumption was also reported in UC patients and patients with active disease [9][30][32].

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