Chapter 4

Dietary Management in IBS Patients

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Abstract

Irritable bowel syndrome (IBS) is a chronic, relapsing functional disorder of the gastrointestinal tract characterized by abdominal pain, bloating, and changes in bowel habits lacking a known structural or anatomic explanation. According to the Rome IV criteria, IBS consists of a set of altered bowel habits over a period of time and includes abdominal pain and discomfort. The pathogenesis of IBS is not completely understood, although it has been noted that various mechanisms are involved determining the onset of symptoms. The risk factors include antibiotics, enteric infection, food intolerance, altered pain perception, altered brain-gut interaction, dysbiosis, increased intestinal permeability, visceral hypersensitivity, and increased activation of the gut mucosal immune system. There has been interest regarding the possible role of food in IBS. Diet is crucial for managing IBS; it plays an important role both in the genesis and in the improvement of symptoms. The aim of the study was to summarize the evidence from the literature, which explains those causes tending to promoting IBS symptoms, such as food content short-chain carbohydrates and the presence of food allergy or food intolerance.

Keywords: IBS, FODMAPs, microbiota, diet, food allergy

1. Introduction

Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder; it is not associated with organic causes which can be detected using current diagnostic tools [1–3]. It is characterized by abdominal pain, distension, bloating and stool irregularities and its

prevalence is 10–20%. The prevalence of IBS differs among countries; this may be due to varying application of the diagnostic criteria, demographic differences and other factors, such as lifestyle including physical activity, dietary habits, distress, and pharmacological treatment [4].

The incidence of IBS in women is twofold that of men, the majority in the <50-year-old age group and having a lower socioeconomic status [5]. Symptom severity varies in different patients, from tolerable to severe, possibly interfering with daily activity [6]; in fact, patients with IBS have a reduction in the quality of life and work productivity, and they sometimes tend to isolate themselves socially [5, 7]. Many patients report avoiding social events due to embarrassment from postprandial symptoms and lack of access to toilet facilities [2, 8].

The pathogenesis of IBS is not completely understood, although it has been suggested that various mechanisms are involved [9], such as the use of antibiotics, enteric infection, food intolerance, altered pain perception, altered brain-gut interaction, dysbiosis, increased intestinal permeability, visceral hypersensitivity, and increased activation of the gut mucosal immune system [7].

Since the etiology of IBS is unknown, there is no specific therapeutic strategy; in fact, treatment is often symptomatic, namely the alleviation of symptoms. The diagnosis of IBS is clinical, based on symptoms according to the Rome IV Criteria [10], which have updated the previous criteria:

The term "discomfort" has been eliminated since it was ambiguous for the patients.

In the past, the relative frequency of abdominal pain as a diagnostic criterion had to be at least 3 days a month.

"Improvement of symptoms with defecation" is no longer quoted; instead "related to defecation" is used since a large number of patients did not have improvement in abdominal pain with defecation but, rather, a worsening [3].

Symptom onset should occur at least 6 months before diagnosis, and the symptoms should be present for three successive months [10, 11].

The following IBS subtypes can be identified according to the predominant change in bowel habit:

- (IBS-C) with predominant constipation
- (IBS-D) with diarrhea
- (IBS-M) mixed
- (IBS-U) unsubtyped [1]

Over time, patients may migrate between the different IBS subtypes, most commonly from IBS-C or IBS-D to IBS-M [7].

2. Diet and lifestyle in IBS

As has already been pointed out, IBS patients frequently report that symptom exacerbation occurs after the ingestion of some foods. In fact, it has been reported that approximately 90% of patients voluntarily restrict their diet in order to prevent or improve their symptoms [2, 7]. Over time, excessive limitation of the quality and/or quantity of foods assumed can lead to malnutrition [12, 13].

Furthermore, the occurrence of exaggerated symptoms after food ingestion, such as gastric hypersensitivity to distension, small intestinal hypersensitivity to fat, and hypersensitivity to the effect of gut hormones, acid, capsaicin, and the products of colonic fermentation, has been observed [1, 14, 15].

This exaggerated response to the ingestion of lipids probably reflects the complexity of digestion and absorption, which is also present in physiological conditions. Another aspect regards the short-chain carbohydrates, which are poorly absorbed in the small bowel; therefore, the increased osmotic load increases the intestinal water content.

Short-chain carbohydrate malabsorption leads to their rapid fermentation which, in turn, leads to the production of short-chain fatty acids and gas, mainly hydrogen, carbon dioxide and methane, which may induce the bloating responsible for the abdominal pain [16, 17].

Owing to the strict relationship between food and symptom development present in patients with IBS, therapeutic management includes dietary and lifestyle advice and, in case of necessity, psychotherapy and pharmacological therapy targeted toward the symptoms [18].

In clinical practice, the following items need to be evaluated in patients with IBS, in order to identify their possible pathogenetic role:

1. Fermentable Oligo-, Di-, Monosaccharide and Polyols (FODMAPs)
2. Food allergy
3. Non-celiac gluten sensitivity (NCGS)
4. Interaction between diet and gut microbiota

2.1. FODMAPs

The term FODMAP was first coined by researchers at Monash University in Melbourne, Australia, to describe a group of short-chain carbohydrates and polyols [16, 19].

In recent years, the intake of FODMAPs has increased in Western diets, in particular, that of fructose due to the greater availability and consumption of fruit and fruit juice, and the extensive use of high fructose corn syrup in a wide variety of processed food and beverages [20].

2.1.1. Fructose

Fructose is a monosaccharide which is dose-dependently and variably absorbed; when an excess of glucose is present, it is taken up by a low-capacity facultative transporter called GLUT 5 [16, 21].

When the concentration of fructose is greater in the lumen with respect to that in the epithelial cells, a gradient of concentration is created which permits the fructose, by means of transport proteins, to enter into the interior of the epithelial cells, thereby being absorbed.

The transport proteins, however, saturate at low fructose concentrations which, in turn, lead to malabsorption [22]. However, when fructose is present with glucose, the fructose is taken up more efficiently by means of the GLUT2 transporter. The fructose-glucose ratio is crucial for adequate fructose absorption, and a 1:1 ratio is considered optimal [16, 21]. It has been observed that 40% of the population has "fructose malabsorption" due to its scarce capacity of absorption at the intestinal level [22–24].

2.1.2. Fructans and galacto-oligosaccharides (GOS)

Fructans are oligo or polysaccharides made up of small chains of fructose units having a terminal molecule of glucose. Fructans with a 2–9 unit length are defined as oligofructose and those with >10 units as inulins [22, 25, 26]. Due to the lack of enzymes capable of completely hydrolyzing the glycosidic bonds of these polysaccharides, the human body absorbs only 5–15% of the fructans; the fructans which are not absorbed are released into the colon where they undergo fermentation [26]. Wheat represents the major source of fructans in the diet (1–4%) [22, 27]. Rye also contains fructans; its chain length is longer than that found in wheat, which could make it less osmotically active or less rapidly fermented. The principal sources of galacto-oligosaccharides (GOS) are raffinose, which is made up of one fructose, one glucose, and one galactose molecule, and stachyose, which has the same composition as raffinose with the exception of having an additional galactose molecule. The human body is not capable of digesting raffinose and stachyose due to the lack of enzymes able to hydrolyze the bonds. Galactooligosaccharides are present, above all, in vegetables [22, 28]. Fructans and galactooligosaccharides are defined as "prebiotics" due to their ability to selectively stimulate the growth of beneficial colonic bacteria, specifically *Bifidobacteria* and *Lactobacilli* [16, 29, 30].

2.1.3. Polyols

Sorbitol, mannitol, xylitol, maltitol, erythirol, polydextrose, and isomalt are sugar alcohols. Sorbitol and mannitol are the major types found in food, and they are found naturally in fruits and vegetables, or as added sweeteners in low-calorie food products [16]. They are identified on food packaging with the following numbers: E420 (sorbitol), E421 (mannitol), E965 (maltitol), E967 (xylitol), and E953 (isomalt) [22]. Their rates of absorption depend largely on molecular size; their absorption is passive and varies between individuals [22, 31].

2.1.4. Lactose

Lactose is a disaccharide consisting of galactose bound to glucose; intestinal absorption requires hydrolysis to its component monosaccharides by brush-border enzyme lactase. Lactase begins its activity at approximately the eighth week of gestation; activity increases until week 34 and the peak of activity is at birth [32].

After the first few months of life, lactase activity begins to decrease; this condition is defined as "lactase non-persistence." However, approximately 30% of the population retains its capacity to digest lactose (lactase persistence); in particular, this is observed in the populations of Northern Europe as a result of the introduction of dairy farming approximately 10,000 years ago [17, 33]. Only 50% of lactase activity is necessary for the efficient digestion of lactose without causing symptoms of intolerance [17]. Lactase deficiency determines lactose intolerance and is defined as markedly reduced brush-border lactase activity, whereas lactose malabsorption occurs when a substantial amount of lactose is not absorbed in the intestine. Three distinct forms of deficiency exist: congenital, primary and secondary. The congenital form is extremely rare; it can be observed in newborns and is characterized by diarrhea from the first exposure to breast milk and by growth defects [32]. "Primary lactase deficiency" refers to the condition of lactase non-persistence, already described above, whereas secondary or acquired lactase deficiency refers to the loss of lactase activity in individuals with lactase persistence. This could be secondary to a gastrointestinal disease, which damages the brush border and is usually reversible. The primary aim in treating lactose intolerance is the improvement of symptoms. Therefore, it is necessary to reduce the malabsorption, and limiting the intake of the lactose found in milk and its derivatives is recommended.

In order to avoid the onset of symptoms, patients with self-reported lactose intolerance, even those with IBS, can ingest at least 12 g of lactose per day [17, 34, 35]. However, it has also been observed that, in many individuals, the restriction of lactose alone was not sufficient for improving functional GI symptoms. This is because lactose intolerance is part of a wider intolerance to FODMAPs [17, 36]. Lactase enzyme replacement products can be found commercially, but they should be used only by those individuals who have isolated lactose intolerance and wish to enjoy dairy products [17]. It is important to remember that dairy products are the major source of calcium in many individuals; therefore, it is reasonable to recommend increasing calcium intake from other foods, water rich in calcium or supplements, especially in the presence of other risk factors for osteoporosis [17].

2.2. Food allergy

Food allergy is an adverse immune response toward food proteins or a form of food intolerance associated with a hypersensitive immune response. There are three types of food allergies: IgE mediated, mixed IgE/non-IgE, which involves eosinophilic and other cellular components, and non-IgE mediated [37].

Food allergy has rapidly increased in prevalence. It suggests the important role of environmental factors in disease susceptibility [37]. Of the 20–30% of the population reported to be

allergic or to have allergic children, the presence of allergy can be ascertained in only 6–8% of children under 5 years of age and in 3–4% of adults [38, 39].

In the presence of food allergies, a small quantity of food can cause an immediate reaction. The symptoms involving the gastrointestinal tract can include nausea, vomiting, abdominal cramps and diarrhea, other signs can involve the oropharyngeal tract or the skin [38, 40]. There has been interest regarding the possible role of food allergies in IBS, but few data are available to support this association [38].

In order to exclude food allergies, it is necessary to proceed on the basis of the results of the following tests:

1. *Total serum IgE*: High values may indicate the presence of some food allergies [38, 41].
2. *Immunoglobulin G (IgG):* It often produces false positive, and it is not recommended, as a diagnostic test, by national and international guidelines [38, 42].
3. *The radioallergosorbent test (RAST) of food/serum food-specific IgE:* A direct correlation exists between increasing concentrations of food-specific serum IgE and the probability that an individual will react to an ingested food [38, 43–45].
4. *The skin prick or scratch test:* Positivity indicates the presence of IgE to specific foods [38, 42, 44–47].

Furthermore, it is necessary to make an interview and administer an accurate food questionnaire in order to identify the correlation between specific food and symptoms.

2.3. Non-celiac gluten sensitivity (NCGS)

Currently, the gluten-related disorders actually documented are celiac disease (CD), non-celiac gluten sensitivity (NCGS) and wheat allergy (WA) [48]. Celiac disease is an autoimmune condition, which is characterized by an immunological response to ingesting gluten, which results in small-intestine villous atrophy with increased permeability and malabsorption of nutrients [48, 49].

Wheat allergy is characterized by an IgE-mediated response against various wheat components, which cause gastrointestinal and respiratory symptoms [48–50].

A food allergy to wheat begins with different symptoms as vomiting, abdominal pain, asthma, allergic rhinitis, urticaria/angioedema, acute exacerbation of atopic dermatitis, and exercise-induced anaphylaxis. The prevalence of IgE-mediated food allergies to wheat, confirmed by a food induce, is unknown.

At the moment, the management of IgE-mediated wheat allergy is mainly based on the avoidance of both food and inhaled wheat allergens. Patients allergic to wheat must be trained to identify relevant food allergens on labels, and written instructions should be given to effectively eliminate wheat from their diet [51].

Non-celiac gluten sensitivity is an emerging clinical problem characterized by various manifestations, in particular by IBS-like gastrointestinal symptoms and extra-intestinal

symptoms, such as malaise, fatigue, headache, mental confusion, anxiety, sleep abnormality, and skin rash related to the ingestion of gluten-containing foods in patients who are not affected by either celiac disease or wheat allergy [52].

The symptoms generally improve after the removal of wheat products from the diet. Due to the lack of biomarkers, the diagnosis of NCGS is mainly based on clinical criteria [52, 53] after having excluded the presence of CD, WA, gluten ataxia, and dermatitis herpetiformis [52].

Similar to IBS, NCGS affects more young women (in their third decade of life; female:male = 3:1) [52]. The consumption of wheat has increased and is correlated not only to its adaptability and potential for high yields but also to its viscoelasticity, which allows it to be processed into several food items, such as bread, baked products, and pastas [54].

There are two components of wheat which could evoke IBS symptoms: proteins (primarily gluten) and short-chain carbohydrates (primarily fructans and galactans) [52].

Various studies have been carried out to verify which of these two components is responsible for the onset of symptoms. A study was carried out on patients with IBS who autonomously started a gluten-free diet; it was observed that the gastrointestinal symptoms significantly and consistently improved with a low-FODMAP diet, and symptoms did not worsen with either a low- or a high-dose challenge with gluten [54, 55].

Another study involving adults who believed that they had NCGS concluded that it was not gluten to induce the clinical picture [54]. Trials carried out on patients with suspected NCGS support the data regarding a greater improvement of the symptoms with a low-FODMAP diet as compared to a gluten-free diet [56–58].

A recent systematic review regarding NCGS concluded that there is insufficient evidence to support the efficacy of a gluten-free diet for NCGS [59].

2.4. Interaction between diet and microbiota

From birth, the gut microbiota plays various roles in the gastrointestinal tract. Postnatal gut function and immune development are largely influenced by intestinal microbiota. In fact, it has a role in gastrointestinal motility and the immune system, it provides protection against infection, it contributes to the development of gut function and the regulation and maintenance of the intestinal barrier, and it promotes food tolerance. Microbial species promote symbiotic host-bacteria interactions, which are fundamental for human health [12, 60, 61].

The gastrointestinal microbiota is determined by host genetic and environmental factors [12, 60, 62]. The composition of the microbiota varies according to prenatal events, delivery methods, infant feeding, infant care environment, and antibiotic use. Emerging evidence has shown that early microbiota colonization may influence the occurrence of eventual diseases [61]. Gut microbiota interferes with the intestinal functions it can be the cause of irregularity of intestinal sensitivity, motility, neuroimmune signaling, such as the alterations of the mucosal barrier and pattern recognition receptor expression and dysfunctions of the hypothalamus-pituitary-adrenal axis [63].

It has an important role in the digestion of dietary components, resulting in metabolites which may directly or indirectly contribute to IBS symptoms [12]. The vast majority of microbial commensal species give rise to symbiotic host-bacterial interactions, which are fundamental for human health [64]. Disruption and/or imbalance of the establishment of stable normal gut microbiota, dysbiosis, may be associated with the pathogenesis of several gastrointestinal conditions, such as inflammatory bowel disease (IBD) and irritable bowel syndrome, and wider systemic manifestations of disease, such as obesity, type 2 diabetes and atopy [12, 64, 65]. In the healthy gut, intestinal microbiota can prevent the adherence of pathogenic bacteria to the wall of the gastrointestinal tract [66]. In addition, dysbiosis in the gut may facilitate the adhesion of enteric pathogens, which could be associated with IBS symptoms [67]. Alteration of the composition of the normal microbiota and disturbed colonic fermentation in IBS patients may play an important role in the development of IBS symptoms, with a significant increase in the ratio of Firmicutes to Bacteroidetes [65, 68]. Dysregulated intestinal immune function, chronic low-grade mucosal inflammation and increased mucosal permeability and barrier dysfunction have all been suggested to be pathogenic mechanisms in IBS in which the intestinal microbiota might have a role [12, 69, 70]. Part of the etiology of IBS may involve the use of antibiotics; in these cases, probiotics are effective in ameliorating symptoms, even if the consistency of benefits across clinical studies is difficult to discern due to variation in strains, product dosages and the duration of the trials [68, 71–73].

Manipulation of the gut microbiota represents a new strategy for the treatment of IBS. Modulating the gut bacterial composition, expanding the bacterial species considered to be beneficial (*Lactobacilli* and *Bifidobacteria*) and reducing the bacterial species considered to be harmful (*Clostridium, Escherichia coli, Salmonella, Shigella*, and *Pseudomonas*) should attenuate IBS symptoms [63].

Several studies using culture-based and culture-independent methods have shown that the microbiota differs between IBS patients and healthy controls [12, 74, 75].

However, the association between IBS symptoms and specific bacterial species is uncertain [12, 76]. Decreased levels of *Lactobacilli* and *Bifidobacteria*, increased levels of anaerobic bacteria, such as *Streptococci* and *Escherichia coli*, as well as increased ratios of Firmicutes, Bacteroidetes, and *Clostridium* species, have been confirmed in several studies [12, 77, 78]. Studies have indicated that the microbiota, its function and its metabolic output are influenced by dietary patterns [79].

Habitual long-term dietary patterns have been directly linked to intestinal microbial enterotypes. Protein and animal fat intake have been associated with the Bacteroides enterotype, whereas a high-carbohydrate intake has been associated with the Prevotella enterotype [12, 80].

Preliminary data suggest that a diet with a low content of FODMAPs can reduce the growth of important species, such as Bifidobatteri [81]. The effect of short-term dietary interventions on the microbiota composition appears to have only a modest effect [12, 80, 82]. Diet and composition of the microbiota are two major interrelated factors, which can modify susceptibility to food allergy [37].

The number of publications describing an altered microbiota in allergic disease has significantly increased in recent years. The increasing use of antibiotics, in both humans and agriculture, and the increasing consumption of a high-fat/low-fiber diet have had a major impact on the gut microbiota and have been associated with an increased allergic response to food in industrialized countries in recent decades [83–85].

The use of probiotics to stabilize microbial homeostasis seems to be promising, but, to better understand the potential beneficial impact from probiotics, prebiotics, and bacterial-produced metabolites in the treatment of allergic disease, additional studies are needed [85].

3. Role of diet

Therefore, diet is crucial for managing IBS despite the lack of solid evidence involving many dietary recommendations for IBS; this issue must be addressed in clinical practice. The British Dietetic Association and NICE guidelines (National Institute for Health and Clinical Excellence) recommend that dietary and lifestyle advice should be routinely provided to patients [2]:

- Patients with IBS should be educated about the importance of self-help in effectively managing their IBS through information regarding lifestyle, physical activity, diet, and symptom-targeted medication.
- Professionals should encourage people with IBS to use their available leisure time to make relax.
- People should be motivated to increase their activity levels.
- In people with IBS diet and nutrition have to be assessed and general advice should be given:
  - Have regular meals during the day and take time to eat with calm.
  - Avoid skip meals and stay for long time without eating.
  - Drink at least 1500 ml of liquid/day, preferring water or other non-caffeinated drinks.
  - No more than three cups per day of tea and coffee.
  - Reduce alcohol and fizzy drinks.
  - Limit the intake of high-fiber food; reduce the intake of "resistant starch" (RS) because they resist to digestion in the small intestine and reaches the colon intact.
  - No more than three portions of fresh fruit per day.
  - In case of diarrhea, it is advisable to avoid sorbitol, an artificial sweetener found in sugar-free sweets, light drinks, and in some diabetic and diet products.
  - In case of wind and bloating, it is advisable to eat oats and linseeds.
- Healthcare professionals should review the fiber intake of people with IBS, adjusting it while monitoring its effect on the symptoms. People with IBS should be discouraged from eating

insoluble fiber. If an increase in dietary fiber is advised, it should be soluble fiber. People with IBS could try probiotics, and they should take the product for at least 4 weeks while monitoring the effect. Probiotics should be taken at the dosage recommended by the manufacturer.

- Discourage the use of aloe vera in the treatment of IBS.
- If a person's IBS symptoms persist while following general lifestyle and dietary advice, offer advice on additional dietary management.
  - Include single food avoidance and exclusion diets such as a low-FODMAP diet.
  - Only be given by a healthcare professional with expertise in dietary management.

A low-FODMAP diet provides for the restriction of all short-chain carbohydrates by finding low-FODMAP alternatives in each food group. The aim is that of reducing the malabsorption induced by these nutrients and the consequences, such as luminal distension and fermentation caused by the bacteria of the colon, which give rise to the symptoms [52].

In healthy adults, FODMAPs do not cause gastrointestinal symptoms; conversely, in IBS patients, this probably is a consequence of the previously mentioned abnormalities in gut physiology and visceral sensation [7, 86, 87].

There is evidence of low-FODMAP diet efficacy; in fact, it has been observed that it reduces and controls the GI symptoms with respect to a high-FODMAP diet [2, 88, 89], and this has also been confirmed by clinical trials [5, 90].

The low-FODMAP diet was compared with the indications of the NICE guidelines in order to verify which of the two approaches was better in controlling the symptoms.

In particular, in one study, it emerged that there was significantly greater satisfaction with symptom response with the low-FODMAP diet (76%) as compared to the NICE guidelines (54%) [5, 89].

Instead, a recent single-blinded random controlled trial (RCT) compared the efficacy of a low-FODMAP diet to the NICE guidelines for a 4-week period at the end of which an improvement in the IBS Symptom Severity Score (IBS SSS) was observed in both groups but without significant differences. At the end of the study, 56% of the patients on the low FODMAP diet and 46% on the traditional IBS diet responded to the treatment, and the IBS SSS was reduced to ≥ 50 relative to baseline. Food diaries demonstrated good adherence to the dietary advice [16, 91].

A recent meta-analysis, which compared IBS patients who followed a Westernized diet with patient who followed a low FODMAPs diet, showed that adherence to a low-FODMAP diet help to ameliorate all the functional IBS's symptoms and their severity also improving the quality of life score [20].

The symptom with the least improvement was constipation; in fact, a typical FODMAP diet can often be lacking in fiber content. If one decides to follow a low-FODMAP diet, it should

be followed for at least 2–6 weeks in order to be able to verify whether there is effectively an improvement in the symptomology [5].

If patients report improvement, dietary rechallenge with FODMAPs may be tried gradually, that is, one food at a time can be reinserted, starting with small quantities [5]. The risk of inserting more than one food at a time is that of not being able to verify which, effectively, is that responsible for the worsening of the symptoms [5].

Decisions related to the food allowed or to that which should be avoided should always be based on individual tolerance; one valid evaluation instrument is a food diary in which patients have to report what they eat qualitatively, thereby being able to identify potential trigger foods [5].

The effect of a long-term FODMAP diet is not clear; few data are available regarding its long-term efficacy and safety. Presumably, a long-term low-FODMAP diet could lead to nutritional inadequacy. In one study evaluating the effect of fermentable carbohydrate restriction as compared with a control diet, no difference was found in micronutrient intake, except for a lower calcium intake, presumably as a result of the lower intake of dairy products [16, 90].

This might pose a problem principally for children and postmenopausal women [16]. The psychosocial risks of imposing a dietary change and the various difficulties encountered by an IBS patient, difficulties in socialization and eating disorders, such as orthorexia nervosa, should not be underestimated [52, 92].

4. Conclusions

The symptoms of IBS can be similar to those of other pathological conditions; it is necessary to exclude them by means of diagnostic examinations. From a nutritional point of view, the symptoms of IBS patients linked to food as well as to their present food habits must be evaluated carefully in order to reach a nutritional diagnosis and the specific objectives to reach, verifying the relative changes by means of successive checkups. Furthermore, a careful evaluation of the nutritional state is recommended with the aim of identifying, if present, the eventual lack of macro/micronutrients. It is possible to consider a dietetic regimen, which has a behavioral checkup linked to the indications of the NICE guidelines; successively, or in association with the execution of the guidelines, a low-FODMAP diet can be proposed which foresees a reduction in the intake of foods containing high quantities of short-chain carbohydrates and polyols in favor of substitutes for each food group in order to avoid undesired weight losses and nutritional deficits. A low-FODMAP diet must be limited to a precise period of time, and the patient must be monitored by keeping a daily food diary where the food consumed over a 24-h period, the subdivision of the meals and the clinical picture present is reported. Use of a diary makes patients feel more understood, with a consequent perception of greater interest in their problems and needs. With the reduction or disappearance of the clinical picture, the foods previously excluded can be reinserted, one at a time, evaluating individual tolerance with the aim of restoring a complete and balanced diet, always with the help of a food diary.

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