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IRRITABLE BOWEL SYNDROME – NEW PERSPECTIVE

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ABSTRACT

Background: IBS is a worldwide chronic gastrointestinal tract disorder. The challenges in treatment include unknown causes, non-specific symptoms, lack of precises testing, diagnosis based on ruling out other ailments. New therapies and probiotics show promise in improving the now available treatments and alleviating symptoms in a more precise manner based on IBS type. The understanding of the gut-brain axis as the basis of novel therapies shows promise in further implementation of more efficient treatment.
Aim: This narrative review analysis the new therapeutical possibilities and probiotic influence on IBS treatment and the gut-brain axis.
Material and methods: A search of relevant research databases was conducted to identify eligible papers.
Results: Management of IBS is still on insufficient level. Some studies showed the opportunities arising from the implementation of prostaglandines or even locally acting agonist of opioid receptors. Probiotics also play an important role. On the other hand, the pathophysiology of the disease and the interactions between hormones and cytokines still recuire further studies.
Conclusions: IBS is a complex medical condition, the usage of probiotics and other novel therapeutic agents may alleviate the severity of the disease.

KEYWORDS

Irritable Bowel Syndrome (IBS), Gut Microbiota, Gut-Brain Axis, Pathophysiology, Probiotics

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1. Introduction

Irritable bowel syndrome (IBS) is a chronic gastrointestinal tract disorder, which affects 10-15 % patients worldwide (1) (2).

Its pathophysiology is characterized by reoccurring symptoms including bloating, abdominal pain, cramping, diarrhea or constipation, which over time may change and vary among individuals (2). While its causes aren't yet fully understood the triggers and possible treatment strategies allow to address the ailment and reduce their severity. However, the gut-brain interaction is one of the theories by which the pathophysiology may be explained. Commonly IBS is triggered by increased stress or anxiety, infections, not following dietary restrictions by consumption of dairy, wheat, citrus fruits or other products which trigger a response in an individual suffering from IBS. The diagnosis itself is based on medical history and symptom evaluation, ruling out other diseases. At this time no tests to definitely diagnose IBS exists (3). The diagnostic criteria used in evaluation include recurrent abdominal pain. The pharmacotherapy of IBS is based on a multifaceted approach. Treatment involves the use of antibiotics, probiotics, symptomatic medications, and targeted therapies tailored to specific forms of IBS. In recent years, researchers have focused particular attention on these targeted therapies (4). Gut microbiota could be a potential modulator for the gut brain-axis, in recent years the understanding of its role in our organisms and its homeostasis has improved, and a connection has been found between the functioning of the blood brain barrier and gut microbiota not only in IBS but also in other diseases (7) (8) (11) (12) (16). Neurotransmitters such as serotonin (5-HT), norepinephrine (NE), gamma-aminobutyric acid (GABA) and glutamate all participate in the functioning of the gastrointestinal tract. In this analysis neurotransmitters are mentioned in regard to their role in the pathophysiology of IBS (19). Medications prescribed for IBS vary in their therapeutical goal based on the IBS subtype. Furthermore, common treatment options focus on addressing the psychological triggers. With the specific ethology of IBS still unknown the therapies focus on pain relief and improvement of daily life. Next-generation probiotics (NGPs) are a promising new type of microbial therapy (26) (27) (30) (34).

2. Aim of the article

This systematic review aimed at summarizing the most meaningful and current information on the topic of patophysiology of IBS and the treatment possibilities in this condition. The research was performed by using search phrases in PubMed.

3. Results

3.1 The gut–brain axis

For many years, the interactions between the central nervous system and the gastrointestinal tract have been a subject of interest to many researchers. This unique connection between these two exceptional human systems has been termed the gut-brain axis. It is now accepted that communication within this axis is bidirectional and involves numerous neuronal, hormonal, immunological and metabolic mechanisms. In recent years, particular attention has been paid to the role of the gut microbiota as a potential modulator of the gut-brain axis (5) (6).

The gut microbiota is the collection of microorganisms that colonise the human digestive tract. It consists primarily of bacteria, but also includes viruses, fungi and archaea. These microorganisms have various relationships with the host organism and can have both positive and negative effects. Commensals are microorganisms that colonise the host organism and benefit from living in its environment without causing any negative effects, such as disease. Under natural conditions, commensal microorganisms play a significant role in maintaining the body's homeostasis. Technological advances in recent years, including advanced DNA sequencing methods and improved microorganism culture techniques, have enabled the much more effective identification of gut microorganisms. This has contributed to a significant expansion of our knowledge regarding the diversity of the human gut microbiota. Thanks to these advances, it has become possible to answer key questions regarding which microorganisms make up the gut microbiota, how they influence its functioning, and how they interact with one another (7) (8).

Both the cells of the digestive tract and those of the brain develop from closely related parts of the embryo (6) (9). The development of the central nervous system is influenced by a range of environmental factors, and the gut microbiome plays a particular role in regulating and guiding neuronal development (10).

One of the key mechanisms linking the gut microbiota to the functioning of the blood–brain barrier (BBB) is its influence on the maturation and integrity of tight junctions in the endothelium of cerebral blood vessels. Under physiological conditions, the presence of a healthy gut microbiota helps maintain the host's

immune and metabolic homeostasis, which indirectly influences the stable expression of tight junction proteins, such as claudins and occludin. In the absence of gut microbiota, as is the case in germ-free (GF) mice, disturbances in the maturation of the blood–brain barrier (BBB) are observed, manifesting as increased permeability in both adult and embryonic individuals (11) (12).

This is primarily due to reduced expression of tight junction proteins, which leads to their abnormal organization and a weakening of the barrier structure. Consequently, there is an increased permeability for substances from the blood into the central nervous system. Bacterial metabolites, in particular short-chain fatty acids (SCFAs), which are produced as a result of the fermentation of dietary components by the gut microbiota, appear to be a key element of this process. SCFAs may act as signaling molecules, influencing the expression of tight junction proteins and supporting the maturation and stability of the BBB. This relationship is supported by the observation that colonization of GF mice with microbiota derived from pathogen-free (PF) animals, or the administration of SCFA-producing bacteria, leads to increased expression of tight junction proteins and reduced permeability of the BBB. It can therefore be concluded that the gut microbiota acts as a regulator of BBB integrity, operating both by influencing the expression of endothelial structural proteins and by producing signaling metabolites that are essential for the proper maturation and functioning of the BBB (12) (9).

Excessive microbial growth in the small intestine is considered an important symptom in patients with irritable bowel syndrome (IBS). Analysis of the composition of the faecal microbiota in patients with IBS diagnosed using the Rome criteria revealed significant quantitative differences compared with the control group. The observed changes in the composition of the gut microbiota may influence the activation of the immune system. Commensal gut bacteria and their structural components, such as lipopolysaccharides (LPS) present in the cell wall of Gram-negative bacteria, act as ligands for Toll-like receptors (TLRs). Furthermore, the TLR5 receptor recognizes flagellin – a protein that forms bacterial flagella. This suggests that alterations in the composition of the gut microbiota in patients with IBS may lead to abnormal activation of the immune response, and that gut dysbiosis may contribute to the development of chronic, low-grade inflammation (13) (14) (15).

Disruptions to the brain-gut-microbiome axis may contribute to the development not only of irritable bowel syndrome and other functional gastrointestinal disorders, but also of numerous psychiatric and neurological disorders such as autism and Parkinson's disease (16). Most of the literature links the composition of the microbiota to the development of the above-mentioned diseases, however, scientific evidence is still insufficient. Nevertheless, data from both preclinical and clinical studies offer hope for new therapies and a better understanding of the pathomechanisms underlying these conditions.

In IBS, the gut-brain axis plays a very important role because of the various disorders that occur along with it, which may be multifactorial. This pathway is influenced by neurotransmitters such as noradrenaline, serotonin, glutamate, GABA, and acetylcholine. Visceral sensations are initiated by the activation of the parasympathetic nervous system and the nerves located in the intestines, which form an extensive network of neurons—the Enteric Nervous System (ENS). This activation is triggered when the EC and EEC detect signals from the lumen of the intestine, leading to the release of serotonin, among other substances (17) (18).

Serotonin (5-HT) is produced mainly in enterochromaffin cells located in the intestinal epithelium; the amino acid tryptophan is essential for its synthesis. Once released, it stimulates peristaltic movements and increases blood flow in the duodenum and ileum. The main serotonin receptors in the gastrointestinal tract include 5-HT1, 5-HT3, and 5-HT4, which are responsible for inhibiting or stimulating intestinal motility and attenuating the transmitted somatogenic pain signals. On the other hand, the 5-HT3 receptor may contribute to the onset of visceral pain due to the activation of neurons involved in pain perception. Its impact is not limited only to the digestive system but also affects our behavior and well-being, which is why the symptoms of IBS are often related to the serotonin system. Some symptoms of IBS, such as excessive sensitivity of the digestive system and diarrhea, may result from reduced expression of SERT, which is a transporter responsible for serotonin reuptake (19) (18) (20).

Norepinephrine (NE) is a catecholamine that plays an important role in the sympathetic nervous system, specifically in the body's response to stressful situations. In the gastrointestinal tract NE influences motility, secretory functions and vascular perfusion. It also impacts the condition of the intestinal microbiota; stress contributes to increased NE levels in the organism, and this can lead to increased gut permeability and inflammatory conditions (19) (21).

Gamma-aminobutyric acid (GABA) is a neurotransmitter that is responsible for the inhibitory function in the central nervous system, its functions in the gastrointestinal tract include the influence on motility, gastrointestinal secretion, and visceral sensation. The effects of dysregulation of the GABAergic system will

vary and will depend on the place where the disorder occurred; it may occur at the level of the central nervous system or within specific regions. Mood disorders and visceral sensitivity will manifest themselves mainly in IBS when there are disturbances at the level of the CNS. Disturbances in gastrointestinal motility are the result of disorders of the GABAergic system at the intestinal level. Reduced levels of GABA and the enzyme GAD2 have been observed in certain subtypes of IBS; therefore, this pathway is significant in the development of symptoms in this disorder (19) (22) (21) (23).

Glutamate is a neurotransmitter that performs many functions in the body; in the central nervous system, it is the primary stimulating neurotransmitter. In the gastrointestinal tract, it helps promote digestion and the absorption of nutritional components. According to some studies, dietary glutamate supplementation has been associated with IBS symptoms such as bloating and abdominal pain (19) (21).

3.2 Probiotics and drugs

The most common subtype is IBS-C, or irritable bowel syndrome with constipation predominance. It is estimated to occur in as many as 27.9% of patients diagnosed with IBS and is more common in young women. The medications used to treat these patients are secretagogues. By affecting ion channels and epithelial receptors, these drugs increase the secretion of fluid substances into the intestinal lumen, which results in softer stools and easier bowel movements (24).

Lubiproston, a prostaglandin E1 derivative, acts selectively on ClC-2 chloride channels in enterocytes, increasing the secretion of water and chloride, thereby improving intestinal motility and transit. This drug has been approved by the U.S. Food and Drug Administration for the treatment of IBS at a dose of 8 μg twice daily for 12 weeks. If no improvement is observed, the drug should be discontinued. The efficacy of lubiprostone has been thoroughly studied in patients with chronic idiopathic constipation and opioid-induced constipation. The efficacy of treatment was assessed by the number of spontaneous bowel movements within a 24-hour period. In the group of patients with chronic idiopathic constipation, an increase in the number of spontaneous bowel movements per day was observed (RR 1.790; 95% CI 1.491–2.150), and in the group suffering from opioid-induced constipation (RR 1.277; 95% CI 1.105–1.475). At the same time, it was found that lubiproston did not affect the incidence of abdominal pain (25). Two independent studies have shown that lubiproston reduces the incidence of abdominal pain in patients with IBS-C (26) (27).

Tenapandor is an inhibitor of Sodium-Hydrogen Exchanger 3 (NHE3), a membrane protein found primarily in the intestinal wall and the proximal tubules of the kidneys. This medication reduces sodium reabsorption, which results in an increase in water within the intestinal lumen and, consequently, improves intestinal transit and softens stool consistency. All of these processes facilitate bowel movements, which is a key issue for patients with IBS-C (24). The efficacy of tenapandor was assessed based on improvements in total spontaneous bowel movements (CSBM) and the incidence of abdominal pain. A comparison of the results between the placebo group and the group receiving tenapandor at a dose of 50 mg twice daily revealed significant differences. In terms of CSBM, a treatment response was observed in 60.7% of patients taking tenapandor, compared with 33.7% ($P<0.001$) of patients taking placebo (28).

Eluksadoline is a medication used to treat Irritable Bowel Syndrome with Diarrhea (IBS-D). Eluksadoline is a μ- and κ-opioid receptor agonist and a δ-opioid receptor antagonist. It acts locally within the intestinal lumen, and its systemic bioavailability is low. By acting on opioid receptors, eluxadoline slows peristalsis and reduces secretion into the intestinal lumen, thereby leading to firmer stools (29). The researchers compared the efficacy of eluxadoline and placebo in improving stool consistency and reducing abdominal pain. An increase in stool consistency was observed in 31.1% of participants who took 75 mg of eluxadoline and in 36.8% of patients prescribed 100 mg daily, compared with 23.9% of patients taking a placebo. A reduction in abdominal pain was observed in 46.3% (for the 75 mg dose) and 48.3% (for the 100 mg dose) vs. 44.0% for the placebo (30). The targeted drugs for specific IBS subtypes mentioned above represent a promising direction in treatment (31).

Primary treatment options for irritable bowel syndrome (IBS) include low-dose antidepressants or antispasmodics, as well as anti-diarrheal or laxative agents to address transit disturbances. Nevertheless, these interventions frequently fail to provide adequate relief from abdominal pain (32).

Although the therapeutic potential of probiotics had already been investigated in 2007, current research enables to achieve more targeted and precise effect with specific probiotics (33).

Next-generation probiotics (NGPs) are a new type of microbial therapy composed of bacteria naturally found in the healthy human gut but not typically used as probiotics. Unlike traditional probiotics, NGPs are

selected for specific roles, such as supporting the intestinal barrier, regulating immune responses, producing beneficial metabolites, and influencing gut–brain communication (34).

In research, Faecalibacterium prausnitzii appears to be a highly promising candidate in IBS treatment. It is one of the major butyrate-producing bacteria in the normal gut microbiome. Butyrate, a short-chain fatty acid, supports colon health, anti-inflammatory responses, and gut barrier integrity. Moreover, reduced levels of F. prausnitzii are observed in several gut disorders, including IBS. The anti-inflammatory properties of this bacterium are mainly connected to butyrate production and the secretion of bioactive metabolites that suppress pro-inflammatory cytokines. Furthermore, butyrate may modulate nociceptive pathways, showing a possible role in reducing abdominal pain and hypersensitivity in IBS. However, the clinical application of F. prausnitzii is still problematic due to its sensitivity to oxygen, which limits its cultivation and formulation as a therapeutic product (35).

Another study shows that the Bacillus coagulans strain MTCC 5856 is also a beneficial probiotic for treating IBS, particularly diarrhea-predominant IBS (IBS-D). In a randomized, double-blind, placebo-controlled clinical trial, supplementation with B. coagulans MTCC 5856 resulted in noticeable improvements in key IBS symptoms compared to placebo. Participants reported reductions in abdominal pain, bloating, diarrhea, vomiting, and stool frequency, along with enhanced quality of life. The positive effects of this strain are linked to its ability to change gut bacteria, block harmful bacteria, strengthen the gut lining, and help control immune responses. B. coagulans MTCC 5856 also makes short-chain fatty acids like butyrate, which help keep the gut lining healthy and may lower inflammation and sensitivity. The study found that the supplement was safe, with no major side effects. These results suggest that B. coagulans MTCC 5856 could be a helpful extra treatment for people with IBS, especially those with IBS-D (36).

4. Conclusions

The patophysiology of IBS is very complex and not yet fully understood, however several studies bring us closer to better, more accurate treatment management of this condition. New perspectives may bring the introduction of NGPs. This bacteria strains may benefit in symptoms alleviation, on the other hand there are still some issues with their stability in oxygen condition. Other therapeutic agents such as prostaglandins or tenapandor may improve the severity of type C of IBS by enhanced CSBM. Moreover, prostaglandins change the consistency of stool and intestinal motility. The IBS type dominated by diarrhea may be managed by eluksadoline, which acts through opiod receptors. Its absorbability to the blood circulation stays on a low level, so it seem quite safe for usage in a wide range of patients.

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