Abdominal Pain in Irritable Bowel Syndrome (IBS)

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Key Points

- Irritable bowel syndrome (IBS) is the most common functional gastrointestinal disorder (FGID), affecting up to 15 % of the general population.
- It is characterized by chronic abdominal pain that can be mild and intermittent, or severe, constant, and debilitating. Pain in IBS, as in other chronic pain disorders, is a complex symptom resulting from the interplay between peripheral (visceral) stimulation (enteric nervous system) and central modulation (central nervous system).
- As the severity of pain increases central processing plays an increasingly important role compared to peripheral input. In IBS, the normal adaptive central inhibitory response to painful visceral stimuli is diminished. This change is modulated by psychosocial factors such as anxiety, depression, poor social support, and impaired coping skills.
- Successful treatment begins with a therapeutic doctor–patient partnership. Medical treatment of IBS includes peripherally acting and centrally acting agents with antidepressants playing a central role. Cognitive behavioral therapy (CBT), interpersonal (psychodynamic) therapy, hypnosis, stress reduction, and mindfulness meditation have been shown to be effective in the treatment of IBS.

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Introduction

Most patients who present with gastrointestinal symptoms have no clear organic cause even after an extensive investigation and are diagnosed with a functional gastrointestinal disorder (FGID). Among the FGIDs, irritable bowel syndrome (IBS) is the most common, affecting up to 15 % of the general population. The hallmark of IBS is chronic abdominal pain associated with irregular bowel movements. The pain can be mild and intermittent or severe, constant, and debilitating. IBS patients are major healthcare utilizers and are seen and treated not only by primary care physicians and gastroenterologists but also by surgeons, gynecologists, pain specialists, and rheumatologists. Thus, it is important for physicians in diverse subspecialties to be familiar with the diagnosis and management of this disorder.

The purpose of this chapter is to review the epidemiology and diagnosis of IBS and provide an in-depth look into the pathogenesis and treatment of pain in IBS patients.

Epidemiology

IBS is a common functional disorder with a symptom-based diagnosis (Rome III diagnostic criteria, Table 6.1) [1]. The reported prevalence of IBS varies from study to study depending on diagnostic criteria used as well as other methodological differences among studies [2]. However, some findings on the epidemiology of IBS appear to hold true and are as follows:

1. IBS is a global problem that affects individuals all over the world [3]. The reported worldwide prevalence rates for IBS range from 5 % to 20 %.
2. In most countries IBS affects women (60–70 %) more than men [4, 5]. The East is unique in that there are reports from China, Taiwan, and Singapore of a similar prevalence between males and females [6, 7]. There are conflicting reports from India with community-based surveys reporting

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Table 6.1 Rome III diagnostic criteria[a] for IBS

Recurrent abdominal pain or discomfort[b] at least 3 days/month in the last 3 months associated with *two or more* of the following:
1. Improvement with defecation
2. Onset associated with a change in frequency of stool
3. Onset associated with a change in form (appearance) of stool

[a]Criterion fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis
[b]"Discomfort" means an uncomfortable sensation not described as pain

higher prevalence of IBS among females in the general population and hospital-based surveys reporting higher proportion of males among patients in gastroenterology clinics [8, 9]. The latter observation might reflect cultural aspects of healthcare-seeking behaviors in Indian society.

3. Although IBS can appear at any age, it is more common in young and middle-aged patients and tends to be less common in the elderly [10, 11].
4. Socioeconomic status may play a role in the epidemiology of IBS, which has been reported in some countries to be more prevalent in lower socioeconomic classes [4, 12, 13], although the data on this factor are not consistent.

As a prevalent chronic disorder, IBS places a major economic burden on health care. A meta-analysis of 18 studies from the USA and the UK estimated the annual direct cost of an IBS patient (drugs, procedures, and doctor visits) at $348–8,750 and the annual indirect costs (loss of work days and deceased productivity) at $355–3,344 [14, 15]. Another US study estimated the overall annual direct cost of IBS to be $228 million in doctor visits and $80 million in drugs [15].

Diagnosis

There is no specific diagnostic finding or biomarker for IBS, so the diagnosis is based on patients' reports of their symptoms. In the past, IBS was considered a diagnosis of exclusion, but inherent to this approach is an exhaustive diagnostic work-up that involves unpleasant and potentially risky tests for the patient and is not cost effective. Thus, a symptom-based diagnostic system, known as the Rome criteria, was developed. The main concept introduced by the Rome criteria is that the diagnostic process of a functional disorder should be based on two components. The first is the presence of a typical cluster of symptoms and the second is the absence of "red flags" including initial presentation of symptoms at an age over 50, unexplained weight loss, fever, nocturnal symptoms, blood in the stool, a family history of gastrointestinal malignancy or disease (e.g., celiac or inflammatory bowel disease), or an abnormal finding on physical examination. Basic laboratory tests, such as a complete blood count and celiac serology, are usually enough to complete the diagnostic process and establish a firm diagnosis. Patients who fulfill the criteria and do not have red flags need a minimal diagnostic work-up after which the diagnosis of IBS can be made with confidence [16, 17].

The latest update of the Rome diagnostic criteria for IBS is Rome III, in which the diagnosis of IBS requires the presence of abdominal pain or discomfort for at least 10 % of the time over the previous three months with symptom onset at least six months earlier [18]. Additionally, pain should be relieved by defecation and associated with a change in the frequency of bowel movements or a change in the form of the stool. Accompanying symptoms, although not essential for the diagnosis, are a feeling of incomplete evacuation, abnormal stool frequency (less than three times a week or more than three times a day) or consistency, straining at defecation, urgency, mucus discharge, and bloating. IBS can be further divided into three main subgroups according to bowel habit as constipation predominant (IBS-C), diarrhea predominant (IBS-D), and those exhibiting an alternating bowel pattern [19]. Patients may switch from one subclass to another during the course of their illness. It has been demonstrated repeatedly that the use of positive symptom-based diagnostic criteria in conjunction with the use of red flags to guide further investigation in selected cases is a reliable and cost-effective approach. After establishing the diagnosis of IBS, based on the Rome criteria, it is rarely necessary to change the diagnosis [20–22].

The Pathophysiology of Pain in IBS

Abdominal pain is a hallmark of IBS and is essential for its diagnosis. In IBS, as in many other chronic pain syndromes, pain is a complex experience resulting from the interplay between peripheral (visceral) stimulation (enteric nervous system) and central modulation (central nervous system [CNS]).

Afferent stimulation from the colon is transmitted to second-order neurons in the spinal cord and then ascends to the brain through the spinothalamic, spinoreticular, and spinomesencephalic tracts. These tracts connect to the somatosensory cortex responsible for registration and localization of painful visceral and somatic stimuli. They also connect to structures in the limbic system that are involved in the reflexive, affective, and motivational responses to pain [23]. The afferent pathways project to the perigenual anterior cingulate cortex (pACC), which is involved in affective modification, and to the midcingulate cortex (MCC), which is involved in the behavioral response.

The amplification of afferent visceral stimulation can result from increased excitability of peripheral receptors or impaired spinal and/or central pain regulatory systems. Increased excitability can produce the two related phenomena

of hyperalgesia (increased pain response to painful stimuli) and allodynia (increased pain response to nonpainful stimuli) [24]. Thus, afferent visceral stimulation can be experienced as painful not only as a result of peripheral intensity but also as a result of central processing that may be modulated by psychosocial factors such as anxiety, depression, poor social support, and impaired coping skills [25]. As the severity of pain increases central processing plays an increasingly important role compared to peripheral input. Once a pattern of central sensitization has taken hold, patients may even experience severe pain without ongoing peripheral nociceptive stimulation [26, 27]. This is the extreme end of the IBS severity spectrum.

While we do not have full knowledge of all the causes of excessive peripheral stimulation, there is good evidence that eating, infection, inflammation, physical injury, hormones (e.g., menses), or colonic motility may play a role.

Up to 15 % of IBS patients attribute the beginning of their symptoms to an acute episode of gastrointestinal infection. A meta-analysis of eight papers including almost 600,000 patients over a follow-up up to one year found that the odds ratio for developing IBS after such an episode is seven [28]. IBS that follows acute intestinal infection has been shown to be associated with a persistent or chronic state of inflammation that cannot be identified by routine clinical tests and procedures [29, 30].

Risk factors for postinfectious IBS are related to not only to the severity of the acute infectious episode (fever, bloody stools, and need for hospitalization) but also to patient characteristics such as female gender, stress, anxiety, and depression [31]. This is a good example of how excessive afferent stimulation, induced in this case by a microinflammatory state, can develop into a chronic condition such as IBS-D after central sensitization occurs in a susceptible person with psychological comorbidity.

Peripheral stimulation and its interplay with central amplification are also reflected in the development of chronic abdominal pain following abdominal or pelvic surgery. IBS patients reported up to twice the number of appendectomies and hysterectomies and up to three times the number of cholecystectomies compared with those without IBS [32]. Surgery may cause visceral afferent sensitization that eventually results in allodynia and chronic pain even in the presence of normal gut function.

This contention is supported by a study that evaluated the development of abdominal pain after elective gynecologic surgery for nonpainful indications [32]. Patients with no prior history of chronic abdominal pain undergoing gynecological surgery for nonpainful indications were followed for the development of de novo abdominal pain following surgery. They were compared with a control group comprised of nonsurgical patients who came to a gynecologic clinic for nonpain-related reasons. At one-year follow-up significantly more patients in the surgery group complained of chronic abdominal pain (15.3 %) than in the control group (3.6 %, $p=0.003$). There was no association between any surgery-related variables and the subsequent development of chronic abdominal pain. The only predictors of chronic abdominal pain at one-year follow-up were associated with the patients' preoperative psychological profile. Patients anticipating difficulty with surgery or recovery from it and those with lower scores on the Sense of Coherence questionnaire (an index of coping skills) were more likely to develop chronic postoperative abdominal pain. In these cases, the interplay of peripheral visceral stimulus together with central sensitization related to psychosocial variables affected the de novo development of chronic abdominal pain.

Studies using functional MRI and PET CT have demonstrated that the ACC, which is responsible for descending pain inhibition, is less active in IBS patients. This phenomenon is also found in other chronic pain syndromes such as fibromyalgia [33–35]. In contrast, the MCC, which is associated with unpleasantness and fear, is overactive. Therefore, in IBS patients the normal adaptive inhibitory response to painful visceral stimuli is diminished and replaced by a maladaptive, presumably even aggravating, response [33, 34, 36]. The factors that ultimately lead to this shift into a maladaptive pattern are psychosocial in nature. This connection was elegantly demonstrated in the case report of a patient with a severe functional gastrointestinal pain syndrome and a history of abuse [37]. Her baseline brain scan demonstrated marked activation of the MCC and the somatosensory cortex. Following successful treatment with antidepressants and psychotherapy a repeated scan demonstrated diminished MCC activity and increased insular activation. Thus, maladaptive brain responses are reversible and so is the patient's clinical situation.

Treatment of Abdominal Pain in IBS

As in other fields of medicine, in particular in patients with chronic painful conditions, the healing process for IBS patients begins when the patient enters the doctor's office before any medicine has been prescribed. It is of the outmost importance to establish a good doctor–patient relationship in order to succeed in the therapeutic process [38, 39]. Some of the essentials of a salutary doctor–patient relationship are discussed below:

1. Allow enough time especially for the first meeting. The patient should feel that the doctor is listening to and him/her and that their symptoms are considered legitimate and are being taken seriously.
2. Take a full detailed history and perform a physical examination: These basic measures of good clinical practice help to foster the doctor–patient relationship.

3. It is very helpful to remember four key questions that patients should be asked:
   a. What brings you here at this time? IBS is a chronic condition and many patients have their symptoms for years before consulting a specialist. Consultation is often driven by a specific anxiety or a stressful situation that should be addressed.
   b. What do you think is the cause of your symptoms? Many IBS patients attribute their symptoms to undiagnosed cancer, infection, inflammatory bowel disease, or food allergy.
   c. What are your concerns or worries? It is important to understand the patient's agenda and to address their primary concerns such as "What exactly do I have?" or "Do I have cancer," or alternatively related to the symptoms like "I can't deal with this pain anymore."
   d. What are your expectations from me? Some patients have the unrealistic expectation of a "quick fix" for their situation that can lead to mutual frustration and treatment failure [40]. It should be emphasized that treating IBS is a process rather than an isolated consultation and that the goal of treatment is to reduce their suffering and to improve their quality of life rather than to "cure" them.

Many IBS patients have never received a comprehensive explanation about the nature of their problem. This may be the basis for the unwarranted fears ("I might have cancer") and feelings of frustration ("why can't they figure out what I have"). A detailed explanation about the nature of functional disorders and their natural history is very important to deal with these issues.

Treating IBS patients is an ongoing process that takes time. Throughout this process patients are likely to encounter difficulties, setbacks, and frustration. Patients should not feel that they are left alone to deal with their setbacks. Scheduling a follow-up phone call, for example, is a simple measure that is often sufficient to allay patients' new concerns [41]. Physicians should inquire about comorbid gastrointestinal and nongastrointestinal functional disorders. IBS patients have a high prevalence of other functional disorders [42], leading some patients to feel that they are very ill. By providing patients with a unifying paradigm that connects different, apparently unrelated, symptoms to one disorder (i.e., central sensitization), we can alleviate much of their fears and concerns.

For some patients with mild symptoms, these steps may be enough to alleviate fears and concerns regarding their symptoms. These patients often continue to cope successfully with their symptoms and need no further treatment. However, the majority of patients will require more specific treatment.

The treatment options for IBS can be divided into pharmacological and nonpharmacological treatment modalities (Fig. 6.1).

Medical Treatment

Medical treatment of IBS includes peripherally acting agents and centrally acting agents.

Peripherally Acting Agents

These drugs act on the gut itself and are targeted against specific IBS symptoms such altered bowel movements, bloating, and cramps. Because they are not key agents in

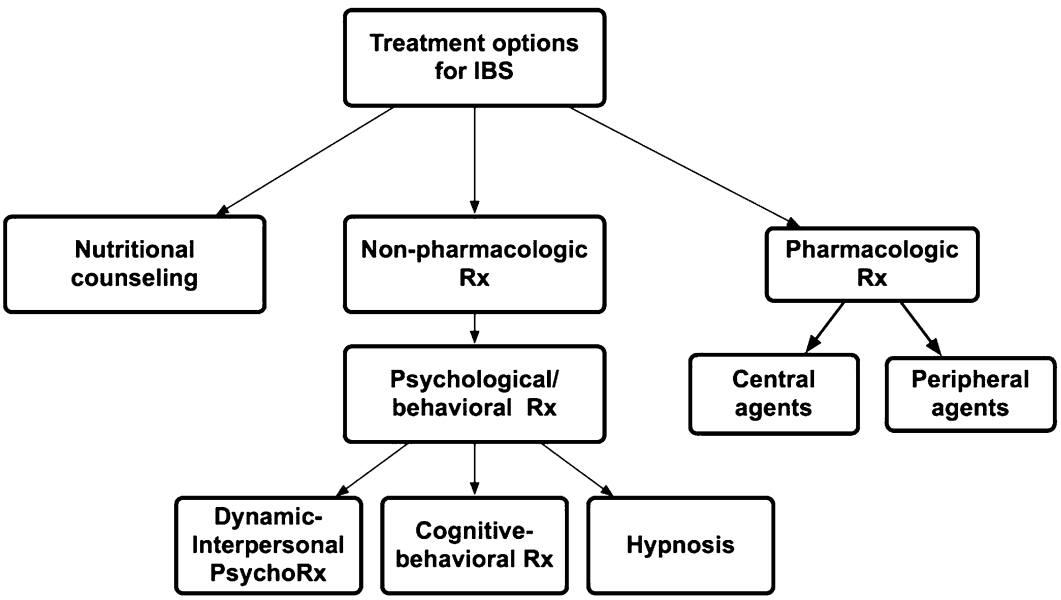

Fig. 6.1 Treatment options for IBS in addition to a therapeutic doctor–patient therapeutic partnership. Although there is not cure for IBS, a large number of treatment options are available to reduce suffering and improve quality of life. Doctors need not feel "empty handed" when coming to treat these patients

Table 6.2 Peripheral agents used most commonly in the treatment of IBS. Peripheral agents, although not primarily directed against pain, have an important role in IBS treatment. In mild IBS cases, they might suffice but in more severe IBS cases and, where pain is a cardinal symptom, central agents are preferred

Class	Drug	Mechanism of action	Comments
• Antispasmodics	• Pinaverium • Mebeverin • Colpermin (peppermint oil) • Hyoscamine dicyclomine	• Direct visceral smooth muscle relaxants • Anticholinergic/antimuscarinic	• Modest effect on IBS spastic pain • Otilinium bromide, hyoscine, and colpermin; best evidence for effectiveness
• Serotonergic and other agents	• Alosetron • Tegaserode	• 5HT3 receptor antagonist • 5HT4 receptor agonist	• Available only through a restricted access program; increased incidence of ischemic colitis • Withdrawn from the US market; an increased incidence of cardiovascular adverse events
	• Linaclotide	• Guanylate cyclase-C agonist	• Recently approved in Europe and the US for IBS-C
	• Lubiprostone	• Chloride channel activator	• In phase 3 studies, lubiprostone was almost twice as effective for IBS symptoms as placebo

IBS pain management only some of them are discussed in detail and the rest is mentioned briefly. Table 6.2 summarizes the main facts about the different peripheral agents. Serotonin (5HT) is an important neurotransmitter that coordinates gut function and has played a key role in research and drug development. It is secreted from enterochromaffin cells in the mucosa and is involved in almost every aspect of gut function including motility, sensation, and secretion. Alosetron is a 5HT3 receptor antagonist that was shown to improve global IBS symptoms and pain in women with IBS-D. A meta-analysis comparing 12 randomized controlled trials that evaluated the efficacy of alosetron compared to placebo found an odds ratio of 1.85 for improvement in the alosetron group [43]. Unfortunately, after initial FDA approval, safety issues and in particular ischemic colitis and severe constipation led to its withdrawal from the market. It was reintroduced in 2002 under a restricted access program. Under this program, alosetron can be prescribed (under some restrictions) to women with severe IBS-D who have failed to respond to traditional medical therapies.

Lubiprostone is a chloride channel activator that has been approved by the FDA for chronic constipation and IBC-C. In phase 3 studies, patients receiving lubiprostone were almost twice as likely to gain relief from overall IBS symptoms compared to patients who received placebo [44]. The main side effect of lubiprostone, nausea, is reported in 8 % of IBS-C patients who receive 8 mcg twice daily.

Centrally Acting Agents

Centrally acting agents should be the cornerstone of treatment in moderate-to-severe cases of IBS [45]. The main classes of drugs that are being used are the selective serotonin reuptake inhibitors (SSRIs), selective serotonin-norepinephrine reuptake inhibitors (SNRIs), and tricyclic antidepressants (TCAs). Other drugs, such as Mirtazapine, Buspiron, and the atypical antipsychotic Quetiapine, can also be used. These drugs were developed for the treatment of anxiety and depression, but can and should be used in IBS as discussed below. The different drugs and dosages are summarized in Table 6.3.

Antidepressants play a central role in medical therapy for IBS for two main reasons. First, they have a direct analgesic effect and are used in various pain syndromes, with or without concomitant depression, to elevate pain thresholds via central and peripheral effects. Second, since many IBS patient have psychological comorbidity, they can gain direct benefit from these drugs. Whether the main effect of antidepressants stems from central mechanisms (modulation of central pain processing) or from peripheral effects (effects on motility and secretion and reduction of afferent pain signals) or just from reducing depression and anxiety is still uncertain. The actual mechanism is probably a combination of all three. A recent meta-analysis found all classes of antidepressants to be effective in IBS with a number needed to treat as low as four [46].

Antidepressants in IBS (especially TCAs) are given at much lower doses then those used for the treatment of depression. The usual starting dose in 25–50 mg and can be increased as needed. SSRIs and SNRIs are usually given in the lower range of the "regular" psychiatric doses, for example, 10–20 mg of escitalopram or 30 mg of duloxetine.

Since TCAs and SNRIs have an independent indication in other pain syndromes, such as neuropathic pain and fibromyalgia, they are the drugs of choice for painful IBS. The choice between them is often based on the therapeutic profile of the drugs including potential adverse effects.

Table 6.3 Common interventions used in IBS. For optimal results these interventions can be used in combination ("augmentation" therapy). The use of more than one drug at a low dose can augment the therapeutic response and minimize the side effects

Drug	Drug (daily dose range [mg])	Comments
TCA	• Desipramine (25–150) • Nortriptyline (25–150) • Amitriptyline (25–150)	• Begin with low dose and titrate by response • Allow 4–8 weeks for maximal response
SSRIs	• Paroxetine (20–60) • Escitalopram (10–20)	• Begin with low dose and titrate by response
SNRIs	• Venlafaxine (25–300) • Duloxetine (20–80)	• Psychological and analgesic effects
Atypical antipsychotics	• Quetiapine (25–100)	• Preliminary reports
Tetracyclic antidepressant	• Mirtazepine (15–45)	• Antiemetic properties
Azaspirodecanediones	• Buspiron (10–60)	• Improves gastric receptive relaxation

For example, TCAs tend to be more constipating and have less anxiolytic properties, so an SNRI would be the preferred option in a patient with constipation or prominent anxiety. However, in many cases a combination of two drugs or more is necessary. Instead of increasing the dose of a single drug to the maximum, the use of a combination of two or more drugs from different classes and in lower doses (e.g., a TCA and an SNRI or SSRI) is recommended. This approach known as "augmentation therapy," helps minimize adverse effects, to which patients with functional GI disorders are prone [45].

Mirtazapine is a tetracyclic antidepressant used primarily in the treatment of depression. It has serotonergic as well as noradrenergic properties. It has antagonistic alpha-2 receptor and 5HT1, 5HT2, and 5HT3 properties as well as moderated peripheral alpha-1 adrenergic and alpha-1 anticholinergic properties. Its 5HT3 antagonistic action is probably responsible for its antiemetic properties. In addition to its antidepressant effects, it is also used at times as a hypnotic, antiemetic, as an appetite stimulant, and for the treatment of anxiety. In IBS, it can be used to augment the antidepressant and anxiolytic properties of other agents (such as a TCA or an SNRI) and for nausea and vomiting or low body weight, as is often seen in patients with a comorbid eating disorder. Data regarding its use in IBS are limited and more studies are needed to explore its exact place.

Quetiapine is an atypical antipsychotic approved for the treatment of schizophrenia, bipolar disorder, and as an add-on to treat depression. It has potential benefits in IBS by reducing anxiety, restoring normal sleep patterns, and potentially through a direct analgesic effect. A recent paper reported a retrospective analysis of its use in low doses (50–200 mg) in patients with severe FGIDs. Of the 21 treated patients, 10 discontinued the drug due to adverse effects or lack of efficacy, but of the 11 patients who stayed on the drug 6 reported improvement [47]. Although this is a small and uncontrolled study, it is encouraging considering that these were patients with extremely severe IBS who did not respond to any previous treatment modality. A larger, prospective, open-label study is currently underway.

Finally, Buspirone is a nonbenzodiazepine anxiolytic agent that is used in psychiatry to augment the effect of antidepressants. It also has a 5HT1 agonist effect, which may contribute to increasing gastric compliance/relaxation as has been shown to occur for functional dyspepsia. Therefore, it might be useful in patients with comorbid dyspeptic symptoms such as epigastric discomfort and early satiety.

There are two main barriers that clinicians face when trying to treat IBS patients with antidepressants. The first is the general reluctance of these patients to take "chemical" and "mind altering" agents. The second is patients' tendency to underestimate the psychological component of their symptoms. A thorough explanation regarding the mechanisms of pain (visceral hypersensitivity modulated by central mechanisms) and the drug's independent analgesic properties is enough in many cases. Some patients view the recommendation for a psychotropic drug as evidence that the doctor does not acknowledge their pain and thinks that they are "crazy." If we emphasize that we are recommending these drugs for their central analgesic effect, we can overcome much of this reticence to take them. This can be accomplished with a statement such as: "The same drug can be used for different reasons. For example, in the past aspirin was the leading drug for reducing fever and relieving pain, but currently it is the number one drug for the prevention of heart disease. Similarly, antidepressant drugs are effective in the treatment of depression at higher doses, but are also effective in lower dosages for pain relief". The patient should always make the final decision regarding the drug. This can be achieved by fostering a feeling of therapeutic partnership instead of an authoritative relationship where the patient has no say about the way he is treated. An example for such an approach would be: "In IBS there are many therapeutic options, with and without drugs. Each has its advantages and disadvantages. Do you want me to tell you about options that could help you with your symptoms?" By making the drug the

patient's choice, we can augment adherence to treatment. Finally, in our experience, the adherence rate for drug therapy increases if the physician is available to address, in real time, early adverse effects, and other concerns that otherwise may lead the patient to discontinue therapy on their own.

Nonpharmacologic Therapy for IBS

Nonpharmacological treatments for IBS include stress reduction, and behavioral and psychological interventions.

Behavioral Interventions

Behavioral interventions are commonly used to treat IBS. They are safe and their benefit may go beyond symptomatic treatment and induce positive physiological changes. They are particularly suited to patients who do not want to take drugs. The effect of different modalities, including cognitive behavioral therapy (CBT), interpersonal (psychodynamic) therapy, hypnosis, stress reduction, and mindfulness meditation, has been evaluated for IBS. All help patients deal with issues such as maladaptive illness beliefs and behaviors, and the relationship between stress, life events, and symptomatology.

CBT can help patients recognize misperceptions and maladaptive thoughts regarding their symptoms and enhance their coping abilities. It can be administered as individual or group therapy [48–50]. In the largest randomized placebo-controlled study conducted to date, the investigators found that 12 weekly CBT sessions were significantly more beneficial than placebo for female patients with moderate-to-severe FGIDs [51].

Interpersonal (psychodynamic) therapy presumes that symptoms are associated with difficulties in interpersonal relationships. Its focus is on the identification of interpersonal situations that lead to symptom exacerbation. The treatment itself involves psychotherapy. The symptoms improve when the conflicts are resolved. Interpersonal dynamic psychotherapy has been shown to improve symptoms and to reduce disability and healthcare costs in IBS [52–54].

The aim of stress reduction (relaxation training) is to counteract the physiologic effects of stress. Reduction in skeletal muscle tension can decrease autonomic arousal and subjective tension/anxiety and may improve gut motility. Stress reduction and relaxation training includes modalities such as guided imagery, relaxation response, meditation, yoga, and biofeedback. Muscle relaxation alone or in combination with CBT and other techniques was shown to reduce IBS symptoms [55]. Mindfulness meditation is a form of relaxation involving an active nonjudgmental awareness of body sensations and emotions. Group mindfulness meditation resulted in improved IBS symptoms and health-related quality of life as well as reduced stress levels in women with IBS [56], effects that persisted at a three-month follow-up assessment.

Hypnosis is a form of guided imagery that uses muscle relaxation and gut-targeted suggestions to improve the gut function and reduce symptoms. Hypnosis involves nonspecific effects of relaxation, stress management, ego strengthening, and gut-directed suggestions of normal functioning and pleasant feeling. Data gathered from studies in different centers support the use of hypnosis as an effective, viable treatment option in IBS [57] that improves IBS symptoms and quality of life and reduces stress and anxiety. Moreover, the beneficial effects of hypnosis have been shown to persist at long-term follow-up [58–60].

The predictors of a favorable outcome in behavioral interventions include confidence in treatment success, perceived sense of control over symptoms, a good relationship with the therapist, and early response [61]. The choice of intervention depends on local expertise and availability as well as patient preference.

Summary and Conclusions

IBS is a common medical problem, which, although not life threatening, has a significant negative impact on patients' quality of life. Its range of severity ranges from mild intermittent symptoms to a disabling condition with a considerable loss of daily function. Pain in IBS is the result of peripheral afferent stimulation and CNS processing. A biopsychosocial perspective, taking into account the patient's psychological status, life experiences, beliefs, and concerns can help doctors provide optimal care. The primary goal of treatment is care rather than cure, and the various treatment options can be highly effective in reducing suffering and improving quality of life. The doctor–patient relationship is the foundation of successful treatment and should be supplemented by pharmacological or nonpharmacological treatments in accordance with the clinical situation and the patient's preference.

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