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REVIEW ARTICLE

Overlap of irritable bowel syndrome with other disorders of gut-brain interaction and other comorbidity

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Abstract

Irritable bowel syndrome (IBS) is a disorder of gut–brain interaction (DGBI) with high prevalence and a significant impact on quality of life. Its frequent overlap with other DGBI such as functional dyspepsia, functional constipation and gastroesophageal reflux disease, as well as with extra-digestive comorbidity including fibromyalgia, chronic fatigue syndrome and endometriosis, represents a relevant clinical challenge. This coexistence is associated with a higher symptom burden, poorer prognosis, fluctuating clinical course, and lower response to conventional treatment. DGBIs share pathophysiological mechanisms such as visceral hypersensitivity, dysmotility, intestinal dysbiosis, dysfunction of the gut–brain axis, and neuroinflammation, which explain their frequent overlap. Evaluation must be comprehensive, taking into account symptoms across different anatomical regions, psychological comorbidities, and the dynamic evolution of subtypes, always prioritizing the most disabling symptom. Treatment should be guided by the predominant symptom axis and combine pharmacologic strategies such as neuromodulators with psychosocial interventions, cognitive behavioral therapy, and lifestyle-based approaches. Overlaps with gynecological and systemic conditions such as interstitial cystitis or bladder pain syndrome and endometriosis require a multidisciplinary approach. Recognizing and addressing IBS overlap with other disorders helps avoid fragmented treatment, improves quality of life, and reduces unnecessary use of healthcare resources. This patient-centered approach, grounded in shared mechanisms, is essential for effective, empathetic, and evidence-based care.

Keywords: Irritable bowel syndrome. Visceral hypersensitivity. Comorbidity. Gut-brain axis. Quality of life.

Sobreposición del síndrome de intestino irritable con otros trastornos de la interacción intestino-cerebro y otra comorbilidad

Resumen

El síndrome de intestino irritable (SII) es un trastorno de la interacción intestino-cerebro (TIIC) con elevada prevalencia y un impacto significativo en la calidad de vida. Su frecuente sobreposición con otros TIIC, como dispepsia funcional, estreñimiento funcional y enfermedad por reflujo gastroesofágico, así como con comorbilidad extradigestiva como fibromialgia, síndrome de fatiga crónica y endometriosis, representa un reto clínico relevante. Esta coexistencia se asocia a mayor carga sintomática, peor pronóstico, fluctuación clínica y menor respuesta al tratamiento convencional. Los TIIC comparten mecanismos fisiopatológicos, como hipersensibilidad visceral, dismotilidad, disbiosis intestinal, disfunción del eje intestino-cerebro y neuroinflamación, que explican su frecuente solapamiento. La evaluación debe ser integral, considerando síntomas en distintas regiones anatómicas, comorbilidad psicológica y evolución dinámica de los subtipos, priorizando siempre el síntoma más incapacitante.

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El tratamiento debe orientarse al eje sintomático predominante y combinar estrategias farmacológicas, como neuromoduladores, con intervenciones psicosociales, terapia cognitivo-conductual y abordajes centrados en el estilo de vida. Las sobreposiciones con enfermedades ginecológicas y sistémicas, como la cistitis intersticial o síndrome de vejiga dolorosa y la endometriosis, exigen un enfoque multidisciplinario. Reconocer y abordar la sobreposición del SII con otros trastornos permite evitar tratamientos fragmentados, mejorar la calidad de vida y reducir el uso innecesario de recursos. Este enfoque centrado en el paciente, basado en mecanismos compartidos, constituye una vía fundamental para una atención efectiva, empática y sustentada en la evidencia.

Palabras clave: Síndrome de intestino irritable. Hipersensibilidad visceral. Comorbilidad. Eje intestino-cerebro. Calidad de vida.

Introduction

Irritable bowel syndrome (IBS) is a disorder of gut–brain interaction (DGBI), symptom-based, defined by the Rome IV criteria, and characterized primarily by recurrent abdominal pain and alterations in stool frequency or consistency[1]. Its global prevalence varies widely, from as low as 1% in France to nearly 35% in Mexico. It is estimated that between 5% and 10% of the Western population is affected[2,3].

Beyond its high prevalence, IBS exerts a considerable impact on quality of life. Although historically considered a functional disorder without structural basis, it is now recognized that multiple pathophysiologic mechanisms underlie its clinical expression, explaining its frequent coexistence with other digestive and extra-digestive disorders.

In this context, the concept of overlap refers to the simultaneous presence of symptoms or diagnostic criteria for 2 or more DGBI in the same patient. This phenomenon may include digestive conditions such as functional dyspepsia (FD) or functional constipation (FC), as well as extra-digestive comorbidities such as fibromyalgia (FM), chronic fatigue syndrome (CFS), headache, sleep disorders, and urogenital conditions.

This article reviews the overlap of IBS with other DGBI and associated comorbidities, emphasizing its clinical relevance, shared pathophysiologic foundations, and management strategies.

Definition of overlap in gastroenterology

In gastroenterology, the term “overlap” refers to the coexistence of symptoms or diagnostic criteria corresponding to 2 or more DGBI in the same patient. This phenomenon is not uncommon and represents both a diagnostic and therapeutic challenge, associated with increased health care costs and a significant deterioration in quality of life.

Overlap also poses challenges for research and clinical practice, as patients often present with symptoms that cannot be explained by a single diagnosis. Therefore, an individualized, multidimensional, patient-centered approach is required, addressing both digestive and extra-digestive aspects[4].

Clinical importance of overlap in irritable bowel syndrome

Overlap among different DGBI and other comorbidities is a frequent finding in patients with IBS and has substantial clinical implications. A significant proportion of these patients meet criteria for more than one DGBI, present with extra-digestive comorbidities, or experience symptoms in different anatomic regions (Fig. 1).

The Rome Global Epidemiology Study[5] reported that more than 30% of the general population meeting criteria for a DGBI under Rome IV had symptoms in 2 or more anatomic regions. In tertiary care settings, this figure may exceed 47%, while in the general population it approaches 26.5%. This suggests that patients managed in specialized units tend to present more complex and refractory conditions.

Overlap of DGBI is associated with greater severity of GI symptoms, poorer quality of life, higher psychological burden (anxiety, depression, somatization), and lower response to first-line therapies. For example, patients with IBS and FD often experience greater symptom intensity and higher emotional burden compared with those presenting a single DGBI. This clinical complexity reinforces the need to identify overlap from the initial evaluation and to adapt the diagnostic–therapeutic approach in an integrated manner, considering all symptomatic axes involved.

In Mexico, the SIGAME Study (Síntomas Gastrointestinales en México)[6] represents the first population-based epidemiologic assessment focused on functional GI disorders, including a specific analysis of overlap. This study, conducted in more than 3,900 adults from diverse geographic regions and socioeconomic strata, showed a prevalence of IBS of 7.6%, while FC and uninvestigated dyspepsia reached

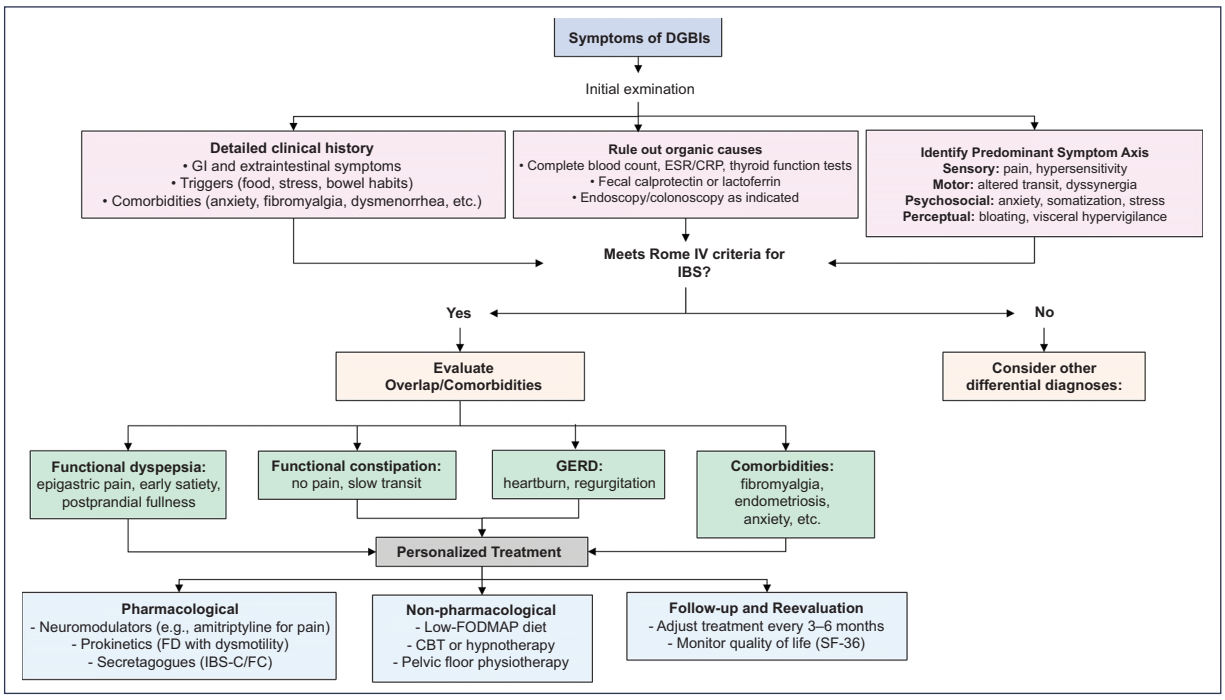

Figure 1. Clinical management algorithm for patients with symptoms compatible with disorders of gut–brain interaction (DGBIs) and possible overlap. FD: functional dyspepsia; FC: functional constipation; GERD: gastroesophageal reflux disease; FM: fibromyalgia; FODMAP: fermentable oligosaccharides, disaccharides, monosaccharides, and polyols; CRP: C-reactive protein; IBS: irritable bowel syndrome; IBS-C: IBS with constipation; CBT: cognitive behavioral therapy; ESR: erythrocyte sedimentation rate.

prevalences of 22.3% and 12%, respectively. These data confirm that DGBI are highly prevalent in the Mexican population, and that a substantial proportion of patients experience symptoms consistent with more than one functional diagnosis.

In particular, the chapter dedicated to overlap in the SIGAME Study documented that the coexistence of IBS with other functional GI disorders is common. Overlap with symptoms of gastroesophageal reflux disease (GERD) occurred in 67 subjects with IBS (22.3%; 95% CI, 17.9-29.2), with dyspeptic symptoms in 54 subjects (17.9%; 95% CI, 14-22.6), and excessive belching in 37 subjects (12.2%; 95% CI, 9-16). These data reinforce the need for a comprehensive, multidimensional evaluation from the first clinical encounter.

Although the findings of SIGAME are consistent with those reported in international studies, they add the value of being based on a representative sample of the Mexican general population, evaluated with validated tools (Rome III, PAGI-SYM, PAGI-QoL). Furthermore, higher frequencies of overlap were identified among women, young adults, and individuals of working age, highlighting the need for diagnostic and therapeutic strategies sensitive to the sociodemographic context.

Incorporating these data into clinical practice strengthens a patient-centered approach, avoids unnecessary treatments and investigations, and favors interventions directed at the predominant symptomatic axis. This approach is essential for improving quality of life, reducing emotional burden, and optimizing the use of resources in health systems with structural limitations such as ours.

Pathophysiologic considerations

DGBIs share pathophysiologic mechanisms that may coexist and potentiate one another, facilitating chronicity and symptom severity. The most relevant include visceral hypersensitivity, motor dysfunction, low-grade immune activation, intestinal dysbiosis, increased epithelial permeability, and alterations in central pain modulation[1,7] (Table 1).

Visceral hypersensitivity

This refers to an exaggerated response to normal visceral stimuli, mediated by central and peripheral sensitization. It is common in IBS, FD, FC, and functional

Table 1. Shared pathophysiological mechanisms in disorders of gut–brain interaction and their clinical expression

Pathophysiological mechanism	Symptoms or clinical signs	Associated digestive disorders	Associated extra-digestive comorbidity
Visceral hypersensitivity	Abdominal pain, bloating, urgency, sensation of incomplete evacuation	IBS, FD, FC, FABD	FM, chronic pelvic pain
Motility alterations	Postprandial fullness and satiety, nausea, constipation, diarrhea	IBS, FD, FC, gastroparesis, GERD	Migraine, IC
Intestinal dysbiosis	Flatulence, bloating, abdominal discomfort, systemic symptoms	IBS, FD, SIBO, PI-IBS	CFS, anxiety, mood disorders
Low-grade inflammation	Persistent pain, hyperalgesia, nonspecific chronic symptoms	IBS, FD, PI-IBS	CFS, FM
Increased intestinal permeability	Abdominal discomfort, systemic inflammation, multi-organ symptoms	IBS, dysbiosis	Endometriosis
Gut-brain axis dysfunction	Anxiety, depression, insomnia, pain amplification, dysautonomia	All DGBIs	Mood disorders, sleep disorders
Central sensitization	Widespread pain, exaggerated response to mild stimuli	IBS, FD	FM, chronic headache
Altered neurological modulation	Circadian rhythm disturbances, appetite changes, hyperalgesia	IBS, FD, FC	Sleep disorders, anxiety, depression

IC; interstitial cystitis; FABD, functional abdominal bloating/distension; FD; functional dyspepsia; FC, functional constipation; GERD; gastroesophageal reflux disease; CFS; chronic fatigue syndrome; FM; fibromyalgia; SIBO, small intestinal bacterial overgrowth; IBS, irritable bowel syndrome; PI-IBS; post-infectious IBS; DGBIs, disorders of gut–brain interaction.

abdominal bloating[8]. Hyperalgesia may involve different digestive regions; for example, patients with FD often present with gastric hypersensitivity, whereas rectal hypersensitivity predominates in IBS[9,10].

Motility disorders

These may affect various segments of the GI tract, manifesting as delayed gastric emptying (in IBS and FD), slow colonic transit (in IBS with constipation [IBS-C] and FC), or hypocontractility. Such disorders may influence each other through reflex and neurohormonal mechanisms, such as interference of colonic transit with gastric accommodation[7]. In addition, fermentation products such as short-chain fatty acids may modulate motility and sphincter tone, exacerbating symptoms of GERD or bloating[5].

Intestinal dysbiosis

An imbalance of the luminal and mucosa-associated microbiota may impair the epithelial barrier, induce subclinical inflammation, and modify neuroimmune signaling of the gut-brain axis (GBA)[7,11]. These alterations are also associated with systemic manifestations such as fatigue, anxiety, or sleep disorders, particularly in patients with multiple DGBI or comorbidities such as FM[12,13].

GBA dysfunction

The GBA is a bidirectional network integrating the central nervous system, the enteric nervous system, the immune system, and the intestinal microbiota[8]. Its disruption may amplify pain perception, impair emotional regulation, and modulate autonomic responses, explaining the multisystemic expression of many DGBI[7,13] (Fig. 2).

Clinical course and symptom fluctuation

Overlap among DGBI leads to a more protracted clinical course, characterized by more persistent and longer-lasting symptoms, greater variability over time, reduced response to conventional therapies, and increased use of diagnostic and health care resources.

Moreover, the coexistence of multiple DGBI favors subtype fluctuation. In IBS, only 24% of patients maintain the same subtype over time, while more than 50% shift between IBS-C, IBS with diarrhea [IBS-D], and mixed IBS[14]. This symptomatic instability requires periodic clinical reassessment and a flexible therapeutic approach adapted to each patient's clinical course.

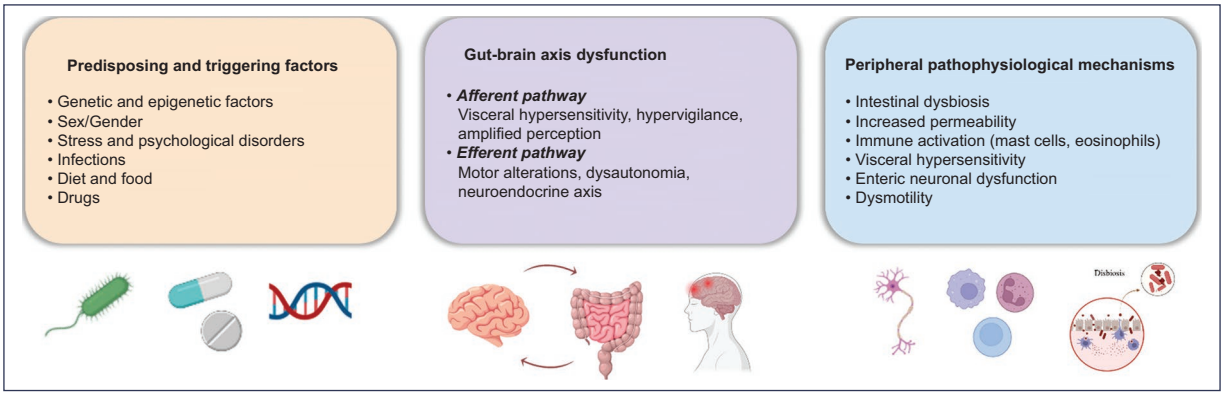

Figure 2. Pathophysiologic model of disorders of gut-brain interaction.

From the outset, the possibility of overlap should be considered. This involves a targeted history exploring symptoms in different anatomic regions, assessment of psychosocial comorbidity, identification of the predominant symptomatic axis, and prioritization of the most disabling symptom as a therapeutic guide.

Recognizing these patterns helps avoid fragmented treatments, reduce unnecessary interventions, and promote an integrated perspective aimed at improving patient quality of life.

Impact on the patients' quality of life

Overlap of IBS with other DGBI and extra-digestive comorbidity negatively affects multiple dimensions of quality of life, including physical, emotional, functional, social, and occupational aspects. This impact extends beyond digestive discomfort, affecting the patient's overall experience.

Several studies have shown that patients with multiple DGBI present with greater symptom severity, higher somatic burden, health-related anxiety, and more significant interference with daily activities. They also tend to use health services more frequently and report lower therapeutic satisfaction[15-18].

The number of DGBI correlates directly with poorer general health perception, greater work absenteeism, and reduced productivity. In the Rome Global Study[19], individuals with symptoms in multiple anatomic regions reported significantly lower scores across all SF-36 domains, particularly in physical, emotional, and functional dimensions.

Furthermore, the coexistence of visceral symptoms with somatization or psychological disorders increases the likelihood of meeting diagnostic criteria for mental disorders according to the Diagnostic and Statistical Manual of Mental Disorders. This fosters a negative feedback loop in which physical symptoms amplify anxiety and catastrophization, which in turn intensify pain perception[7,20,21].

Many patients also report feeling stigmatized or invalidated by medical personnel, which may lead to frustration, repeated health care seeking, unnecessary interventions, and poor treatment adherence. Lack of recognition of the complexity of their clinical presentation contributes to this perception.

For all these reasons, quality of life should be considered an explicit therapeutic goal. The approach must be integrative and patient-centered, including:

– Symptom relief directed at the predominant axis.
– Psychosocial interventions (eg, cognitive-behavioral therapy [CBT] or mindfulness).
– Empathetic validation of the patient's experience.
– Education about the chronic and multifactorial nature of the disorder.
– An interdisciplinary strategy with realistic goals, which may substantially improve health perception and reduce overall disease burden[2,22].

Diagnostic and therapeutic implications of IBS overlap

Recognition of shared pathophysiologic mechanisms among DGBI has important clinical implications. In practice, it is common for patients to present symptoms in more than one anatomic region or to meet criteria for multiple DGBI, which demands a more flexible and integrative diagnostic approach.

Diagnosis in the context of overlap

Although the Rome IV criteria are useful for classifying DGBI, their strict application may limit the identification of overlapping conditions. For example, a significant proportion of patients with FD also meet criteria for IBS, particularly in the subtypes of postprandial distress syndrome and epigastric pain syndrome, with overlap prevalence ranging from 17% to 55%[7].

A diagnostic approach should focus on:

– Detailed history exploring symptoms in different anatomic regions and their relationship with diet, stress, or defecation.
– Systematic evaluation of psychological or somatic comorbidity.
– Use of validated tools such as questionnaires or pictograms to facilitate communication between physician and patient.
– Identification of the predominant or most disabling symptom as the therapeutic target.

Studies have shown that a clinical diagnosis based on these elements is usually stable over time, with a low risk of missing relevant organic disease if the initial evaluation is adequate.

Clinical course and symptom fluctuation

As noted previously, DGBI are dynamic disorders with high temporal variability. In IBS, more than 50% of patients modify their subtype during follow-up. This instability requires periodic reassessment and individualized therapeutic adjustments according to symptom progression[14].

Therapeutic implications

Treatment should target the dominant pathophysiology and the most disabling symptomatic axis. Rigid or purely symptom-based approaches should be avoided. In many cases, a rational combination of therapies is more effective. For example, treating constipation in patients with IBS-C may also improve postprandial fullness in cases overlapping with postprandial distress syndrome.

The use of neuromodulators—such as tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), serotonin–norepinephrine reuptake inhibitors (SNRIs), or calcium-channel ligands—has shown efficacy in the management of pain, dysmotility, anxiety, and visceral hypervigilance. Recommendations include:

– Avoid using terms such as antidepressant or antipsychotic to reduce stigma and improve adherence.
– Start with low doses and adjust progressively.
– Avoid combining agents from the same class to reduce the risk of adverse effects, such as serotonin syndrome.

Additionally, some neuromodulators have proven beneficial in frequent comorbidities such as FM, migraine, or interstitial cystitis.

Role of mental health

Identifying affective symptoms is key to an effective therapeutic strategy. Referral to psychiatry or psychology is recommended in cases of moderate-to-severe affective disorders and in patients with persistent somatic symptoms. Non-pharmacologic therapies (eg, CBT, hypnotherapy, or mindfulness) should also be considered as part of an integrative treatment plan[2].

Overlap between IBS and other disorders of gut-brain interaction

IBS and FD

FD is the condition most frequently overlapping with IBS. Up to 64% of patients with FD also meet criteria for IBS, particularly IBS-D and postprandial distress syndrome[23,24]. This combination is associated with greater symptom severity, higher emotional burden, and poorer quality of life.

In a Pan-Asian survey[25], between 25% and 33% of IBS patients reported epigastric pain as their predominant symptom, forming an independent symptom cluster.

Both conditions share several pathophysiologic mechanisms, such as visceral hypersensitivity, duodenal dysfunction (with increased mast cells and eosinophils), dysbiosis, and an exaggerated gastrocolic reflex. Overlap may also occur as a post-infectious sequela after acute gastroenteritis.

Treatment should target the predominant symptom, and strategies include:

– Visceral neuromodulators (eg, amitriptyline, duloxetine).
– H1/H2 antihistamines in patients with allergic features or postprandial hypersensitivity.
– Prokinetics in cases with associated dysmotility.
– Low-FODMAP diet.
– Diaphragmatic relaxation techniques and psychosocial interventions.

IBS and FC

Differentiating IBS-C from FC can be challenging, as they share symptoms such as infrequent bowel movements, straining, excessive effort, and incomplete evacuation. The presence of recurrent abdominal pain supports IBS-C, whereas its absence favors FC.

Studies based on Rome III criteria have reported that 18-44% of patients meet criteria for both disorders[26,27], reflecting significant symptomatic overlap. Furthermore, the Rome Global Epidemiology Study found that nearly one-third of patients initially diagnosed with IBS-C or FC migrated to the other diagnosis over time, highlighting their dynamic nature[23].

From a pathophysiologic standpoint, slow colonic transit and pelvic floor dyssynergia are more frequent in FC, whereas visceral hypersensitivity predominates in IBS-C. However, these findings are not mutually exclusive: defecatory dyssynergia has been documented in 59% of FC patients but also in a relevant proportion of IBS-C patients. Similarly, slow transit is observed in 47% of FC and 22-30% of IBS-C patients[28].

Clinically, IBS-C/FC overlap is associated with poorer quality of life, higher anxiety and depression rates, and more frequent medical visits. These differences also affect treatment response. While secretagogues and prokinetics may benefit both groups, antidepressants and CBT appear more effective in IBS-C, whereas pelvic floor biofeedback is key for dyssynergia, typical of some FC cases[29].

Overall, IBS-C and FC should be regarded as related conditions within a shared clinical and pathophysiologic spectrum. Management should be individualized according to the presence or absence of pain, transit pattern, available physiologic testing, and the patient's psychological profile.

IBS and GERD

IBS and GERD overlap in up to 47% of cases, according to observational studies[23]. This coexistence is not only common but also clinically significant, as it is associated with greater refractoriness to conventional therapy and worse quality of life.

Both disorders share pathophysiologic mechanisms such as visceral hypersensitivity, segmental dysmotility, and psychosocial vulnerability[10]. Manometric studies have shown that colonic distension may induce transient lower esophageal sphincter relaxations, exacerbating GERD symptoms[30], suggesting a reflex interaction between different segments of the digestive tract.

Moreover, patients with IBS and GERD have higher prevalence of anxiety, somatic hypervigilance, and catastrophization, which amplifies symptom perception.
Treatment should address global GBA dysfunction, and strategies include:

- Neuromodulators such as duloxetine or low-dose tricyclic antidepressants.
- CBT or emotional regulation therapies.
- Specific dietary adjustments (eg, avoiding large meals and trigger foods).
- Optimization of antisecretory therapy in cases of erosive GERD.

It is essential to avoid unnecessary escalation of proton pump inhibitor therapy when symptoms persist despite endoscopic normalization and to consider functional mechanisms associated with IBS.

IBS and functional abdominal bloating

Abdominal bloating is a highly prevalent symptom in IBS patients, particularly those with IBS-C; it is estimated that up to 90% of IBS patients experience it at some point[31]. In many cases, visible distension is not related to objective gas accumulation but rather to motor disturbances, diaphragmatic dysfunction, or abnormal somatic perception.

Studies using abdominal plethysmography and magnetic resonance imaging have shown that functional distension may result from a dyssynergic breathing pattern, with paradoxical diaphragmatic descent during inspiration and secondary abdominal protrusion. This alteration may be unconscious and reflect a component of somatization or visceral hypervigilance.

From a pathophysiologic standpoint, impaired intestinal accommodation to normal volumes of gas or luminal contents has also been proposed, exacerbated by segmental dysmotility and alterations in the microbiota.
Therapeutic approaches include:

- Diaphragmatic breathing training with abdominal biofeedback.
- Postural and breathing pattern re-education therapies.
- Neuromodulators in cases with predominant visceral hypersensitivity.
- Psychosocial interventions to reduce excessive somatic attention (eg, CBT or mindfulness)

Patient education regarding the functional pathophysiology of bloating and validation of their experience are essential to improve adherence and reduce symptom-related anxiety.

Extra-digestive comorbidity

IBS and IBD

Although IBD and IBS are distinct entities, up to 30-40% of patients with IBD in clinical remission present IBS-like symptoms. This situation is more frequent in ulcerative colitis and represents a significant diagnostic and therapeutic challenge[7,32].

In these cases, functional symptoms may be due to persistent alterations of the intestinal mucosa, even in the absence of evident active inflammation. Frequent findings include:

– Visceral hypersensitivity assessed by barostat, associated with higher mast cell density and overexpression of receptors such as TRPV1.
– Subclinical inflammation, with elevated fecal calprotectin, intraepithelial lymphocyte infiltration, and increased proinflammatory cytokines (eg, TNF-α), despite apparent clinical and endoscopic remission[33].
– Post-inflammatory neuroimmune changes that perpetuate visceral sensitization through persistent plasticity in afferent pathways[32,34]

Clinically, it is crucial to distinguish functional symptoms from an inflammatory flare. Biomarkers (eg, fecal calprotectin), imaging, and, when necessary, endoscopy are recommended.

Recognition of IBS–IBD overlap prevents unnecessary use of immunosuppressants or biologics and allows management to focus on strategies targeting functional symptoms, such as:

– Low-FODMAP diet.
– Intestinal neuromodulators.
– Psychosocial approaches such as CBT or hypnotherapy

This integrative strategy improves symptom control and reduces both the emotional and medical burden of patients[35].

IBS and urogynecologic disorders

Women with IBS frequently present concomitant pelvic dysfunctions, particularly dysmenorrhea, dyspareunia, chronic pelvic pain, and overactive bladder. This coexistence reflects shared pathophysiologic mechanisms, such as viscerovisceral hypersensitivity and sensory convergence at the spinal level.

It has been proposed that bladder or gynecologic irritation may sensitize shared afferents, exacerbating digestive symptoms. Additionally, factors such as dysbiosis, low-grade inflammation, and autonomic dysfunction may modulate both pain perception and pelvic motility.

Evaluation of these cases should include a detailed history with a multidisciplinary focus. Therapeutic management benefits from collaboration among gastroenterology, gynecology, urology, pelvic floor physiotherapy, and mental health. Recommended strategies include:

– Pelvic floor rehabilitation exercises and biofeedback.
– Neuromodulators with visceral and somatic action (eg, amitriptyline, duloxetine).
– Psychotherapeutic interventions addressing hypervigilance, anxiety, or chronic pain.
– Specific treatments for urinary or gynecologic dysfunctions (per specialist assessment)

Symptom validation, integrative management, and interdisciplinary coordination are key to improving clinical outcomes and quality of life in this population[7].

Interstitial cystitis or bladder pain syndrome

Interstitial cystitis or bladder pain syndrome (IC/BPS) is characterized by chronic bladder pain in the absence of demonstrable urinary infection. Its prevalence in IBS patients is estimated at 30-40%[36], reflecting a clinically significant association.

Both conditions share similar pathophysiologic mechanisms, such as cross-sensitization between intestinal and bladder visceral afferents, chronic neuroinflammation, and autonomic dysfunction. Increased mast cells in the bladder mucosa and enhanced local histamine release—also described in the intestinal mucosa of IBS patients—have been observed[1,36].

Clinically, IBS-IC/BPS overlap presents with greater pain intensity, persistent urinary symptoms, abdominal discomfort, and a marked impact on quality of life, particularly in young women.

Management must be multidisciplinary and personalized, including:

– Neuromodulators addressing both visceral and somatic pain (eg, duloxetine, pregabalin).
– Psychological therapies focused on chronic pain and anticipatory anxiety.
– Pelvic physiotherapy to improve tone and muscular relaxation.
– Local measures in selected cases (eg, bladder instillations, antihistamines, topical analgesics)

Timely diagnosis and coordinated treatment are essential to prevent chronification and improve functional outcomes.

Dysmenorrhea and chronic pelvic pain

Between 20% and 50% of women with IBS report dysmenorrhea, and up to 35% experience chronic pelvic pain. This coexistence has been attributed to central and peripheral sensitization mechanisms, as well as viscerovisceral convergence phenomena that amplify pain perception[7].

Cyclic gynecologic pain may potentiate intestinal hyperalgesia and vice versa, generating a cross-feedback phenomenon between pelvic organs. Dysmenorrhea and chronic pelvic pain are also associated with higher prevalence of anxiety, sleep disorders, and autonomic dysfunction—all of which may exacerbate IBS.

Differential diagnosis with other causes of pelvic pain (eg, endometriosis, IC/BPS, uterine fibroids) is essential and requires appropriate gynecologic evaluation.

Management must be integrative and individualized, including:

- Dual neuromodulators effective against both visceral and somatic pain.
- Hormonal interventions when a predominant gynecologic etiology is suspected.
- CBT and stress management.
- Pelvic floor physiotherapy in cases of associated muscular dysfunction

Symptom validation and interdisciplinary coordination are fundamental to prevent excessive medicalization, reduce functional impact, and improve quality of life in these patients.

Endometriosis

Endometriosis is a chronic inflammatory disease that affects approximately 10% of women of reproductive age and is frequently associated with GI symptoms that mimic or coexist with IBS. The overlap of both conditions is common and clinically relevant. Studies have reported that between 30% and 50% of women with endometriosis meet diagnostic criteria for IBS[37].

Even after surgical treatment of endometriosis, many patients continue to experience IBS-compatible symptoms, reflecting the persistence of underlying functional mechanisms. Shared pathophysiologic factors include:

- Chronic low-grade inflammation.
- Visceral hypersensitivity.
- Intestinal dysbiosis.
- Persistent central and peripheral sensitization.

The coexistence of endometriosis and IBS is associated with greater symptom severity, impaired quality of life, increased medical consultations, and more frequent use of empirical treatments before achieving a definitive diagnosis.

Management should include:

- Systematic evaluation of gynecologic symptoms in patients with IBS.
- Close coordination with gynecology in complex or refractory cases.
- Rational use of neuromodulators with both visceral and somatic effects.
- Consideration of nonpharmacologic therapies such as pelvic floor physiotherapy, CBT, and chronic pain management strategies.

Since many women with endometriosis initially present to the gastroenterologist, it is essential to maintain a high index of suspicion, particularly in young patients with chronic abdominal pain, changes in bowel habits, and concurrent cyclic or gynecologic symptoms.

Fibromyalgia (FM) and chronic fatigue syndrome (CFS)

FM and CFS are frequent comorbidities in patients with IBS. Up to 65% of IBS patients meet criteria for FM or CFS, while approximately 70% of FM patients report IBS-like symptoms.

These conditions share pathophysiologic mechanisms:

- Central and peripheral sensitization.
- Dysfunction of the GBA and the autonomic nervous system.
- Visceral and somatic hypersensitivity.
- Low-grade neuroinflammation.
- Immune alterations and intestinal microbiota disturbances

Furthermore, they are associated with sleep disorders, somatic hyperalertness, hypervigilance, catastrophizing, and avoidance behaviors, all of which contribute to symptom chronicity and severity.

Patients with IBS and FM or CFS present greater functional disability, worse quality of life, higher rates of absenteeism, and increased healthcare utilization. Some studies have shown that these patients seek medical attention up to four times more often than those without comorbidity and have a poorer response to conventional treatment[12,38].

Similar dysbiosis patterns have also been identified in patients with IBS and FM, suggesting a possible common pathway of alteration in the microbiota-gut-brain axis.

Therapeutic management should be multimodal and include:

– Dual neuromodulators such as duloxetine, pregabalin, or tricyclic antidepressants.
– Stress regulation techniques and chronic pain education.
– Cognitive and psychoeducational therapies.
– Lifestyle interventions, including sleep hygiene, gradual physical activity, and functional nutrition.

Treatment should focus on improving functionality and quality of life rather than complete symptom elimination, reinforcing patient autonomy and avoiding excessive interventions.

Conclusions

The overlap of IBS with other DGBIs and systemic comorbidities is a frequent, complex, and highly relevant clinical phenomenon. Recognition of this overlap implies a paradigm shift: moving away from addressing symptoms in isolation toward an integrated, patient-centered perspective based on shared underlying mechanisms.

The coexistence of multiple DGBIs and comorbidities such as FM, endometriosis, or IC/BPS not only increases symptom burden and worsens quality of life but also requires more careful evaluation and individualized therapeutic strategies. Shared mechanisms—including visceral hypersensitivity, gut–brain axis dysfunction, neuroinflammation, and intestinal dysbiosis—justify a common pathophysiologic approach.

From a diagnostic standpoint, it is essential to perform a targeted history, identify the predominant symptom axis, and consider the dynamic evolution of subtypes. Therapeutically, rational use of neuromodulators, psychotherapeutic interventions, lifestyle-based therapies, and patient validation are crucial to improve clinical outcomes.

Ultimately, successful management of these patients requires empathy, a deep understanding of the DGBI spectrum, and a collaborative interdisciplinary approach. Integrating these elements will enable more human, effective, and evidence-based care.

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Ethical considerations

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Declaration on the use of artificial intelligence. The author declares that no generative artificial intelligence tools were used in the preparation of this manuscript.

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