Irritable Bowel Syndrome

PRESENTER: LILLIAN N. MUSYOKA

FACILITATOR: DR. NONE RAMONATE

Outline

Introduction

Epidemiology

Rome V criteria

Pathophysiology

Approach and work up

Management

Proposed algorithms

Conclusion



Introduction

Irritable Bowel syndrome(IBS) is defined by recurrent abdominal pain and/or discomfort associated with changes in bowel habits.

Disorder of brain gut interaction(DGBI) formerly functional GIT Disorders.

Prevalence: 4.3% in the general population-UK,US and Canada, Rome IV vs 10% Rome III.[1]

Peak prevalence 18-39 years.

F>M(1.8:1)

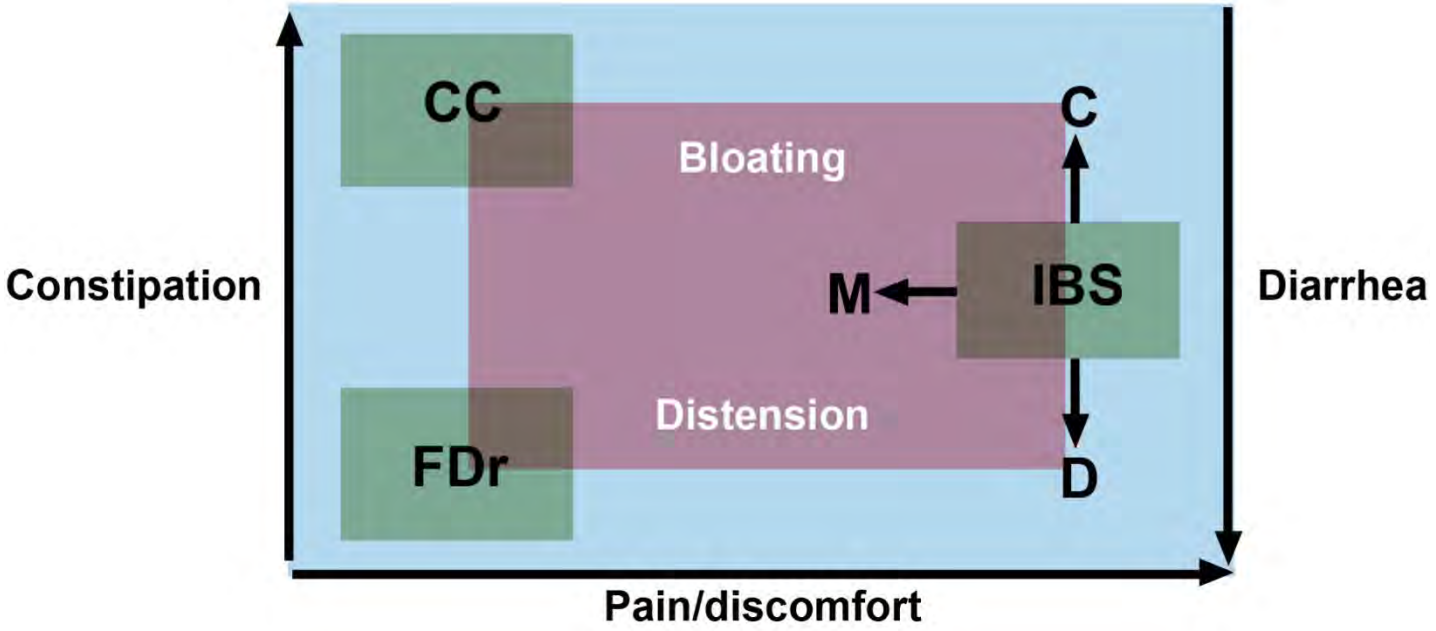
SSA prevalence reported to be 16% in a 2026 meta-analysis.[2]

Accounts for reduced patient quality of life, increased healthcare costs and missed work.

Represents 5-50% of referrals to gastroenterologists.

40% of patients who meet the diagnostic criteria don’t have a formal diagnosis.

Spectrum of bowel disorders



CC: Chronic constipation
FDr: Functional diarrhea
IBS-C: Irritable bowel syndrome with predominant constipation
IBS-D: Irritable bowel syndrome with predominant diarrhea
IBS-M: Irritable bowel syndrome with mixed bowel habits (D and C)

Diagnostic criteria for IBS (Rome v)

Recurrent, but not continuous, abdominal pain or discomfort on average at least **3 days per month** in the past 3 months, associated with 2 or more of the following criteria:

1. Related to defecation
2. Associated with a change in frequency of stool
3. Associated with a change in form (appearance) of stool

Criteria symptom onset at least 6 months before diagnosis.

Supportive criteria: abdominal pain/discomfort should not be only related to menses.

Corsetti M et al **Bowel Disorders** Gastroenterology, 2026

Rome III diagnostic criteria for IBS	Rome IV diagnostic criteria for IBS	Rome V diagnostic criteria for IBS*
1 Recurrent abdominal pain **or discomfort** at least **3 days per month** and associated with two or more of the following: (a) **improvement** with defecation (b) onset associated with a change in frequency of stool (c) onset associated with a change in form of stool	1 Recurrent abdominal pain, on average, at least **1 day per week** in the past 3 months and associated with two or more or the following: (a) **related** to defecation (b) associated with a change in frequency of stool (c) associated with a change in form of stool	1 Recurrent, **but not continuous**, abdominal pain **or discomfort** at least **3 days per month** and associated with two or more of the following: (a) **related** to defecation (b) onset associated with a change in frequency of stool (c) onset associated with a change in form of stool
2 Criteria fulfilled for the past 3 months with symptom onset at least 6 months before diagnosis	2 Criteria fulfilled for the past 3 months with symptom onset at least 6 months before diagnosis	2 Criteria fulfilled for the past 3 months with symptom onset at least 6 months before diagnosis

What's the change and why?

Rome V reintroduced "discomfort" and lowered the frequency threshold for abdominal pain or discomfort to ≥3 days per month.

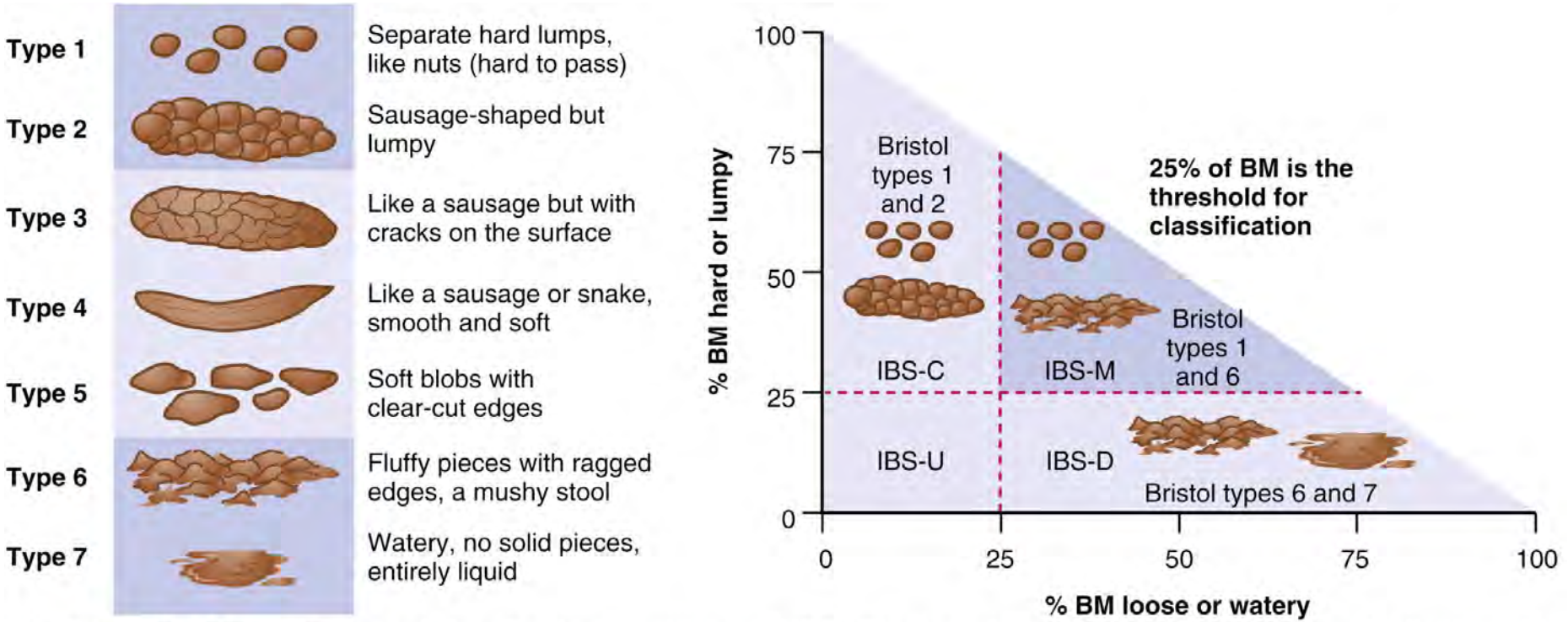
Prevalence of IBS decreased significantly compared with Rome III.

Attributed to the higher frequency threshold for pain in Rome IV ,and to a lesser extent to the removal of "discomfort".

Abdominal pain or discomfort should not be continuous to differentiate IBS from centrally mediated abdominal pain syndrome.

Abdominal pain or discomfort should not be only related to menses has been added to differentiate IBS from menstrual-related symptoms.

Diagnostic criteria for IBS subtypes



The Bristol Stool Form Scale and classification of subtypes of IBS. *BM* , bowel movement; *C,* constipation; *D,* diarrhea; *M,* mixed; *U,* unsubtyped.

Based on BSFC on days with abnormal stools off laxatives and antidiarrheal agents

Pathophysiology

• Uncertain pathogenesis: The exact underlying mechanism and primary drivers of IBS remain systematically unconfirmed.

•Currently recognized as an intricate disorder resulting from intersecting physiologic elements with a focus on gut motility, sensation and permeability.

•Decades of clinical studies have yielded highly variable and often contradictory outcomes.

• To date, no single biological or physiological marker has been isolated as specific to IBS.

Pathophysiology:

Proposed mechanisms:

- Dysregulated gut motility
- Visceral hypersensitivity
- Barrier dysfunction/ Permeability
  - Mucosal inflammation
  - Post infectious IBS
  - Alteration in gut microbiome
  - Food sensitivity
- Genetics
- Psychosocial dysfunction

Dsyregulated gut motility

No uniform motor pattern defines IBS, but observed abnormalities include prolonged transit time in constipation-predominant cases (IBS-C) and exaggerated postprandial motor responses in diarrhea-predominant cases (IBS-D).

Visceral hypersensitivity: Patients experience abdominal pain and awareness at significantly lower intestinal balloon distension volumes than healthy controls, showing heightened cerebral cortical activity.

Patients with IBS experience bloating and excess gas with similar gas volumes as controls.

It is still unclear whether the hyperalgesia is mediated by ENS or by central modulation from the brain or by both.

GI mediators, serotonin, kinins and acticvation of NMDA receptors in the spinal cord are thought to play a role.

The recognition of the ENS and CNS interaction led to the concept of Disorder of Brain-Gut interaction.

Barrier dysfunction/ Permeability: Increased permeability allows penetration of luminal antigens into the sub epithelial space where they can activate the immune system.

Mucosal inflammation: Microscopic immunohistologic analysis from jejunal biopsies reveals active mucosal immune system up regulation, particularly in IBS-D and post-infectious subtypes.

Lymphocyte infiltration, elevated activated mast cells and circulating pro inflammatory cytokines have been demonstrated in the colon and SI.

These cells release mediators (nitric oxide, histamine and proteases) capable of stimulating the ENS, leading to abnormal motor and visceral responses within the intestine.

Post infectious IBS: Follows acute bacterial, protozoan, helminthic or viral enteritis.

Triggers a fourfold increase in the long-term risk of developing chronic IBS symptoms.

Highest risk in females, younger populations, individuals with preexisting anxiety, or those experiencing a severe initial infection (diarrhea >3 weeks, hospitalization).

Symptoms are driven by persistent physiological changes, including idiopathic bile acid malabsorption and localized immune activation.

Increased serotonin leads to increased GI motility and hypersensitivity.

Use of antibiotics for GI or other infections has been observed to predispose to development of functional bowel symptoms.

Gut microbiome and dysbiosis:

IBS patients exhibit gut dysbiosis, characterized by significantly decreased overall alpha diversity and a disrupted microbial population.

Biomarkers frequently show an increased Firmicutes-to-Bacteroidetes ratio, with a sharp drop in beneficial, anti-inflammatory, butyrate-producing genera like Bifidobacterium and Faecalibacterium.

Metabolic & Barrier Impacts: Dysregulated commensal flora produce altered metabolites (short-chain fatty acids, bile acids, proteases, endotoxins) that disrupt epithelial tight junction integrity and trigger increased intestinal permeability ('leaky gut').

Patients report reduced flatulence with probiotics use without alteration in intestinal microbiota.

Small Intestinal Bacterial Overgrowth: occurs in a subset of patients, where premature carbohydrate fermentation in the SI causes excessive gas production, bloating, and altered motility.

Whether there is association between IBS and SIBO is conflicting.

High breath hydrogen levels vs rapid orocecal transit in IBS.

Response to antibiotics- improvement in motility and change in flora vs SIBO

Food sensitivity: Role in development of IBS unclear.

45 to 60 % of IBS patients report worsening of symptoms after eating, and some perceive intolerance to certain foods . Factors contributing food insensitivity in IBS include:

Food allergy: Higher positive food skin-prick tests in IBS patients with no exacerbation of symptoms with those foods.

Carbohydrate malabsorption: The theory holds that FODMAPs in patients with IBS or IBD enter the distal SI and colon where they are fermented, leading to symptoms and increased intestinal permeability and possibly inflammation.

Gluten sensitivity: Several studies suggest some overlap between celiac disease and IBS.

Dietary gluten has been shown to alter SI permeability and had a greater effect on bowel movement frequency in patients who were HLA-DQ2/8 positive.

Celiac disease should be excluded in IBS patients with serological tests suggestive of IBS.

Genetics: Familial studies and studies on select gene polymorphisms suggest a genetic susceptibility in some patients with irritable bowel syndrome (IBS) eg.Serotonin transporter gene.

This results in altered serotonin reuptake efficacy that affects intestinal peristalsis.

A parent with IBS is a higher predictor of IBS than a twin with IBS –social learning

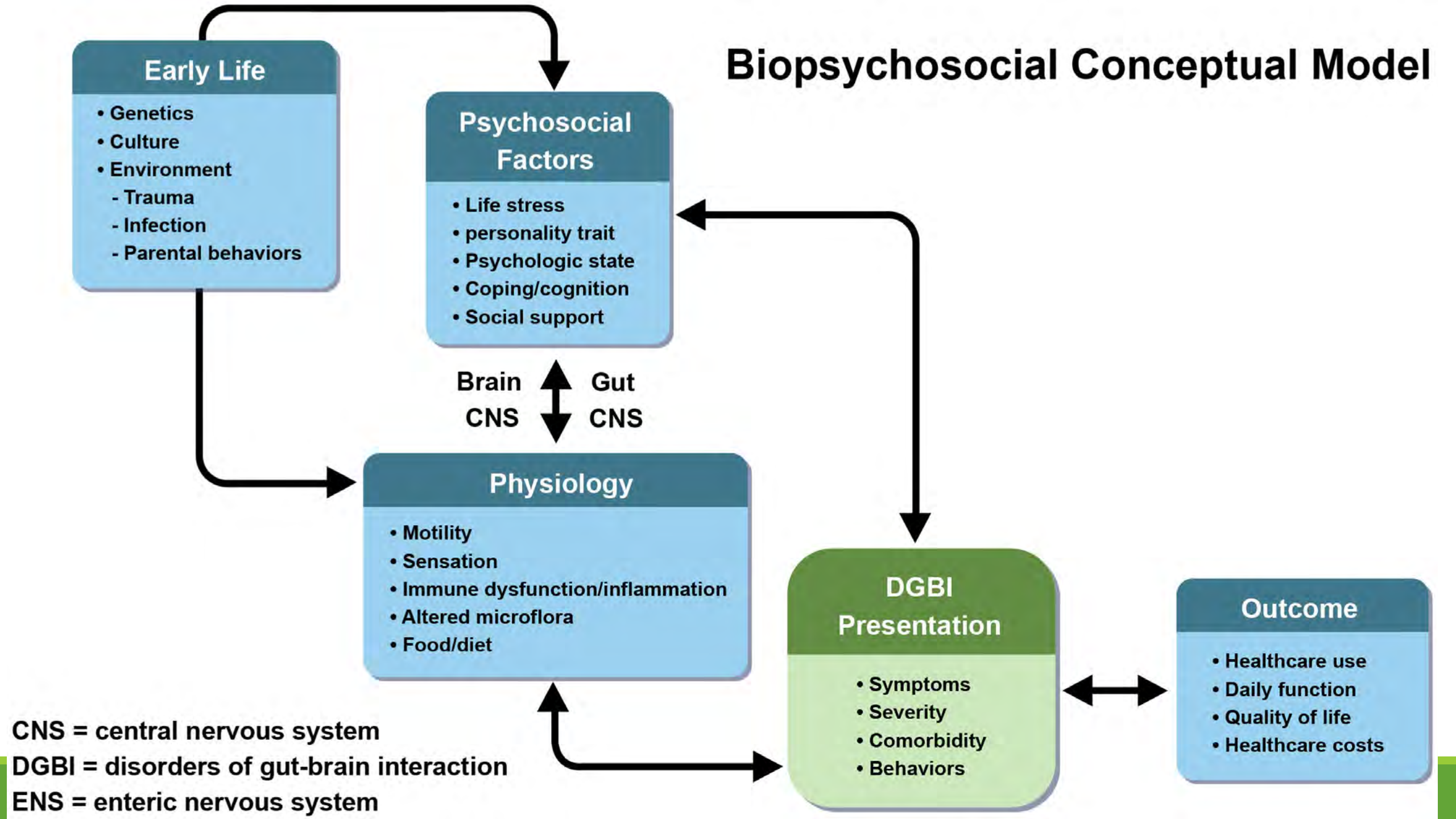
Psychosocial dysfunction: Patients with IBS exhibit increased anxiety, depression, phobias, and somatization compared with controls.

Positive association between IBS and abuse has been also reported.

Eating disorders in adolescence and early childhood have also been associated with development of IBS.

Corticotropin releasing factor (CRF) from the periventricular nucleus is thought to mediate this stress response.

Biopsychosocial Conceptual Model



CNS = central nervous system
DGBI = disorders of gut-brain interaction
ENS = enteric nervous system

Risk factors

- Post-infections
- Higher socio-economic status
- Previous antibiotic use
- Food intolerance
- Extra-intestinal somatic symptoms
- Perinatal factors
  - Young maternal age
  - Caesarian section
  - Low birth weight

Clinical evaluation

Move towards a POSITIVE diagnosis based on symptoms.

IBS is no longer a diagnosis of exclusion.

Little utility for wide array of tests in the absence of alarm features.

Negative test results do not necessarily improve reassurance or quality of life.

Empathy and active listening skills are key.

History

Ascertain onset and duration of symptoms.

Review co-morbities, medications, recent infection, surgery, psychological trauma or travel.

Confirm location of pain/discomfort and relationship(aggrevated, relieved or associated) with bowel habits.

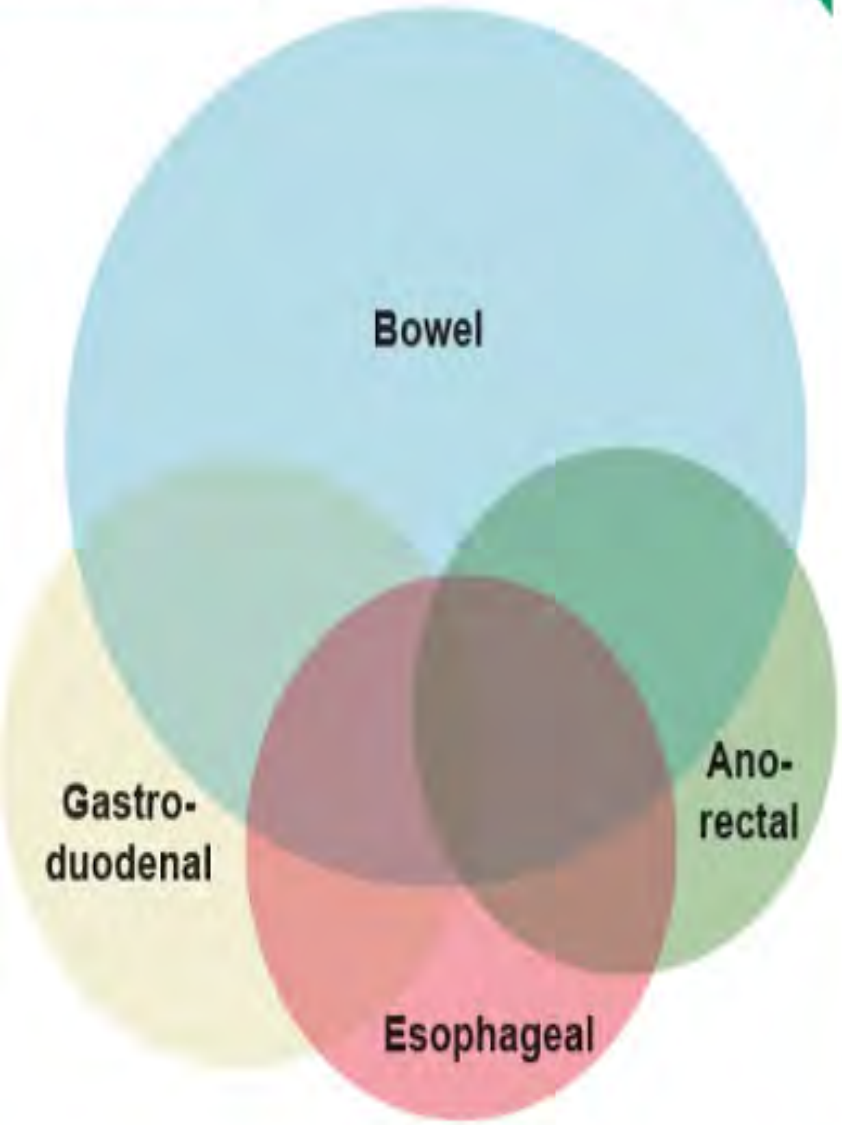
Assess for presence of other DGBI and GI conditions.

Exclude alarm features.

Dietary assessment(restrictive diet, intake of FODMAPS and alcohol).

Box 3

DGBI/FBD





Fibromyalgia



Chronic headache



Migraine headache



Chronic back pain



Chronic Fatique syndrome



Temporomandibular joint disorder



Interstitial cystitis



Chronic pelvic pain; Dyspareunia; Premenstrural syndrome; Dysmennorhea



Non-GI conditions

Alarm features

SYMPTOMS

Blood in stool

Chronic diarrhea < 1 year

Unexplained weight loss

Unexplained recurrent vomiting

Progressive dysphagia

FHX of IBD, CRC or coeliac disease

Short duration of symptoms

Anaemia

New-onset symptoms in older age >50

Nocturnal diarrhea

Travel history

SIGNS

Abdominal mass

Active arthritis

Dermatitis hepertiformis or pyoderma gangrenosum

Blood on DRE

Signs of thyroid dysfunction

Features of intestinal malabsoption.

Signs of abdominal obstruction.

Clinical examination

Abdomen:

- Tenderness, distention, masses.
- Differentiate distention from ascites and adiposity.

Rectal exam:

- Identify causes of anal rectal bleeding.
- Perianal appearance, sensation, anal reflexes, anal sphincter tone and contraction pattern.
- Abnormal wall contraction during stimulated defecation (dyssnergic defecation or pelvic floor dysfunction)

Blood tests

CBC- all patients at baseline

CRP/ESR- all patients

CRP ≤0.5 mg/dL yielded a 1% probability of IBD with good accuracy

Celiac serology- X3 higher positive rate compared to general population regardless of IBS sub type.

Clinical acumen should be the guiding principle.

Stool tests

Fecal calprotectin/ fecal lactoferrin

Indication: IBS-D without alarm features

High negative predictive value for IBD

BSG cut offs :<100 normal and >250 ug/g indication for colonoscopy to r/o IBD .

100-249 : Repeat off PPIs and NSAIDs

Fecal calprotectin elevated in >45 years, obesity, infection, malignancy, PPIs, NSAIDs.

Despite a low IBD pre test probability at diagnosis, the incidence was noted to be 2.65 times higher than controls at 5 years in 2 separate studies.

Stool tests

Stool analysis for ova, bacteria and parasites-Routine testing not recommended

Testing should be based on the appropriate clinical setting e.g Giardiasis in persons with history of travel to endemic areas, poor water quality, camping, and daycare exposure.

Fecal occult blood test (FOBT) and Fecal immunochemical test(FIT): not recommended as routine tests.

Fecal elastase: not recommended as routine tests.

Should be considered when steatorrhea is reported.

Colonoscopy

Both the ACG and BSG recommend against routine colonoscopy in patients <45 years with IBS.

Low yield of polyps even when adjusted for age compared to non–IBS population; 8% vs 26%.

Indicated when there is concern for microscopic colitis i.e Female, >50 years, severe or nocturnal watery diarrhea, co-existent autoimmune disease, weight loss, duration <12 months and use of potential precipitating drugs e.g PPIs, NSAIDs.

Bile acid malabsorption

BAM testing not recommended in the initial evaluation.

Can be considered in patients with chronic diarrhea unresponsive to standard therapy, nocturnal diarrhea or prior cholecystectomy.

The 23-seleno-25-homotaurocholic acid (SeHCAT) retention test is considered the gold standard test.

Other alternatives: serum 7α-hydroxy-4-cholesten-3-one (7C4) blood assay and FGF-19

Food allergies and sensitivities: testing not recommended unless there are reproducible symptoms.

Only 2-3% of adults have food allergies despite 20% reporting food allergies.

Anorectal Physiology Testing(ARPT)

ARPT should be performed in patients with IBS and symptoms suggestive of a pelvic floor disorder and/or refractory constipation not responsive to standard medical therapy.

Accurate diagnosis of defecation disorder(DD)requires physiologic testing with abnormalities of a defecation disorder identified in 2 of 3 tests (e.g., ARM, BET, and/or impaired evacuation by imaging)

Utility: positive response of both pain and bowel symptoms to biofeedback therapy

Table 6. Clinical symptoms, rectal examination findings, and anorectal physiologic testing suggestive of a pelvic floor disorder[a]

Testing	Findings
Rectal examination findings on inspection	Dermatitis/perianal erythema Rectal prolapse Gaping anus Hemorrhoids Fistula or fissure Rectal scar Anorectal mass
Digital rectal examination findings suggesting dyssynergic defecation	Impaired sensory perception of stool (perineal sensation testing) Rectal distension and stool impaction Contraction of the diaphragm, abdomen, and rectum during push maneuvers (abdominal pressure and rectal examination must be performed simultaneously) Abnormal relaxation of external anal sphincter and puborectalis muscles (or no relaxation with Valsalva maneuver)
Anorectal physiology findings suggesting pelvic floor disorders	Uncoordinated abdominal, rectoanal, and pelvic floor muscles Rectal hyposensitivity Paradoxical increase in sphincter pressure/puborectalis muscle pressure during relaxation or simulated evacuation Prolonged balloon expulsion time Inadequate anal relaxation during push maneuvers Inadequate abdominorectal propulsive forces

[a]Many of these symptoms and examination findings are seen in all subtypes of

Diagnosis

Diagnosis should be communicated clearly and confidently using simple language.

BSG provides a patient summary.

40% of patients meeting the diagnostic criteria don't have a diagnosis.

Build a strong patient-provider alliance.

Establish sub-type- what do your abnormal bowel movements usually look like?(off-meds) a 2 week stool chart reasonable.

Management based on predominant symptom.

Management


Multidisciplinary approach
Patient centered – Physician continuity
Brain-gut behavioral therapies
CBT – Hypnotherapy – Psychotherapy
Pharmacotherapy – dominant symptom
Constipation – Diarrhea – Pain - Gas/bloating –
Psychological Distress - Multiple
Dietary advice
including probiotics
Diagnosis and explanation
Reassurance
Precipitating factors - Life style
Assessment of symptom profile and severity

Management

A diagnosis of IBS needs to be communicated clearly to the patient using simple words and explanations.

Establish a patient-doctor alliance- review previous treatments, offer the options available, consider patient preferences and manage expectations.

Therapies are directed towards the predominant symptom, or symptoms, experienced by the patient.

First line therapies

Diet and exercise-ALL

Soluble fibre e.g ispaghula, effective for global symptoms and abdominal pain.

Avoid insoluble fibre e.g wheat, beans- fermented with worsening symptoms

Diet low in FODMAPS- GI dietician directed

3 stages

- Elimination
- Re-introduction
- Personalization

Restrictive - micronutrient deficiency and disruption of microbiome.

Elimination based on IgG Ab testing and gluten free diet not recommended.

HIGH FODMAP FOODS

Foods to avoid on a low FODMAP diet

LEGUMES	DAIRY	FRUITS	VEGETABLES	GRAINS
				
Lentils Chickpeas Black beans Kidney beans Soybeans	Dairy, including milk from cows, goats, or sheep Yogurt Soft cheeses, Ice cream Custard	Apples Pears Watermelon Cherries Mangoes Honey High fructose-corn syrup Agave syrup Sweeteners	Onions Garlic, Cauliflower Asparagus Mushrooms Broccoli Cabbage	Wheat Rye Barley Bread Pasta Cereals

Probiotics may be effective for global symptoms and abdominal pain.

No specific species or strain recommended.

A trial of 12 weeks reasonable.

Stop if no response.

Peppermint oil recommended for relieve of global symptoms.

Antispasmodics e.g. hyoscine butylbromide –may be effective for global symptoms but use limited by SEs: dry mouth, dizziness and visual disturbance. ACG doesn't recommend.

IBS-C

Polyethylene glycol (PEG) for relieve of global symptoms in IBS-C

Soluble fiber supplements

Secretagogues

Chloride channel activators for global IBS-C symptoms

- Lubiprostone 8 ug BD PO
- SE: Diarrhea

Guanylase cyclase C agonists effective for IBS-C

- Linaclotide 290 ug PO OD
- Plecanatide 3 ug PO OD
- SE: Diarrhea

Retainagogues

Sodium-hydrogen exchange-3 inhibitor(NHE3), is efficacious for IBS-C

- Tenapanor 50 mg PO BID

IBS-C

5-HT 4 receptor agonist. Improves pain and constipation.

- Tegaserod 6 mg PO BD
- patients <65 years of age with IBS-C without pre-existing cardiovascular disease who have not responded to other therapies.
- SE:Diarrhea
- Initially withdrawn from the market due to serious cardiovascular events.

IBS-D

Loperamide

SE: abdominal pain, bloating, nausea and constipation

Start low and up-titrate

Alosetron

5-HT3 receptor antagonists are efficacious drugs for IBS-D

Women with severe IBS-D. Withdrawn -life threatening ischemic colitis and severe constipation.

Available only through special prescribing at a lower dose of 0.5mg PO BD

Alternatives :Ramosetron (ASia , Ondansetron 4-8mg TID(improvement in bloating, urgency, consistency but not pain.

Rifaximin –less effective for abdominal pain, 550mg PO TID for 10-14 days

Can be repeated after loss of efficacy.

IBS-D

Eluxadoline is a μ-opioid and κ-opioid receptor agonist and δ-opioid receptor antagonist

licensed for IBS-D.

Dosage 100 mg PO BD

SE: pancreatitis, Sphincter of Oddi spasm

Contraindications: prior sphincter of Oddi problems or cholecystectomy, alcohol dependence, pancreatitis or severe liver impairment

Gut directed psychotherapy

Recommended after 12 months of pharmacological therapy with no improvement.

Can be considered earlier in the management course.

Cognitive behavioural therapy

Gut directed hypnotherapy

Severe and refractory IBS

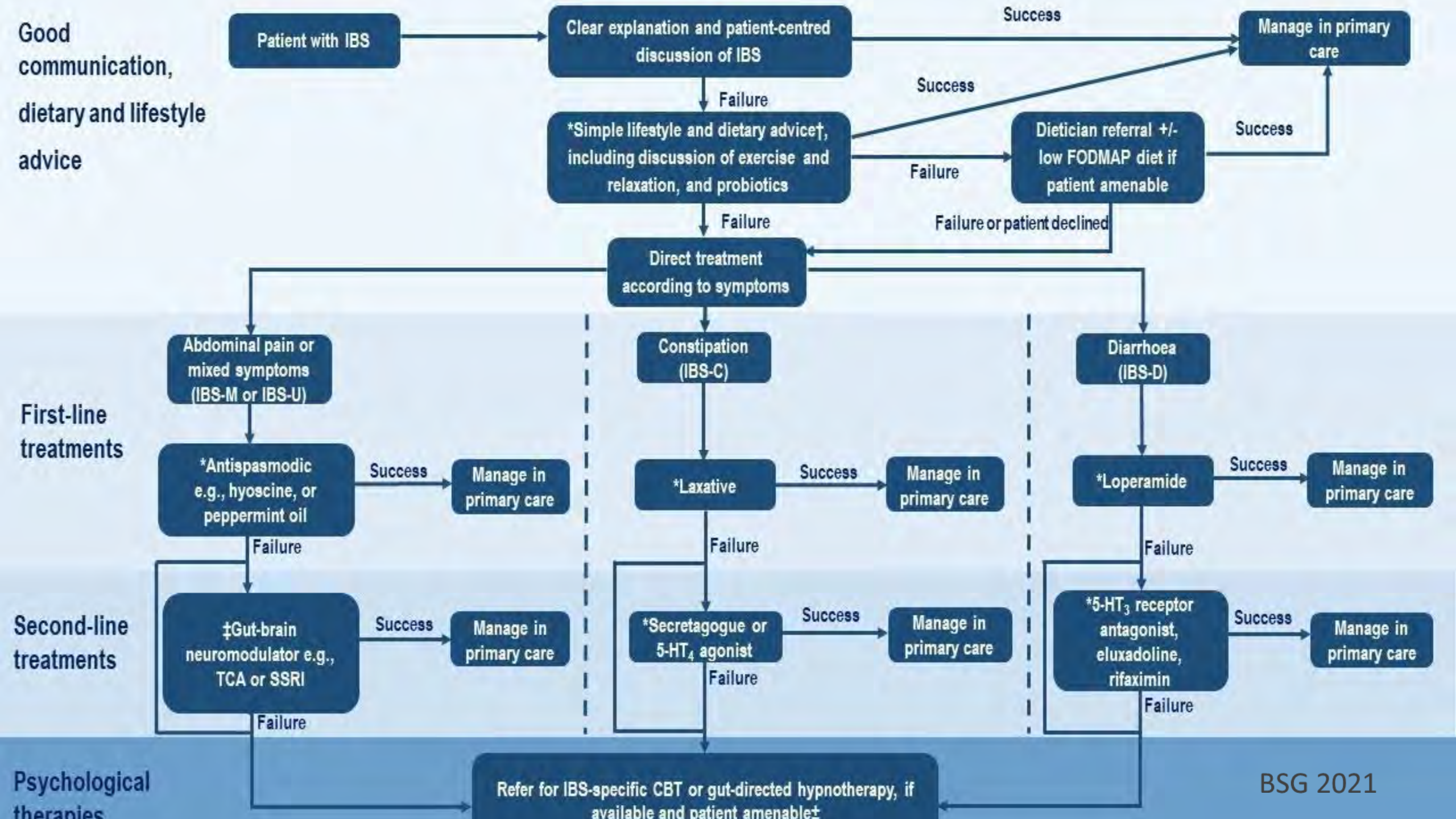
Should prompt review of diagnosis and further targeted investigation.

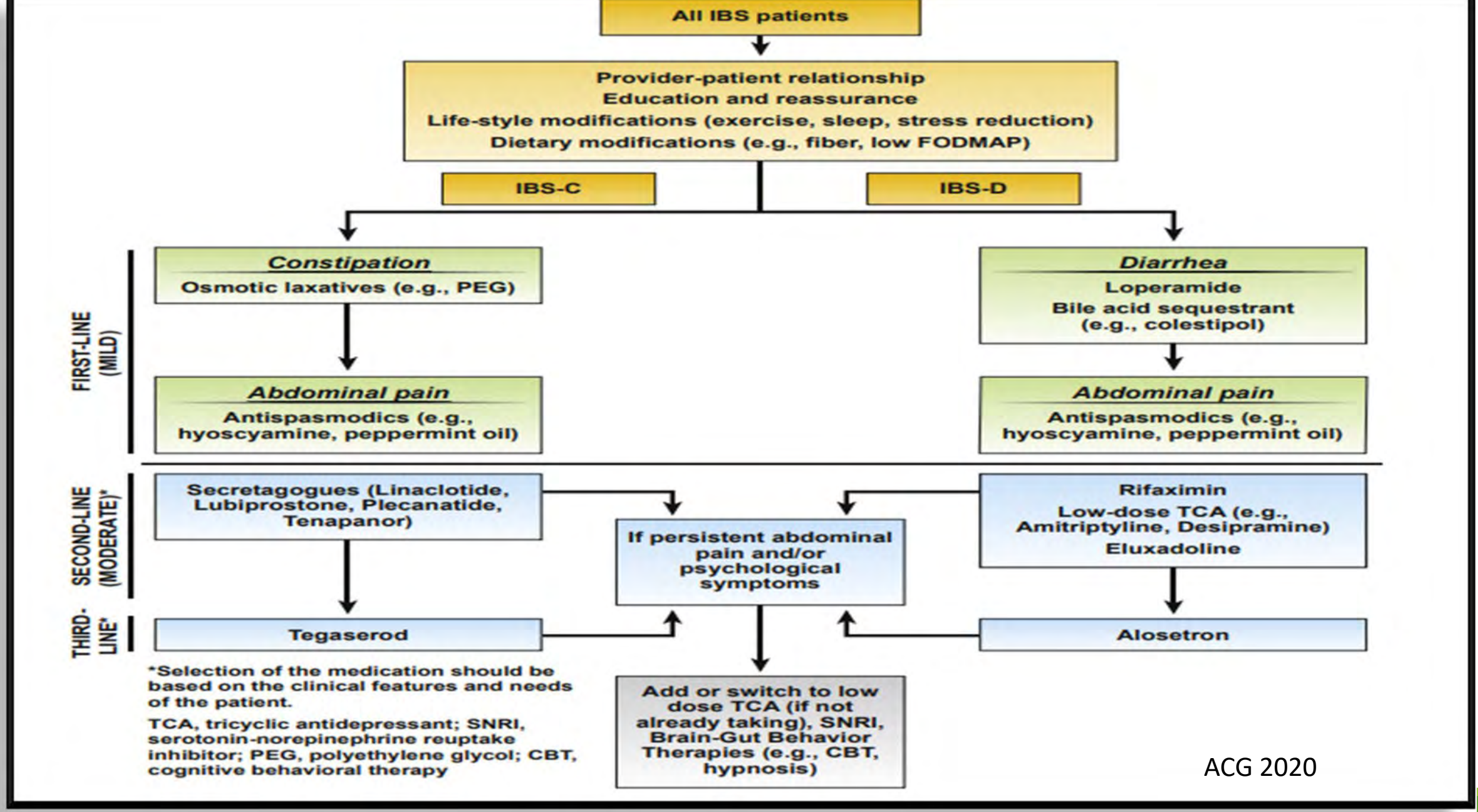
TCAs

- Amitryptyline
- Imipramine
- *Desimipramine*
- *Nortriptyline*
- *Less sedation and constipation*

SSRIs

- Escitalopram
- Citalopram
- Fluoxetine
- Sertaline
- Paroxetine


Good communication, dietary and lifestyle advice
Patient with IBS
Clear explanation and patient-centred discussion of IBS
Success
Manage in primary care
Failure
Success
*Simple lifestyle and dietary advice†, including discussion of exercise and relaxation, and probiotics
Dietician referral +/- low FODMAP diet if patient amenable
Success
Failure
Failure
Failure or patient declined
Direct treatment according to symptoms
First-line treatments
Abdominal pain or mixed symptoms (IBS-M or IBS-U)
Constipation (IBS-C)
Diarrhoea (IBS-D)
*Antispasmodic e.g., hyoscine, or peppermint oil
Success
Manage in primary care
*Laxative
Success
Manage in primary care
*Loperamide
Success
Manage in primary care
Failure
Failure
Failure
Second-line treatments
‡Gut-brain neuromodulator e.g., TCA or SSRI
Success
Manage in primary care
*Secretagogue or 5-HT4 agonist
Success
Manage in primary care
*5-HT3 receptor antagonist, eluxadoline, rifaximin
Success
Manage in primary care
Failure
Failure
Failure
Psychological therapies
Refer for IBS-specific CBT or gut-directed hypnotherapy, if available and patient amenable‡
BSG 2021


All IBS patients
Provider-patient relationship
Education and reassurance
Life-style modifications (exercise, sleep, stress reduction)
Dietary modifications (e.g., fiber, low FODMAP)
IBS-C
IBS-D
FIRST-LINE (MILD)
Constipation
Osmotic laxatives (e.g., PEG)
Abdominal pain
Antispasmodics (e.g., hyoscyamine, peppermint oil)
Diarrhea
Loperamide
Bile acid sequestrant (e.g., colestipol)
Abdominal pain
Antispasmodics (e.g., hyoscyamine, peppermint oil)
SECOND-LINE (MODERATE)*
Secretagogues (Linaclotide, Lubiprostone, Plecanatide, Tenapanor)
Rifaximin
Low-dose TCA (e.g., Amitriptyline, Desipramine)
Eluxadoline
If persistent abdominal pain and/or psychological symptoms
THIRD-LINE*
Tegaserod
Alosetron
Add or switch to low dose TCA (if not already taking), SNRI, Brain-Gut Behavior Therapies (e.g., CBT, hypnosis)
*Selection of the medication should be based on the clinical features and needs of the patient.
TCA, tricyclic antidepressant; SNRI, serotonin-norepinephrine reuptake inhibitor; PEG, polyethylene glycol; CBT, cognitive behavioral therapy

ACG 2020

Take home points

IBS is not a diagnosis of exclusion.

Limited investigations as guided by presence or absence of alarm features.

Firm and empathetic communication of diagnosis is key in establishing a doctor-patient alliance.

Multi-disciplinary management of the patient.

Psychotherapy is also treatment, don’t be afraid to offer

References

1. Lacy, B. E., Pimentel, M., Brenner, D. M., Chey, W. D., Keefer, L. A., Long, M. D., & Moshiree, B. (2020). ACG Clinical Guideline: Management of Irritable Bowel Syndrome. *American Journal of Gastroenterology*
2. Alemayehu, Z. G., Zewdie, Y. A., Woldeselassie, B. G., Sime, B. L., Ayalew, B. D., Keraga, A. S., ... & Awaso, Y. T. (2026). Prevalence and Associated Factors of Irritable Bowel Syndrome in Selected Sub-Saharan African Countries: A Systematic Review and Meta-Analysis. *Journal of Medicine, Surgery, and Public Health*
3. Vasant, D. H., Paine, P. A., Black, C. J., Houghton, L. A., Everitt, H. A., Corsetti, M., ... & Ford, A. C. (2021). British Society of Gastroenterology guidelines on the management of irritable bowel syndrome. *Gut*
4. Drossman, D. A., Chang, L., & Tack, J. (2026). Disorders of gut–brain interaction and the Rome V process. *Gastroenterology*
5. Corsetti, M., Shin, A., Lacy, B. E., Cash, B. D., Simrén, M., Schmulson, M. J., ... & Lembo, A. (2026). Bowel disorders. *Gastroenterology*
6. Grover, M., Vanuytsel, T., & Chang, L. (2025). Intestinal permeability in disorders of gut–brain interaction: from bench to bedside. *Gastroenterology*
7. *UpToDate*

Thank you