A GI-Led Approach to Chronic Pelvic Pain

From Anatomy to Management: A Gastroenterologist's Perspective

Amber Charoen, MD
Gastroenterology • Hepatology • Gastrointestinal Endoscopy
Women's Gastrointestinal Health • Nutritional-related GI Conditions
PROSSER DIGESTIVE HEALTH CENTER | PROSSER MEMORIAL HEALTH
820 MEMORIAL ST, SUITE 3 | PROSSER, WA 99350
o: (509) 786-5599 | c: (857) 312-6114 | f: (509) 788-0099

WHY THIS MATTERS

Epidemiology & Burden of CPP

26
% Women Affected
Up to 26.6% prevalence

6
Months Duration
Diagnostic threshold

50
% CPP Have IBS
Median co-occurrence

50
% Have Comorbidities
Endo, IBS, IC combined

Definition

Non-cyclical or cyclical pain in the lower abdomen/pelvis lasting **≥6 months**, causing functional impairment and negative impact on quality of life

GI Contribution

GI disorders are among the **most common** causes of CPP — IBS and pelvic floor dysfunction frequently drive or perpetuate the pain cycle

Outline

01 **Pelvic Anatomy**

GI structures in the pelvis and their innervation

02 **Pathophysiology**

Visceral pain, cross-sensitization, and central sensitization

03 **GI Conditions**

IBS, IBD, pelvic floor dyssynergia, endometriosis overlap

04 **Differential Diagnosis**

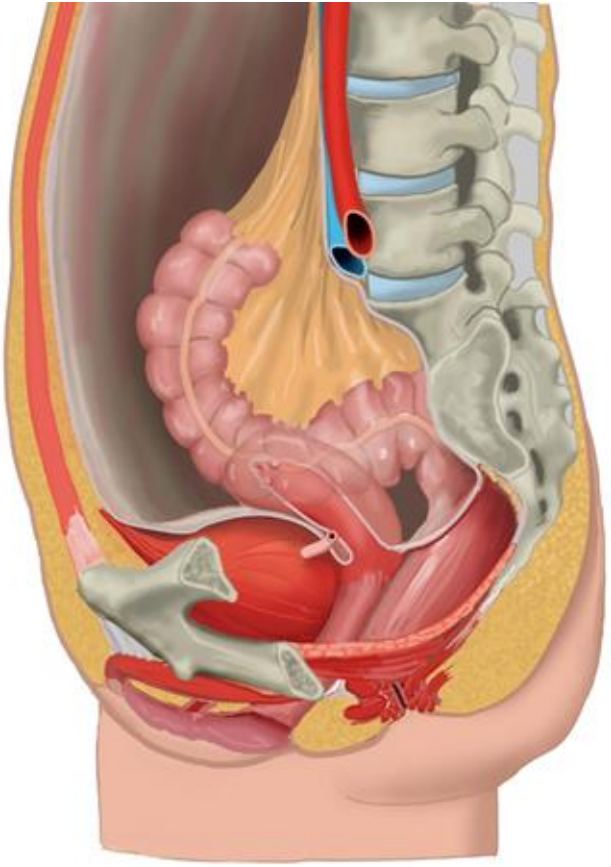
Distinguishing GI from non-GI causes of pelvic pain

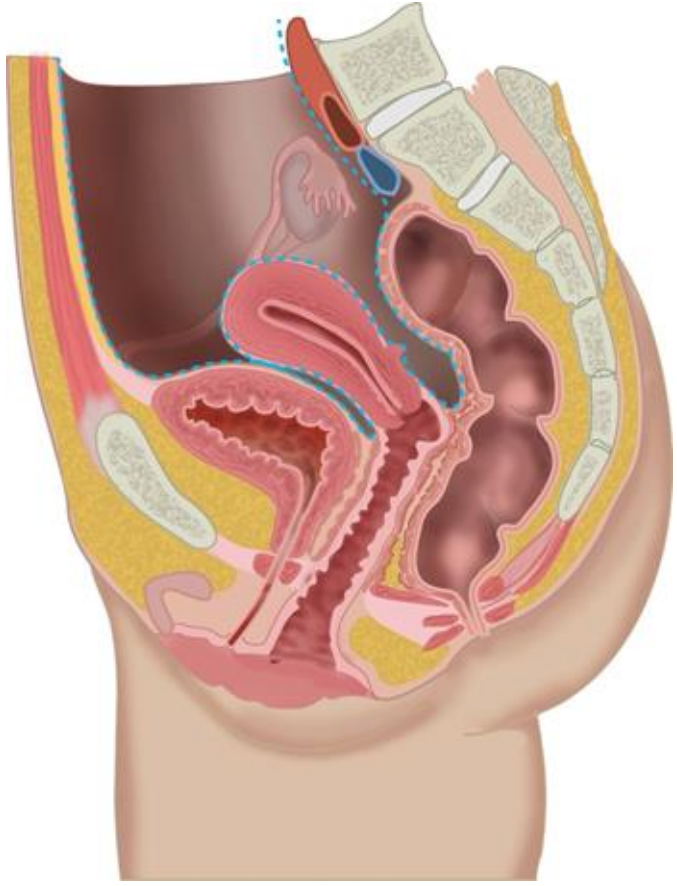
05 **Treatment Approach**

Multidisciplinary management from a GI perspective

Pelvic Anatomy

GI structures, innervation, and shared neural pathways in the pelvis





GI Structures in the Pelvis

Key Structures

Sigmoid colon
S-shaped, 25–40 cm; mobile intraperitoneal structure

Rectum
Begins at S3; retroperitoneal; stores feces

Anal canal
3–4 cm; internal and external sphincters

Appendix and cecum
Right iliac fossa; may cause chronic pelvic pain

Anatomic Relationships

Anterior to sigmoid:
Bladder, uterus, upper vagina (females)

Posterior to sigmoid:
Rectum, sacrum, ileum

Pouch of Douglas:
Between uterus and rectum; common site for endometriosis

Blood supply:
IMA → sigmoidal branches, superior rectal artery

Source: StatPearls – Sigmoid Colon, TeachMeAnatomy

Pelvic Innervation Relevant to GI

Pathway	Segments	Structures Innervated	Clinical Relevance
Sympathetic (thoracolumbar)	T10–L2	Uterus, adnexa, lower ileum, sigmoid, rectum	Shared afferents explain organ-to-organ pain overlap
Parasympathetic (sacral)	S2–S4	Distal colon, rectum, bladder, lower uterine segment	Pudendal nerve; visceral cross-sensitization
Somatic (pudendal nerve)	S2–S4	External anal sphincter, pelvic floor, perineal skin	Levator ani syndrome, pudendal neuralgia
Hypogastric plexus	T10–S4	Main autonomic center; fibers throughout pelvis	Key relay for viscerovisceral convergence

Source: BioMed Res Int 2014, MedCentral

Pathophysiology

Why pelvic organs share pain: visceral sensitization and convergence

Mechanisms of Chronic Pelvic Pain

Visceral Hypersensitivity

Sensitization of primary afferents innervating the viscera leads to lowered pain thresholds. The gut becomes hyper-responsive to normal distension.

~35% of CPP patients improve when treated for functional bowel disorders.

Cross-Sensitization

Noxious stimulus from a diseased pelvic organ transmits to adjacent normal structures:

– Dichotomizing DRG neurons (3–10%)
– Convergence on spinal dorsal horn

Highest density between distal colon and urinary bladder.

Central Sensitization

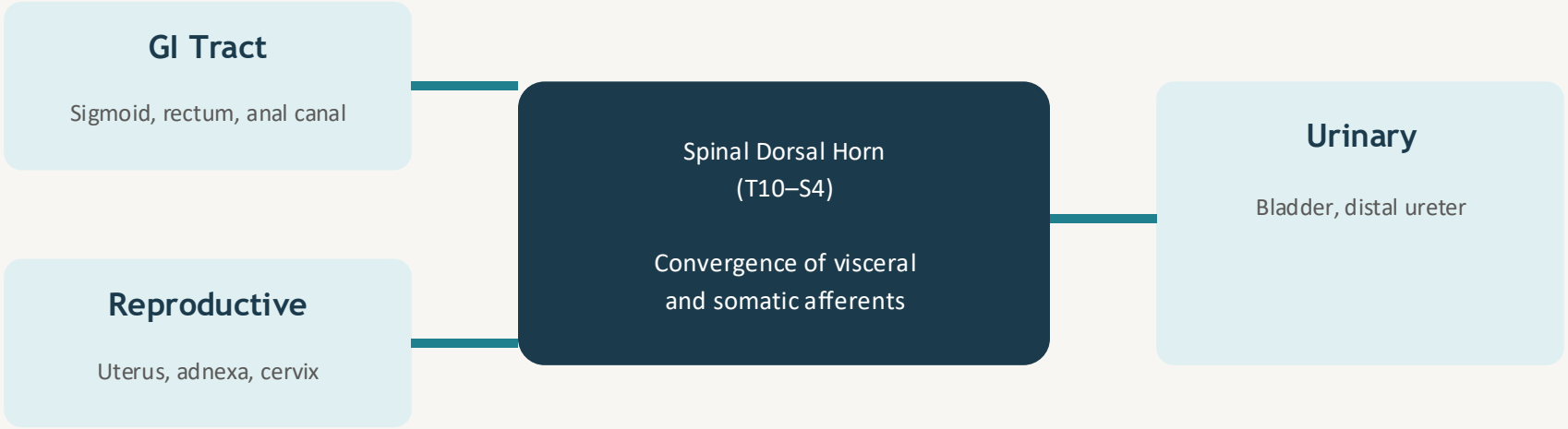
Persistent pain alters CNS processing → heightened sensitivity and amplified discomfort even after the original pathology resolves.

Manifests as hyperesthesia, allodynia, and overlap with other chronic pain conditions.

Source: Handb Exp Pharmacol 2011, BioMed Res Int 2014, StatPearls

Viscerovisceral Convergence

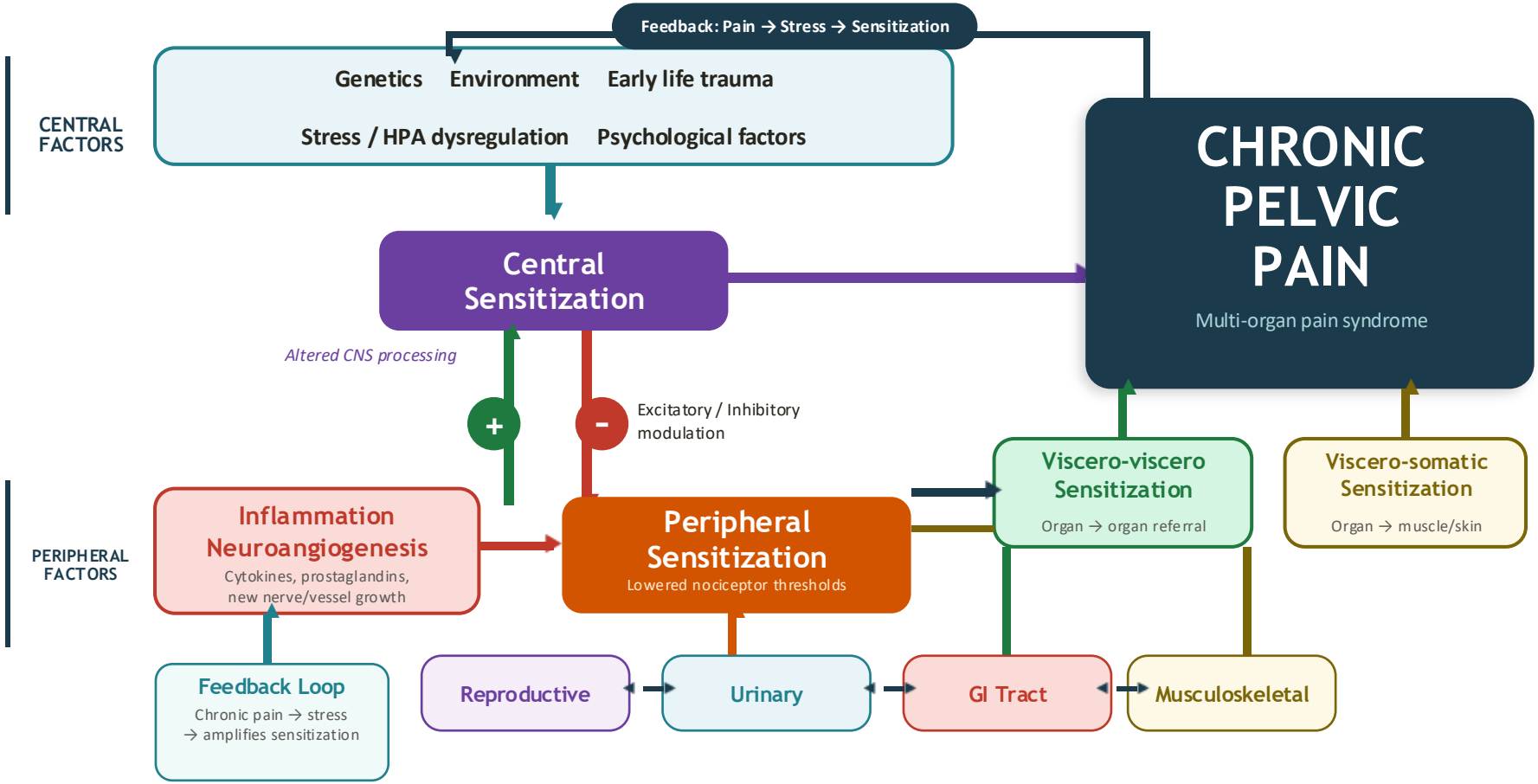
Organ-to-organ pain overlap via shared spinal pathways



Key insight: T10–L1 afferents innervating the uterus and adnexa also supply the lower ileum, sigmoid, and rectum — pain can originate from any of these structures.

Source: BioMed Res Int 2014, MedCentral 2022

Pathophysiology of Chronic Pelvic Pain (Endometriosis model)



Source: As-Sanie S, 2017 | Amber Charoen, MD

GI Conditions in Pelvic Pain

Functional, structural, and inflammatory GI disorders in the differential

IBS and Chronic Pelvic Pain

Evidence of Overlap

50% of women with CPP have IBS (median across studies)

– IBS and CPP co-occur more than chance ($p<0.01$)
– IBS patients have 2x somatic comorbidities
– Somatization predicts IBS-CPP overlap
– Women with IBS report more dyspareunia, headaches, bladder discomfort
– Similar prevalence, psychiatric comorbidity, abuse history

Shared Pathophysiology

Visceral hypersensitivity
Heightened gut sensitivity extends to pelvic floor and adjacent organs

Abnormal muscle contractions
Intestinal dysmotility extends to pelvic floor muscles

Central sensitization
Persistent pain alters CNS processing; amplifies perception in both GI and pelvic domains

Psychosocial factors
Depression, anxiety, abuse history common to both and may represent the pathophysiologic link

Source: J Clin Gastroenterol 2014, World J Gastroenterol 2007

Pelvic Floor Dyssynergia

A key GI diagnosis in the evaluation of chronic pelvic pain

Definition (Rome IV)

Inappropriate contraction of the pelvic floor during defecation with adequate propulsive forces.

Diagnostic criteria:

1. Functional constipation or IBS-C met
2. Impaired evacuation on ≥2 of 3 tests:
   – Abnormal balloon expulsion test
   – Abnormal anorectal manometry
   – Impaired evacuation on defecography

Symptoms ≥3 months, onset ≥6 months prior.

Pelvic Pain Connection

Common symptoms:

– Chronic straining at stool
– Incomplete evacuation
– Digital maneuvers to facilitate BM
– Perineal and rectal pain/pressure
– Associated depression and anxiety

Why it causes pelvic pain:

Chronic puborectalis/levator ani spasm → myofascial pelvic pain, exacerbated by shared S2–S4 innervation with bladder and reproductive organs.

Source: Rome IV Criteria, Curr Gastroenterol Rep 2017

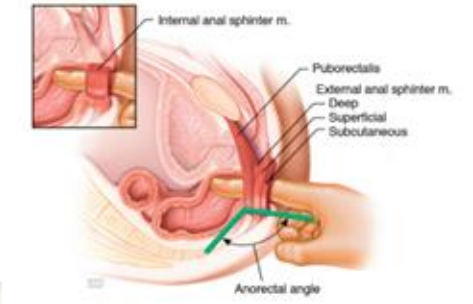
Functional Anorectal Pain Disorders

Levator Ani Syndrome

– Chronic rectal pain/ dull aching, worse with sitting,
– Episodes ≥30 minutes, often hours
– Puborectalis tenderness on DRE
– Exclude IBD, abscess, fissure
– Tx: biofeedback, sitz baths, muscle relaxants

Unspecified Anorectal Pain

– Meets levator ani criteria
– No tenderness on puborectalis traction
– Diagnosis of exclusion
– Tx: neuromodulators (TCAs, gabapentinoids)



Proctalgia Fugax

– Recurrent rectal pain, not related to defecation
– sudden and sharp
– Lasts seconds to minutes (max 30 min)
– No pain between episodes
– Exclude structural causes
– Tx: reassurance, salbutamol, topical diltiazem

Source: Rome IV Criteria, Mayo Clin Proc 2016, Tally N et al American Journal of Gastroenterology 2008

Other GI Conditions in Pelvic Pain

Inflammatory Bowel Disease

– Perianal Crohn's: fistulae, abscesses, strictures
– 10–30% of CD patients develop pelvic abscesses
– Rectovaginal fistulae in ~10% of anorectal CD
– Colorectal inflammation → rectal/perineal/back pain

Endometriosis – GI Overlap

– >90% present with GI symptoms first
– 83% report bloating as presenting symptom
– Associated with SIBO
– Bowel lesions → fibrosis, luminal narrowing
– Can mimic IBS, CRC, or appendicitis

Additional GI Causes to Consider

– Chronic appendicitis
– Colorectal carcinoma
– Chronic constipation
– Rectal Intussusception / Prolapse
– Chronic anal fissure
– Diverticular disease
– Hernias (inguinal, obturator)
– Adhesive disease (post-surgical)

CAPS (Centrally Mediated Abdominal Pain Syndrome)

Differential Diagnosis

Distinguishing GI from non-GI causes of chronic pelvic pain

GI vs. Non-GI Pelvic Pain

Feature	Suggests GI Etiology	Suggests Non-GI Etiology
Pain timing	Related to meals, defecation, bowel distension	Related to menses, intercourse, urination
Associated Sx	Bloating, altered bowel habits, rectal bleeding, tenesmus	Dysuria, vaginal discharge, menstrual irregularity, dyspareunia
Physical exam	Levator tenderness, fissure, hemorrhoids, rectal mass	Adnexal mass, uterine tenderness, abdominal wall triggers
GI treatment response	Improves with fiber, Low FODMAP diet, antispasmodics, neuromodulators, biofeedback	No response to GI-directed therapies
Cyclical pattern	Related to eating or stool consistency	Catamenial (endometriosis), worse with bladder filling
Red flags	Rectal bleeding, weight loss, iron deficiency, family hx CRC/IBD	Post-menopausal bleeding, pelvic mass, hematuria, neuro deficits

GI Diagnostic Workup

1	**History and Physical**	Bowel symptom history, dietary triggers, menstrual correlation, abuse history. DRE for tone, puborectalis tenderness, masses.
2	**Laboratory Studies**	CBC, CRP/ESR, celiac serologies, fecal calprotectin, C. diff. Thyroid function if constipation-predominant.
3	**Endoscopy**	Colonoscopy if red flags (bleeding, weight loss, family hx). Rule out IBD, neoplasia, solitary rectal ulcer.
4	**Anorectal Physiology**	HR anorectal manometry: resting tone, squeeze, dyssynergia pattern. Balloon expulsion test. Rectal sensory testing.
5	**Imaging**	MR defecography for pelvic floor. CT enterography if Crohn's suspected. Pelvic MRI for structural pathology.

Source: Gastro Hep Adv 2024, Gastroenterol Clin N Am 2008

Treatment Approach

GI-directed and multidisciplinary management strategies

TREATMENT STEP 1

Dietary & Lifestyle Interventions

Low FODMAP Diet

IBS Evidence

First-line therapy for IBS-C, IBS-D, IBS-M. Targets fermentable carbs (Oligos, Disaccharides, Monos, Polyols) that trigger visceral hypersensitivity.

Endometriosis + CPP Evidence

60% response rate in endo patients vs. 26% control. **65%** reported reduced chronic pelvic pain. GI symptom severity 40% lower vs. control diet (Monash 2025).

75% of women with endometriosis have GI symptoms that respond to dietary modification

Additional Dietary & Lifestyle Strategies

Anti-Inflammatory Diet

↑ vegetables, omega-3s, fiber — may reduce IBD and endo-related pelvic pain

Trigger Avoidance

Caffeine, alcohol, spicy foods, acidic items — bladder and bowel pain triggers

Hydration + Fiber Optimization

Prevents constipation-driven pelvic pressure; soluble fiber for IBS-D regulation

Bowel Habit Regulation

Timed defecation, squatting posture, biofeedback preparation

TREATMENT STEP 2

Pharmacologic, Behavioral and Physical Therapy — GI-Directed

Behavioral and Physical

Biofeedback therapy (first-line for dyssynergia)
– 60–80% success rate
– Sustained improvement at 1 year (RCT)
– Normalizes dyssynergia pattern
– Effective with or without concurrent IBS

Pelvic floor physical therapy
– Myofascial release, trigger point therapy
– Addresses levator ani spasm

Pharmacologic

Neuromodulators
– TCAs (amitriptyline, nortriptyline): visceral analgesic
– SNRIs (duloxetine): dual pain/mood benefit
– Gabapentinoids: neuropathic component

Antispasmodics
– Dicyclomine, hyoscyamine for IBS spasm

Disease-specific therapy
– IBD: biologics, immunomodulators, antibiotics
– Constipation: secretagogues (linaclotide)
– Levator ani: muscle relaxants, diltiazem

Source: Am J Gastroenterol 2010, Bioelectronic Med 2023

GI-Directed Procedures

Biofeedback Therapy

Anorectal biofeedback for PFD, levator ani syndrome, and dyssynergic defecation — **22% fewer analgesics** used post-therapy

Botulinum Toxin Injections

High-tone PFD, levator ani, puborectalis hypertonia — relaxes spastic pelvic floor muscles

Sacral Neuromodulation

Fecal incontinence, refractory urgency, functional pelvic pain — targets S2–S4 nerve roots

Superior Hypogastric Plexus Block

For refractory visceral pelvic pain from bowel, bladder, or gynecologic sources — refers to interventional pain

TREATMENT STEPS 4

Multidisciplinary Management

Avoid diagnostic tunnel vision — coordinate care across specialties

Gynecology

Endometriosis, PID, ovarian pathology, hormonal therapy

Gastroenterologist

IBS management, anorectal physiology, biofeedback, colonoscopy, IBD therapy, motility evaluation

Urology

Interstitial cystitis, bladder pain syndrome, pelvic congestion

Pain Medicine

Nerve blocks, neuromodulation, trigger point injections

PT / Rehab

Pelvic floor PT, myofascial release, biofeedback support

Psychology

CBT, trauma-informed care, coping strategies

Source: MedCentral 2022, Semin Reprod Med 2023

Key Takeaways

1. GI disorders account for a large proportion of chronic pelvic pain: IBS, pelvic floor dyssynergia, and functional anorectal pain.
2. Shared innervation (T10–S4) creates organ-to-organ pain overlap via visceral cross-sensitization and convergence on the spinal dorsal horn.
3. IBS and CPP likely represent overlapping entities — somatization and central sensitization are key pathophysiologic links.
4. Thorough GI evaluation (DRE, anorectal manometry, endoscopy, imaging) can identify treatable causes in "idiopathic" pelvic pain.
5. Biofeedback is first-line for dyssynergic defecation (60–80% success); neuromodulators address visceral hypersensitivity.
6. Multidisciplinary collaboration avoids tunnel vision and improves patient outcomes.

Selected References

1. Bharucha AE, Lee TH. Anorectal and Pelvic Pain. Mayo Clin Proc. 2016;91(10):1471-1486.

2. Giamberardino MA et al. Visceral Pain: The Neurophysiological Mechanism. Handb Exp Pharmacol. 2011.

3. Origoni M et al. Neurobiological Mechanisms of Pelvic Pain. BioMed Res Int. 2014.

4. Williams RE et al. IBS and Chronic Pelvic Pain. J Clin Gastroenterol. 2014;48(1):52-57.

5. Matheis A et al. IBS and CPP: A Singular or Two Syndromes? World J Gastroenterol. 2007;13(25):3446.

6. Rao SS et al. Long-term Efficacy of Biofeedback for Dyssynergic Defecation. Am J Gastroenterol. 2010.

7. Rome IV Criteria for Functional GI Disorders. The Rome Foundation. 2016.

8. Vitton V et al. Update on Rome IV for Colorectal Disorders. Curr Gastroenterol Rep. 2017;19(4):15.

9. Rana MV et al. Pelvic Pain in Crohn's Disease. J Clin Med. 2023;12(2):555.

10. McClurg AB, Carey ET. Evaluation and Management of CPP. Semin Reprod Med. 2023;41(4):175-188.

Thank You

Questions and Discussion

Email: **Amber.Charoen@gmail.com**