Gastrointestinal and Hepatic Disease in Fibromyalgia

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KEYWORDS

• Irritable bowel syndrome • Central sensitization • Fibromyalgia • Abdominal pain • Hyperalgesia

KEY POINTS

- Considerable overlap exists between fibromyalgia (FM) and functional gastrointestinal diseases such as irritable bowel syndrome (IBS) in terms of pathophysiology, lack of a biomarkers, and treatment options.
- Central and peripheral sensitization accounts for both visceral and somatic pain experienced by patients with FM and IBS.
- Pharmacologic, psychological, and dietary treatments for FM and IBS are similar and effective. The degree of improvements in patients' quality of life depends on the strength of the patient-physician relationship.
- Hepatic disease in patients with FM is rare, although comorbid conditions such as obesity may increase the risk of nonalcoholic steatohepatitis.

OVERVIEW

Gastrointestinal (GI) symptoms and conditions are common in fibromyalgia (FM), as well as in other rheumatologic diseases, including connective tissue disorders, spondylarthritides, and rheumatoid arthritis. FM has been linked to several GI and hepatology diseases, including but not limited to irritable bowel syndrome (IBS), gastroesophageal reflux disease, functional dyspepsia, and viral hepatitis. The most well-substantiated evidence and literature relates to IBS, a common GI condition characterized by abdominal pain, and altered bowel habits otherwise unexplained by

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alternative methodologies or diagnoses. Like FM, the underlying pathogenesis for IBS remains unclear, although dysregulation within the brain-gut axis resulting in a hyperalgesic state has been hypothesized.

Aside from its pathogenesis, IBS and FM share many other similarities, including a female predominance, fatigue, insomnia, and well-described psychiatric aspects. These common manifestations and pathogenesis of disease serve as a foundation for overlapping treatment modalities, including pharmacologic and psychological therapies (see later discussion). Indeed, the tremendous heterogeneity in terms of pathogenesis and symptom profile among patients with both disorders prevents the use of a universally applicable treatment algorithm. Instead, treatment using trial-and-error and a multidisciplinary approach is required for optimal management.

GASTROINTESTINAL DISORDERS IN FIBROMYALGIA

Introduction

GI diseases are commonly seen in FM in addition to all other rheumatologic diseases and disorders, including connective tissue disorders such as system lupus erythematosus, scleroderma, Ehler-Danlos syndrome, and rheumatoid arthritis. This article focuses predominantly on the overlap between FM and IBS, a functional disorder that not only overlaps with other GI disorders such as functional dyspepsia but also with non-GI disorders such as temporomandibular joint disorder, interstitial cystitis, and chronic fatigue syndrome. Evidence is lacking about whether patients with FM have higher rates of other common GI disorders (besides IBS). These disorders may include gastroesophageal reflux disease, esophageal hypersensitivity, and functional dyspepsia. Interestingly, studies have suggested an alteration of central brain activation, immune dysfunction, and an enhanced pain perception that is similar for these visceral pain disorders and FM (a somatic pain syndrome) with similar psychosocial risk factors that impair patient's quality of life.[1] This article discusses how IBS and FM have shared multifactorial bases and how, therefore, their treatments are similar.

Irritable bowel syndrome

Overview IBS is a common, widely prevalent GI condition characterized by abdominal pain associated with an alteration in bowel habits otherwise unexplained by anatomic, structural, or metabolic pathologic assessment.[2,3] Frequently, nonspecific GI symptoms of bloating, flatulence, nausea, and fecal urgency are also present. The diagnosis is established by the fulfillment of the recently updated Rome IV guidelines in which patients manifest recurrent abdominal pain on average at least 1 day per week in the last 3 months with symptom onset for at least 6 months prior, associated *with 2 or more of the following criteria*:[4,5]

1. Relation to defecation
2. Change in form (appearance) of stool
3. Change in stool frequency.

The underlying pathogenesis of patients with IBS remains unclear, although dysregulation within the brain-gut axis is generally regarded as the central, unifying hypothesis.[6] The dysregulation is likely multifactorial and results from a combination of impairment in gut motility, visceral hypersensitivity, altered mucosal and immune function, change in gut microbiota, and central processing of sensory input.[7,8]

Classification of irritable bowel syndrome Given that IBS is a clinical diagnosis that involves multiple subjective components, multiple different disease indices have been developed to afford better quality research and treatment outcomes. These