Understanding Disordered Eating Risks in Patients with Gastrointestinal Conditions

EMERGING DATA ARE ILLUMINATING the connection between gastrointestinal (GI) conditions such as disorders of gut–brain interaction (DGBI), including irritable bowel syndrome (IBS), celiac disease, and inflammatory bowel disease (IBD) with disordered eating.[1-6] In turn, eating disorders (EDs), such as anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) can lead to alterations in GI function.[7,8] In a systematic review of 94 studies with an accurate ED diagnosis, the weighted mean of lifetime ED were 8.4% (range, 3.3% to 18.6%) for women and 2.2% (range, 0.8% to 6.5%) for men. A doubling in prevalence over the study timeline was also noted; the weighted means of point ED prevalence increased from 3.5% for the 2000-2006 period to 7.8% for the 2013-2018 period.[9] It is important to interpret prevalence rates for men with caution because they are underrepresented in ED research and less likely to be screened or referred for treatment.[10]

Only about 20% of patients with EDs will seek treatment, often when the disorder is more severe.[11] Due to the significant physical toll caused by an ED, patients may seek treatment for GI, exocrine, or cardiovascular complaints instead of the ED, placing them at serious health risk.[11] Therefore, it is imperative that all health care professionals, including various specialists in the field of gastroenterology, regularly assess for eating behaviors to meet their patient's clinical needs.[12]

Because individuals with EDs may attempt to hide their illness and avoid health care interventions, a gastroenterology visit can too often be a missed opportunity to assess a patient for disordered eating.[13] Although it is not the job of gastroenterology providers to treat EDs, it is important to identify at-risk patients and direct them to the appropriate treatment. This article highlights the importance of recognizing disordered eating in patients with GI conditions, provide screening guidance, and review the importance of a multifaceted team approach for treatment.

*This article was written by **Megan E. Riehl**, PsyD, MA, assistant professor of medicine, University of Michigan, Ann Arbor; and **Kate Scarlata**, MPH, RDN, For a Digestive Peace of Mind, LLC, Medway, MA.*

https://doi.org/10.1016/j.jand.2021.03.001
Available online xxx

A Primer: Disordered Eating vs ED

Disordered eating is observed in individuals who do not follow eating patterns that are deemed the cultural norm, such as skipping meals, limiting many foods, or following restrictive diets.[4] Patients with DGBIs, such as IBS, may engage in disordered eating as a means of avoiding or preventing symptoms.[4] For example, up to two-thirds of patients with IBS attribute foods they eat to abdominal symptoms and bowel changes, leading to increased interest in restrictive diets in hopes of minimizing postprandial discomfort.[14] Severity of eating behaviors, motivation driving those behaviors, and negative medical or psychological effects caused by those behaviors distinguish disordered eating from an ED (Figure 1).[4]

The main EDs described with detailed diagnostic criteria in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) include AN, which presents with excessive dieting leading to severe weight loss and associated with pathological fear of becoming fat, BN associated with episodes where loss of control over eating is followed by inappropriate behaviors such as self-induced vomiting or laxative abuse to avoid weight gain and BED. Unlike simply the act of overeating, BED is more severe and is also associated with episodes where the individual experiences loss of control over their eating with significant physical and psychological problems.[15] It is important for gastroenterology providers to be aware of these diagnoses, considering a staggering 41% to 52% of patients with EDs also have IBS.[16]

In the revised edition of the DSM-5, diagnoses that were previously classified as feeding disorder and EDs of infancy or early childhood, including pica, rumination disorder, and avoidant/restrictive food intake disorder (ARFID) removed age considerations and are now considered independent diagnostic feeding disorder and ED categories. Pica is characterized by eating nonfood, nonnutritive substances and rumination disorder is defined as repeated, effortless regurgitation of ingested food. ARFID shares characteristics similar to other EDs such as food restriction and avoidance, medical risks, and comorbid anxiety, but the founding psychopathology of ARFID makes it distinct in that it lacks an overvaluation on weight or body size.[15,17]

Other specified feeding and eating disorders (OSFED) are a clinical category in the DSM-5 used to describe disordered eating behaviors that do not fully meet criteria for a specific eating disorder. Orthorexia, where an individual has an obsession with eating foods that they deem as healthy, may be given a diagnosis of OSFED—atypical anorexia or OSFED-other—but there is debate in the field over the diagnostic criteria.[18] In addition, it has been noted that orthorexia is less commonly captured in clinical practice because individuals typically will not seek treatment unless they begin to experience significant physical or psychological distress[18]; yet GI clinicians are encouraged to be aware of orthorexia given the potential consequences of recommending diet therapies, further placing a microscope on food.[19]

Psychiatric Comorbidities and EDs

Unsurprisingly, EDs are associated with comorbid psychiatric disorders;

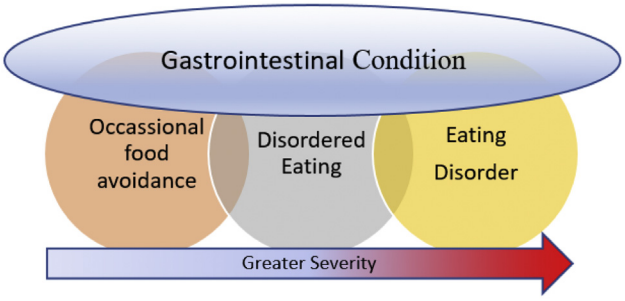

Figure 1. Food avoidance related to food intolerance, such as removing lactose-containing milk from a diet due to lactose intolerance, is not pathological. The severity of the food avoidance provokes greater health risk. Not all patients with disordered eating have an eating disorder; however, all patients with eating disorders will engage in disordered eating. Weight and body mass index do not always correlate with eating disorder risk and should not be the only red flag or metric to initiate screening.[20] Careful assessment of disordered eating may prevent the development of an eating disorder and guide proper treatment for comorbid gastrointestinal conditions.

similarly, there are increased rates of affective disorders among patients with GI conditions.[12,21-23] The act of eating often creates significant stress for both groups of patients. With the strong connection between food and the experience of digestive conditions, careful attention and assessment must be made.[16,24] For example, individuals reporting more food triggers experience a reduced quality of life and report more severe IBS symptoms.[25] Further, depression and anxiety symptoms can often exist secondarily to an ED and it may be difficult to tease apart primary depression and anxiety disorders from depression and anxiety symptoms due to malnutrition; both share low energy, psychomotor agitation, irritability, poor concentration, and low mood.[12] Proper screening, empathic conversations about eating behaviors, and the potential dangers for missing an ED diagnosis early in patients with GI conditions are necessary.[12] The lethal nature of EDs is illustrated in a recent meta-analysis concluding that suicidal behaviors are a significant concern in those with EDs, with awareness critical for targeted prevention and opportunities to employ harm reduction interventions.[26] It should be noted that the nature of severe psychological comorbidities in ARFID, rumination disorder, and pica, especially in GI patient populations, is limited.

Influence of EDs on GI Symptoms

The intersection of EDs and alterations of GI function is complex. It is difficult to distinguish whether digestive conditions or symptoms are inherent manifestations of EDs or the outcome of malnutrition stemming from ED behaviors such as laxative abuse, self-induced vomiting, or the restriction of food.[8] Alternatively, a clinician has to factor in the possibility that the disordered eating may result from the influence of the ED on GI function. Likely risk factors for these GI manifestations include severe malnourishment from ED-related symptoms such as self-induced purging, loss of appetite, dysphagia, constipation, bloating, or refeeding.[27-29] Malnutrition and muscular atrophy can lead to pelvic floor dysfunction, which has been shown to be a predictor of abdominal distention in EDs.[27,30] Malnutrition and associated metabolic myopathy and electrolyte imbalance have been shown to influence gastric motility, gastric emptying, and intestinal transit leading to significant impairments.[27]

Research in patients with AN and BN highlights the complex nature of EDs and GI conditions where nausea, vomiting, and gastric fullness have been associated with slow gastric emptying in some studies with patients with AN,[31,32] whereas others have not found a connection in body weight, gastric emptying, and GI symptoms in patients with AN and BN.[33-35] Current literature highlights that GI symptoms are therefore not reliable indicators of gastric emptying among those with EDs and this is an area requiring further research.[8] Considerations for the numerous GI manifestations associated with EDs are highlighted in Figure 2.

GI Conditions and the Relationship to Disordered Eating and EDs

GI conditions are associated with greater risk of disordered eating and eating disorders (Figure 1). A systematic review by Satherley and colleagues[37] found that 23.7% of individuals with GI conditions display disordered eating, defined as behaviors of food restriction, meal skipping, or overeating not yet meeting criteria for an ED. In IBS, Reed-Knight and colleagues[38] found that food avoidance, irregular eating, and skipping meals is common in patients with IBS. In this study of nearly 100 adolescents with IBS, eating-related symptoms were found to be exponentially higher in IBS (91.7%) compared with healthy controls (28%). This study revealed that 43% of IBS patients tried to control eating-related symptoms by not eating any food, even when hungry. It is important to note that the presence of disordered eating in this cohort did not result in significant weight change, serving as a reminder not to utilize weight change as the only measure to determine the presence of disordered eating.[38] In celiac disease, clinicians should be aware of patient concerns regarding weight gain after diagnosis and treatment with a gluten-free diet as well as the potential for hypervigilance around applying a gluten-free diet. Prevalence for disordered eating is suggested to occur in 22% to 29% of individuals with celiac disease.[39] Further, in a recent study assessing the frequency of EDs in patients with chronic constipation (79% women), revealed that 19% had clinically significant ED pathology. ED was associated with higher general anxiety scores, as well as greater GI-specific anxiety (a cognitive, affective, and behavioral response that results from fear of GI symptoms and the circumstances in which these visceral symptoms occur).[3] Screening for ED in individuals with chronic constipation is recommended, particularly when bloating and abdominal pain are present.[3]

New data reveal the presence of suspected ARFID in GI clinics. Although one should interpret this research with caution given screening measures for

Mouth and salivary glands	Gingivitis, periodontitis, cheilosis, oral infections, salivary gland enlargement
Esophagus	Mallory-Weiss tear, gastroesophageal reflux disease, esophagitis, esophageal motility disorders, acid regurgitation, dysphagia, achalasia
Stomach	Gastric bezoar, gastric dilation, dysmotility, delayed gastric emptying for liquids and solids, postprandial fullness, dyspepsia, disturbance in peripheral hormone secretion
Small + large intestine	Superior mesenteric syndrome, functional and defecatory disorders such as irritable bowel syndrome or functional constipation, pelvic floor dyssynergia, rectal prolapse, abdominal distention, bloating, feeling of anal blockage, increased and decreased colonic transit times
Pancreas	Acute pancreatitis
Liver	Elevated transaminases, nonalcoholic fatty liver disease

Figure 2. The influence of eating disorders on the gastrointestinal tract.[7,8,27,36]

ARFID are not yet validated in DBGIs,[40] preliminary data by Zia and colleagues[41] revealed approximately 21% of patients with functional GI disorder in their sample met criteria for ARFID. In the first large prospective study screening adult gastroenterology patients for ARFID (n = 317; 65.6% women), 19.6% screened positive in the setting of a functional bowel and motility clinic.[42] Further, patients with IBS were two times more likely than non-IBS patients to screen positive. Within the same group, a multidisciplinary team completed a retrospective analysis of 223 adult patients receiving psychogastroenterology services and found that 12.6% (23 of 28 patients were women) of patients met DSM-5 criteria for ARFID.[20] In that study, a majority of patients (71.4%) screening positive for ARFID had been prescribed a restrictive diet without fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (low-FODMAP diet). It has also been found in 1 study that high adherence to the low-FODMAP diet in patients with IBS was associated with ED behaviors.[43] These results underline the importance of assessing risk of maladaptive eating in this population, as well as the need for evidence-based guidelines for clinical practice regarding which IBS patients are appropriate for a low-FODMAP diet.

It should be noted that there are significant limitations in interpreting questionnaire studies that assess prevalence for ARFID in patients with GI conditions. These questionnaires do not directly assess GI symptoms, such as early satiety, which can be the catalyst for changes in the patient's eating habits and would be skewed in patients with DGBIs. Further, GI symptoms in DGBIs are often triggered by food secondary to visceral hypersensitivity to normal digestive function. Therefore, many patients following prescribed restrictive diets will score points even when disordered eating is not a factor, leading to increased scores on ARFID questionnaires even in the absence of true disordered eating. To best serve patients with GI conditions, a validated tool to assess EDs and disordered eating in this population is needed.

Rumination syndrome is a functional GI diagnosis that can be missed, delayed, or untreated leading to significant health consequences.[44] In the GI literature, rumination syndrome is described as the subconscious physiological contraction of abdominal muscles while voluntarily relaxing the lower esophageal sphincter, which allows food and liquid to be regurgitated leading to re-chewing, re-swallowing, or spitting out.[45] The true prevalence of rumination syndrome is likely underestimated due to lack of physician awareness; and although it is perceived as predominantly a diagnosis seen in women, this is likely related to women seemingly more likely to seek health care for DBGIs.[46] Without careful clinical assessment, the observation of sudden or gradual weight loss in the setting of regurgitating food can leave patients vulnerable to a misdiagnosis of AN, further perpetuating distress. Murray and colleagues[44] underscore the importance of making a definitive diagnosis and treating with a behavioral intervention like diaphragmatic breathing. In clinical practice, this author (M.E.R.) has successfully implemented diaphragmatic breathing treatment over the course of one to three sessions often after patients have experienced incorrect diagnoses such as AN, extensive and invasive medical workups, medication trials, and in the worst case, hospitalization with a consult for enteral nutrition.[47]

Physicians must be careful in making a diagnosis of an ED because GI symptoms can mimic a feeding disorder and ED.[48] In a study by Murray and colleagues[48] assessing for the presence of feeding disorders and EDs in patients with symptoms of gastroparesis and dyspepsia, 55% of patients had clinically significant FED symptoms, particularly symptoms of ARFID (23% to 40%); the likelihood of having feeding disorder and ED symptoms increased with more severe gastroparesis symptoms, but not gastric retention. These emerging data highlight the complexity of these already difficult to treat GI conditions while adding disordered eating assessment to the treatment algorithm. Furthermore, it is plausible that factors other than gastric retention may play a role in symptomology such as gut microbial alterations.

There is paucity of research assessing eating behaviors in IBD. However, a systematized review focused on comorbidity between IBD and ED in 2017 found Crohn's disease and AN most frequently reported.[49] The vast majority of comorbid IBD and ED was found in women; and aspects of IBD care, such as corticoid refusal, medication abandon, and deliberate exacerbation of IBD symptoms were reported to augment weight loss.[49] To date, our understanding of the connection between individuals with IBD and ED is unfolding, but recognizing associations are critical due to the potential that the relationship of the 2 can worsen the prognosis of both.[50] Further, diet may play a role in changing the microbiota,

Variable	Level 1: Outpatient lower risks	Level 2: Intensive outpatient	Level 3: Partial hospitalization (full-day outpatient care)	Level 4: Residential treatment	Level 5: Higher risks Inpatient treatment
Medical status	Medically stable	Medically stable	Medically stable	Medically stable w/ o need of nasalgastro tube, daily laboratory tests	Heart rate <40 bpm; blood pressure <90/60 mm Hg; glucose <60 mg/dL; potassium <3 mEq/L; temperature <97°F; dehydration; poorly controlled diabetes
Weight	>85% healthy body weight	>80% healthy body weight	>80% healthy body weight	<85% healthy body weight	<85% healthy body weight; acute weight loss with food refusal even if not <85% healthy body weight
Motivation to recover	Fair to good	Fair	Partial motivation, preoccupied with intrusive, repetitive thoughts >3 h/d	Poor to fair motivation, preoccupied with intrusive repetitive thoughts 4-6 h/d	Very poor to poor motivation
Co-occurring disorders	Presence of comorbid condition may influence choice of care	Presence of comorbid condition may influence choice of care	Presence of comorbid condition may influence choice of care	Presence of comorbid condition may influence choice of care	Any existing psychiatric disorder (eg, risk of self-harm, severe depression)
Structured program needed for eating and weight restoration	Self-sufficient	Self-sufficient	Needs structure to gain weight	Needs supervision at all meals or will restrict eating	Needs supervision during and after all meals or requires nasalgastro feeding tube
Purging behavior (use of laxatives or diuretics, forced vomiting)	Can reduce purging in unstructured setting, no medical complications	Can reduce purging in unstructured setting, no medical complications	Can reduce purging in unstructured setting, no medical complications	Requires some support to inhibit purging	Needs supervision; multiple daily episodes of purging

Figure 3. Understanding levels of care in adult patients with eating disorders. Adapted from https://www.nationaleatingdisorders.org/toolkit/parent-toolkit/level-care-guidelines-patients. Accessed February 25, 2021.

metabolome, host barrier function, and innate immunity in patients with IBD.[51]

Considerations for Dietary Interventions in GI Conditions

Recent literature points to a bidirectional relationship between EDs and patients with celiac disease, Crohn's disease, and ulcerative colitis in women; however, the authors were careful to highlight that the associations could be related to dietary restrictions and eating behaviors.[6] GI symptoms associated with celiac disease, DBGIs, and IBD can all be managed with dietary modifications.[52] The specifics of the recommended dietary interventions and foods associated with triggering symptoms vary across GI conditions. For example, patients with celiac disease must follow a lifelong strict, gluten-free diet, whereas patients with DBGIs and IBD often require trial and error[53] with long-term goals of identifying a diet that is conducive to the individual's lifestyle

and as liberal as possible. Diet-controlled GI disorders and related behaviors associated with food monitoring may place individuals at risk for disordered eating.[37] Research in young adults sheds light on the importance of early screening in patients with diet-controlled health conditions because they have been found to be twice as likely to be diagnosed with an ED compared with controls.[54]

Disordered Eating Assessment in GI Conditions

GI conditions can be managed with dietary interventions; therefore, careful assessment of the patient's application of these interventions must be included in regular office visits. Patients demonstrating significant restrictions, dysmorphia, unintentional weight loss, food-related fears and ongoing GI symptoms despite restriction of food triggers highlight warning signs (Figure 1).[4] It is recommended that GI clinicians be prepared to talk with patients pertaining to concerns of restrictive eating (see Figure 3). The 5-question SCOFF (Sick, Control, One Fat, Food) questionnaire[55] can be used as a means of facilitating a conversation with patients suspected of disordered eating, where the provider believes a referral for more comprehensive ED treatment is recommended.[56] The Coeliac Disease Food Attitudes and Behaviors Scale is a validated tool to assess disordered eating in celiac disease. High scores are associated with psychological distress and impaired quality of life.[39] Careful assessment about eating behaviors in patients with organic GI disease should be routine in clinic visits. For example, food preoccupation, dietary restriction, and laxative use, purging are documented ED behaviors in celiac disease. Although food preoccupation and dietary restriction in IBS and IBD are ED behaviors in this population, other potential risk of ED or disordered eating include body shame, significant fear associated with abdominal pain with eating, as well as preoccupation of dietary management.[57]

Once an individual has been screened for EDs and/or disordered eating, the next step is to understand the level of care the patient may require depending on the severity and risks associated with their ED clinical presentation. Figure 3 provides some general guidance on ED levels of care.

Further, in patients with quiescent IBD, up to 30% may experience IBS-like symptoms, stimulating interest in the low-FODMAP diet; however, undernutrition commonly observed in IBD should caution the suggestion of any restrictive diet unless the patient is carefully supervised by a registered dietitian.[52] When providers make dietary recommendations as part of IBD treatment, regular evaluation of eating behaviors, beliefs about eating, dietary changes, and implications on quality of life must be monitored. The Food-Related Quality of Life-29 questionnaire was developed specifically to assess such domains in patients with IBD.[58]

To more thoroughly evaluate the presense and severity of feeding disorders and EDs, the Pica, ARFID, and Rumination Disorder Interview was recently developed; in an effort to streamline individualized treatment plans, the Pica, ARFID, and Rumination Disorder Interview includes subscales for sensory sensitivity, fear of adverse consequences, and lack of interest in eating or food.[59] In a multidisciplanary team, a mental health professional will likely be the team member to complete the assessment with the patient.

Guiding Patients to Appropriate Treatment

It will not be the responsibility of GI clinicians to treat EDs; however, guiding patients to proper treatment is imperative. Mental health professionals with expertise in psychogastroenterology who are apart of multidisciplanary treatment teams in outpatient GI practices are not typically trained in the treatment of EDs. However, they can aid in screening, diagnosis, and recommendations for more comprehensive treatment.[60,61] In patients with active ED, psychological treatment considerations will be determined by severity and patient motivation.[62] Treatment options progress from outpatient treatment with a mental health professionals specializing in EDs, to intensive outpatient day treatment or partial residential treatment (typically for patients who are medically stable) to inpatient hospitalization (offering medical refeeding and monitoring to assist with medical stablization).[62-64] Although psychological treatment approaches will vary by age and ED type, cognitive behavioral therapy interventions are implemented for adults.[65]

Outpatient Care for Disordered Eating

Screening and open dialogue begins the conversation with patients with GI conditions about available treatment options for improving their eating behaviors, food related anxiety, and management of GI symptoms. Understandably, restrictive diets, such as the low-FODMAP diet are not recommended for patients demonstrating disordered eating in the setting of GI dysfunction.[59,66] Patients may consider evidence-based psychological interventions such as gut-directed hypnotherapy with a trained mental health expert.[66,67] In patients not meeting criteria for EDs, a multidisciplanary approach to aid a patient in expanding his or her diet can include the patient's gastroenterologist, a mental health professional specializing in the treatment of GI conditions, and a registered dietitian (Figure 4).[68] Given the complexity of patients with GI conditions, when available, early integrative psychogastroenterology treatment should be considered.[61,69,70]

Best Practices for the Treatment of Disordered Eating and EDs

Patients with GI conditions with a history of ED should consider alternative behavioral interventions as part of their medical treatment. Regular discussion and screening pertaining to eating behaviors, GI symptoms, and quality of life should take place during clinic and virtual visits. This can guide appropriate treatment.

The psychological ramifications of eating restrictions and GI conditions can be devastating, overwhelming, and escalate from disordered eating to eating disorders (Figure 1). Validate the patient's experience and connect them to the appropriate multidisciplinary team (see Figure 4). For patients with an ED, be prepared to refer for more comprehensive ED treatment to meet the patient's clinical needs.

Although some GI conditions may benefit from weight loss, such as nonalcoholic fatty liver disease and

Medical provider	Mental health professional	Registered dietitian
Who: primary care physician, gastroenterologist, physician assistant, nurse practitioner • Ensure that the disordered eating behaviors are not entirely due to a co-occuring medical condition • Confirm that patient is medi cally stable for outpatient care • Inform goals of treatment (eg, liberalize diet, gain weight, improve deficiencies in nutrition)	Who: GI psychologist, behavioral health provider, disordered eating specialist • Complete a clinical interview to assess the presense and severity of the disordered eating • Develop treatment plan with patient • Delivery of behavioral interventions • Or, refer patients with more severe disordered eating or eating disorder for treatment with eating disorder specialist	Who: Registered dietitian with specialization in gastroenterology and/or disordered eating • Complete full nutrition assess ment, including disordered eating or eating disorder screening • Provide education and treat ment plan that encourages food variety, adequate nutrition, and regular meal times • Refer to mental health provider with eating disorder expertise in patients with severe disordered eating or eating disorder

Conversation starters for eating behaviors in the clinic visit[a]	
SCOFF Questionnaire	**Additional questions to facilitate discussion of eating behaviors in a clinic visit**
(1) Do you make yourself sick because you feel uncomfortably full? (2) Do you worry you have lost control over how much you eat? (3) Have you recently lost more than 1 stone (14 lb) during a 3-mo period? (4) Do you believe yourself to be fat when others say you are too thin? (5) Would you say that food dominates your life? Scoring: each positive response to these questions yields a score of 1; a score of 2 or greater indicates an increased risk of ED behavior	(1) Does eating make you anxious? (2) How has your appetite been? (3) Do you ever skip meals as a way to avoid symptoms? (4) Do you feel full after a couple bites? (5) What time in the day do you typically have your first meal? (6) Do you eat a variety of foods or are you limited to certain things? (7) What kinds of beverages are you consuming throughout the day? (8) Do you spend much of your day thinking about food-related decision and meal planning? (9) Have you ever been diagnosed with an eating disorder? (10) I am afraid to eat outside my home (11) I enjoy going out for meals as much as I did before my diagnosis (12) I will only eat food that I have prepared myself (13) I have a lack of variety in my diet
[a]Keep in mind that adjustments in conversation starters may need to be made based on patient's eating behaviors and beliefs.	

Figure 4. The multidisciplanary team approach for disordered eating in patients with gastrointestinal (GI) conditions.

gastroesophageal reflux disease, discussions around weight control should be utilized with caution. The unintended consequences of weight stigma (prejudiced behaviors directed toward those living in larger bodies) can lead to reduced utilization of the health care system and can trigger disordered eating.[71] Assessment for disordered eating should take place in all patients regardless of body size.

Focus clinical attention toward the use of evidence-based interventions based on the medical, psychological

and nutrition needs of the patient. All members of a patient's treatment team will work together to provide the patient with a unified prespective on their goals, treatment progress, and long-term relapse prevention. It is the job of each specialist to assess the patient and provide a treatment recommendation. Goals of nutrition therapy may include weight restoration in underweight patients, normalization of eating patterns, achieve normal awareness of hunger and satiety, replete nutrient deficiencies, and educate on specific diet requirements for the GI condition (eg, gluten-free diet education for celiac disease).

Eating Disorder Risk Reduction: Prevention Efforts

Successful ED programs focus on key modifiable risk factors for EDs. Prospective studies have identified key risk factors, including belief in the cultural thin-ideal (called thin-ideal internalization), perceived pressure to be thin, body dissatisfaction, self-reported dieting, and negative affect (eg, depressive symptoms).[72] Prevention programs and clinical interventions should assess and focus on these risk factors in an effort to reduce risk. Most interventions geared toward prevention have been implemented in interactive group sessions. In patients with GI conditions specifically, providers aware of disordered eating risks, should screen and address these concerns early in the treatment plan to help mitigate the development of an eating disorder.[72] Research to help identity early eating disorder symptoms and barriers to treatments remain gaps in the literature.[72] ED prevention programs enhance the participants' knowledge of EDs but have shown only small improvements on the behavioral outcome variables, dieting behaviors, and general eating abnormalities for a range of population groups.[57,73]

CONCLUSIONS

GI conditions prompt food-related symptoms that can result in maladaptive eating. Individuals with GI conditions are at greater risk for disordered eating. Moreover, EDs can result in GI alterations and symptoms. It is the role of GI clinicians to assess for disordered eating and/or EDs, to ensure best outcomes. In overt EDs, an ED specialist should be consulted because these conditions require expert care. Details of how to find experts are reviewed elsewhere.[24] Furthermore, although not reviewed in this article, the exploration of the role of gut microbiota alterations in GI manifestations and disordered eating is emerging. This area of research deserves more attention to better understand the role of gut microbiome alterations in the etiology of these conditions from GI function to mental health and the potential for therapeutic microbiome targeted treatments.[74-76]

References

1. Singh P, Arora A, Strand TA, Leffler DA, Catassi C, Green PH, et al. Global prevalence of celiac disease: systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2018;16(6):823-836 e2.
2. Houghton LA, Heitkemper M, Crowell M, Emmanuel A, Halpert A, McRoberts JA, et al. Age, gender and women's health and the patient. *Gastroenterology*. February 15, 2016 [Epub ahead of print].
3. Murray HB, Flanagan R, Banashefski B, Silvernale CJ, Kuo B, Staller K. Frequency of eating disorder pathology among patients with chronic constipation and contribution of gastrointestinal-specific anxiety. *Clin Gastroenterol Hepatol*. 2020;18(11):2471-2478.
4. Harer KN. Irritable bowel syndrome, disordered eating, and eating disorders. *Gastroenterol Hepatol (N Y)*. 2019;15(5):280-282.
5. Passananti V, Siniscalchi M, Zingone F, Bucci C, Tortora R, Iovino P, et al. Prevalence of eating disorders in adults with celiac disease. *Gastroenterol Res Pract*. 2013;2013:491657.
6. Hedman A, Breithaupt L, Hubel C, Thornton LM, Tillander A, Norring C, et al. Bidirectional relationship between eating disorders and autoimmune diseases. *J Child Psychol Psychiatry*. 2019;60(7):803-812.
7. Hetterich L, Mack I, Giel KE, Zipfel S, Stengel A. An update on gastrointestinal disturbances in eating disorders. *Mol Cell Endocrinol*. 2019;497:110318.
8. Santonicola A, Gagliardi M, Guarino MPL, Siniscalchi M, Ciacci C, Iovino P. Eating disorders and gastrointestinal diseases. *Nutrients*. 2019;11(12).
9. Galmiche M, Dechelotte P, Lambert G, Tavolacci MP. Prevalence of eating disorders over the 2000-2018 period: a systematic literature review. *Am J Clin Nutr*. 2019;109(5):1402-1413.
10. Cottrell DB, Williams J. Eating disorders in men. *Nurse Pract*. 2016;41(9):49-55.
11. Eating disorders: innovation and progress urgently needed. *Lancet*. 2020;395(10227):840.
12. Treasure J, Duarte TA, Schmidt U. Eating disorders. *Lancet*. 2020;395(10227):899-911.
13. Smink FR, van Hoeken D, Hoek HW. Epidemiology, course, and outcome of eating disorders. *Curr Opin Psychiatry*. 2013;26(6):543-548.
14. Kamal A, Pimentel M. Influence of dietary restriction on irritable bowel syndrome. *Am J Gastroenterol*. 2019;114(2):212-220.
15. American Psychiatric Association. *American Psychiatric Association. DSM-5 Task Force. Diagnostic and statistical manual of mental disorders: DSM-5*. 5th ed. Washington, DC: American Psychiatric Association; 2013. xliv, 947.
16. Sato Y, Fukudo S. Gastrointestinal symptoms and disorders in patients with eating disorders. *Clin J Gastroenterol*. 2015;8(5):255-263.
17. Cognitive-behavioral therapy for avoidant/restrictive food intake disorder. In: Thomas JJ, Eddy KT, eds. *Cognitive-Behavioral Therapy for Avoidant/Restrictive Food Intake Disorder: Children, Adolescents, and Adults*. Cambridge, United Kingdom: Cambridge University Press; 2018. i-i.
18. Cena H, Barthels F, Cuzzolaro M, Bratman S, Brytek-Matera A, Dunn T, et al. Definition and diagnostic criteria for orthorexia nervosa: a narrative review of the literature. *Eat Weight Disord*. 2019;24(2):209-246.
19. Chey WD. Elimination diets for irritable bowel syndrome: approaching the end of the beginning. *Am J Gastroenterol*. 2019;114(2):201-203.
20. Harer KN, Jagielski C, Chey WD, Riehl M. Avoidant/restrictive food intake disorder (ARFID) among adult gastroenterology behavioral health patients: comparison of ARFID vs. non-ARFID patients. *J Am Coll Gastroenterol*. 2019;114(Suppl):S271-S272.
21. Koloski NA, Jones M, Talley NJ. Evidence that independent gut-to-brain and brain-to-gut pathways operate in the irritable bowel syndrome and functional dyspepsia: a 1-year population-based prospective study. *Aliment Pharmacol Ther*. 2016;44(6):592-600.
22. Popa SL, Dumitrascu DL. Anxiety and IBS revisited: ten years later. *Clujul Med*. 2015;88(3):253-257.
23. Mikocka-Walus A, Knowles SR, Keefer L, Graff L. Controversies revisited: a systematic review of the comorbidity of depression and anxiety with inflammatory bowel diseases. *Inflamm Bowel Dis*. 2016;22(3):752-762.
24. Boyd C, Abraham S, Kellow J. Psychological features are important predictors of functional gastrointestinal disorders in patients with eating disorders. *Scand J Gastroenterol*. 2005;40(8):929-935.
25. Pace LA, Crowe SE. Complex relationships between food, diet, and the microbiome. *Gastroenterol Clin North Am*. 2016;45(2):253-265.
26. Mandelli L, Arminio A, Atti AR, De Ronchi D. Suicide attempts in eating disorder subtypes: a meta-analysis of the literature employing DSM-IV, DSM-5, or ICD-10 diagnostic criteria. *Psychol Med*. 2019;49(8):1237-1249.
27. Norris ML, Harrison ME, Isserlin L, Robinson A, Feder S, Sampson M. Gastrointestinal complications associated with anorexia nervosa: a systematic review. *Int J Eat Disord*. 2016;49(3):216-237.

28. Salvioli B, Pellicciari A, Iero L, Di Pietro E, Moscano F, Gualandi S, et al. Audit of digestive complaints and psychopathological traits in patients with eating disorders: a prospective study. *Dig Liver Dis*. 2013;45(8):639-644.

29. Abraham S, Kellow JE. Do the digestive tract symptoms in eating disorder patients represent functional gastrointestinal disorders? *BMC Gastroenterol*. 2013;13:38.

30. Abraham S, Luscombe GM, Kellow JE. Pelvic floor dysfunction predicts abdominal bloating and distension in eating disorder patients. *Scand J Gastroenterol*. 2012;47(6):625-631.

31. Benini L, Todesco T, Dalle Grave R, Deiorio F, Salandini L, Vantini I. Gastric emptying in patients with restricting and binge/purging subtypes of anorexia nervosa. *Am J Gastroenterol*. 2004;99(8):1448-1454.

32. Rigaud D, Bedig G, Merrouche M, Vulpillat M, Bonfils S, Apfelbaum M. Delayed gastric emptying in anorexia nervosa is improved by completion of a renutrition program. *Dig Dis Sci*. 1988;33(8):919-925.

33. Hutson WR, Wald A. Gastric emptying in patients with bulimia nervosa and anorexia nervosa. *Am J Gastroenterol*. 1990;85(1):41-46.

34. Nickl NJ, Brazer SR, Rockwell K, Smith JW. Patterns of esophageal motility in patients with stable bulimia. *Am J Gastroenterol*. 1996;91(12):2544-2547.

35. Abell TL, Malagelada JR, Lucas AR, et al. Gastric electromechanical and neurohormonal function in anorexia nervosa. *Gastroenterology*. 1987;93(5):958-965.

36. Bern EM, Woods ER, Rodriguez L. Gastrointestinal manifestations of eating disorders. *J Pediatr Gastroenterol Nutr*. 2016;63(5):e77-e85.

37. Satherley R, Howard R, Higgs S. Disordered eating practices in gastrointestinal disorders. *Appetite*. 2015;84:240-250.

38. Reed-Knight B, Squires M, Chitkara DK, van Tilburg MA. Adolescents with irritable bowel syndrome report increased eating-associated symptoms, changes in dietary composition, and altered eating behaviors: a pilot comparison study to healthy adolescents. *Neurogastroenterol Motil*. 2016;28(12):1915-1920.

39. Satherley RM, Howard R, Higgs S. Development and validation of the coeliac disease food attitudes and behaviours scale. *Gastroenterol Res Pract*. 2018;2018:6930269.

40. Scarlata K, Catsos P, Smith J. From a dietitian's perspective, diets for irritable bowel syndrome are not one size fits all. *Clin Gastroenterol Hepatol*. 2020;18(3):543-545.

41. Zia JKRM, DeCou CR, McCann BS, Heitkemper M. Prevalence of eating disorders, especially DSM-5's avoidant restrictive food intake disorder, in patients with functional gastrointestinal disorders: a cross-sectional online survey. *Gastroenterology*. 2017;152(Suppl):S715-S716.

42. Harer K, Baker J, Reister N, Collins K, Watts L, Phillips C, et al. Avoidant/restrictive food intake disorder in the adult gastroenterology population: an under-recognized diagnosis? *J Am Coll Gastroenterol*. 2018;113(Suppl):S247-S248.

43. Mari A, Hosadurg D, Martin L, Zarate-Lopez N, Passananti V, Emmanuel A. Adherence with a low-FODMAP diet in irritable bowel syndrome: are eating disorders the missing link? *Eur J Gastroenterol Hepatol*. 2019;31(2):178-182.

44. Murray HB, Juarascio AS, Di Lorenzo C, Drossman DA, Thomas JJ. Diagnosis and treatment of rumination syndrome: a critical review. *Am J Gastroenterol*. 2019;114(4):562-578.

45. Halland M, Pandolfino J, Barba E. Diagnosis and treatment of rumination syndrome. *Clin Gastroenterol Hepatol*. 2018;16(10):1549-1555.

46. Absah I, Rishi A, Talley NJ, Katzka D, Halland M. Rumination syndrome: pathophysiology, diagnosis, and treatment. *Neurogastroenterol Motil*. 2017;29(4).

47. Riehl ME, Kinsinger S, Kahrilas PJ, Pandolfino JE, Keefer L. Role of a health psychologist in the management of functional esophageal complaints. *Dis Esophagus*. 2015;28(5):428-436.

48. Burton Murray H, Jehangir A, Silvernale CJ, Kuo B, Parkman HP. Avoidant/restrictive food intake disorder symptoms are frequent in patients presenting for symptoms of gastroparesis. *Neurogastroenterol Motil*; 2020. 2020;e13931.

49. Ilzarbe L, Fabrega M, Quintero R, Bastidas A, Pintor L, Garcia-Campayo J, et al. Inflammatory bowel disease and eating disorders: A systematized review of comorbidity. *J Psychosom Res*. 2017;102:47-53.

50. Mascolo M, Geer B, Feuerstein J, Mehler PS. Gastrointestinal comorbidities which complicate the treatment of anorexia nervosa. *Eat Disord*. 2017;25(2):122-133.

51. Levine A, Sigall Boneh R, Wine E. Evolving role of diet in the pathogenesis and treatment of inflammatory bowel diseases. *Gut*. 2018;67(9):1726-1738.

52. Gibson PR. Use of the low-FODMAP diet in inflammatory bowel disease. *J Gastroenterol Hepatol*. 2017;32(Suppl 1):40-42.

53. Yamamoto T, Nakahigashi M, Saniabadi AR. Review article: diet and inflammatory bowel disease–epidemiology and treatment. *Aliment Pharmacol Ther*. 2009;30(2):99-112.

54. Quick VM, McWilliams R, Byrd-Bredbenner C. Case-control study of disturbed eating behaviors and related psychographic characteristics in young adults with and without diet-related chronic health conditions. *Eat Behav*. 2012;13(3):207-213.

55. Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. *BMJ*. 1999;319(7223):1467-1468.

56. Riedlinger C, Schmidt G, Weiland A, Stengel A, Giel KE, Zipfel S, et al. Which symptoms, complaints and complications of the gastrointestinal tract occur in patients with eating disorders? A systematic review and quantitative analysis. *Front Psychiatry*. 2020;11:195.

57. Quick VM, Byrd-Bredbenner C, Neumark-Sztainer D. Chronic illness and disordered eating: a discussion of the literature. *Adv Nutr*. 2013;4(3):277-286.

58. Hughes LD, King L, Morgan M, Ayis S, Direkze N, Lomer MC, et al. Food-related Quality of Life in Inflammatory Bowel Disease: development and validation of a questionnaire. *J Crohns Colitis*. 2016;10(2):194-201.

59. Bryant-Waugh R, Micali N, Cooke L, Lawson EA, Eddy KT, Thomas JJ. Development of the Pica, ARFID, and Rumination Disorder Interview, a multi-informant, semi-structured interview of feeding disorders across the lifespan: a pilot study for ages 10-22. *Int J Eat Disord*. 2019;52(4):378-387.

60. Keefer L, Palsson OS, Pandolfino JE. Best practice update: incorporating psychogastroenterology into management of digestive disorders. *Gastroenterology*. 2018;154(5):1249-1257.

61. Riehl ME, Kinnucan JA, Chey WD, Stidham RW. Nuances of the psychogastroenterology patient: a predictive model for gastrointestinal quality of life improvement. *Neurogastroenterol Motil*. 2019;31(9). 2019;e13663.

62. Peckmezian T, Paxton SJ. A systematic review of outcomes following residential treatment for eating disorders. *Eur Eat Disord Rev*. 2020;28(3):246-259.

63. Twohig MP, Bluett EJ, Torgesen JG, Lensegrav-Benson T, Quakenbush-Roberts B. Who seeks residential treatment? A report of patient characteristics, pathology, and functioning in females at a residential treatment facility. *Eat Disord*. 2015;23(1):1-14.

64. Anzai N, Lindsey-Dudley K, Bidwell RJ. Inpatient and partial hospital treatment for adolescent eating disorders. *Child Adolesc Psychiatr Clin N Am*. 2002;11(2):279-309.

65. Linardon J, Wade TD, de la Piedad Garcia X, Brennan L. The efficacy of cognitive-behavioral therapy for eating disorders: a systematic review and meta-analysis. *J Consult Clin Psychol*. 2017;85(11):1080-1094.

66. Hill P, Muir JG, Gibson PR. Controversies and recent developments of the low-FODMAP diet. *Gastroenterol Hepatol (N Y)*. 2017;13(1):36-45.

67. Peters SL, Yao CK, Philpott H, Yelland GW, Muir JG, Gibson PR. Randomised clinical trial: the efficacy of gut-directed hypnotherapy is similar to that of the low FODMAP diet for the treatment of irritable bowel syndrome. *Aliment Pharmacol Ther*. 2016;44(5):447-459.

68. Chey WD, Keefer L, Whelan K, Gibson PR. Behavioral and diet therapies in integrated care for patients with irritable bowel syndrome. *Gastroenterology*. 2021;160(1):47-62.

69. van Tilburg MAL. Psychogastroenterology: a cure, Band-Aid, or prevention? Children (Basel). 2020;7(9):121.

70. Basnayake C, Kamm MA, Stanley A, Wilson-O'Brien A, Burrell K, Lees-Trinca I, et al. Standard gastroenterologist versus

multidisciplinary treatment for functional gastrointestinal disorders (MANTRA): an open-label, single-centre, randomised controlled trial. *Lancet Gastroenterol Hepatol*. 2020;5(10):890-899.

71. Alberga AS, Edache IY, Forhan M, Russell-Mayhew S. Weight bias and health care utilization: a scoping review. *Prim Health Care Res Dev*. 2019;20:e116.

72. Ciao AC, Loth K, Neumark-Sztainer D. Preventing eating disorder pathology: common and unique features of successful eating disorders prevention programs. *Curr Psychiatry Rep*. 2014;16(7):453.

73. Langmesser L, Verscheure S. Are eating disorder prevention programs effective. *J Athl Train*. 2009;44(3):304-305.

74. Mack I, Cuntz U, Gramer C, Niedermaier S, Pohl C, Schwiertz A, et al. Weight gain in anorexia nervosa does not ameliorate the faecal microbiota, branched chain fatty acid profiles, and gastrointestinal complaints. *Sci Rep*. 2016;6:26752.

75. Mack I, Penders J, Cook J, Dugmore J, Mazurak N, Enck P. Is the impact of starvation on the gut microbiota specific or unspecific to anorexia nervosa? A narrative review based on a systematic literature search. *Curr Neuropharmacol*. 2018;16(8):1131-1149.

76. Shin A, Preidis GA, Shulman R, Kashyap PC. The gut microbiome in adult and pediatric functional gastrointestinal disorders. *Clin Gastroenterol Hepatol*. 2019;17(2):256-274.

AUTHOR INFORMATION

Address correspondence to: Megan E. Riehl, PsyD, MA, University of Michigan, 3912 Taubman Center, SPC 5362, Suite 34361500 E. Medical Center Drive, Ann Arbor, MI 48109-5362. E-mail: mriehl@med.umich.edu

STATEMENT OF POTENTIAL CONFLICT OF INTEREST

M. Riehl has received advisory fees from Gastro Girl, Inc., a co-parent owner of GI OnDEMAND and consultant fees from Health Union, LLC. K. Scarlta has published low-FODMAP diet books and educational materials for FODY Food Co; is a paid advisor and has equity in Epicured LLC; has equity and received advisory fees from Gastro Girl, Inc., a co-parent owner of GI OnDEMAND, and has received payment for developing educational content for A2 Milk Company, Green Valley Creamery, and Enjoy Life Foods.

FUNDING/SUPPORT

There is no funding to disclose.

AUTHOR CONTRIBUTIONS

M. Riehl was responsible for drafting, editing, and finalizing the manuscript. K. Scarlta was responsible for drafting, editing, and finalizing the manuscript.