Common and Emergent Oral and Gastrointestinal Manifestations of Eating Disorders

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Abstract: Eating disorders (EDs) such as anorexia nervosa, bulimia nervosa, and avoidant/restrictive food intake disorder are associated with restricted diets and abnormal compensatory behaviors, frequently leading to malnutrition and oral and gastrointestinal manifestations. Dental and oral complications are generally caused by malnutrition, micronutrient deficiency, and chronic acid exposure; hence, treatment of the ED and frequent dental examinations are essential to reduce morbidity. Gastrointestinal manifestations are multifactorial in origin, and may be caused by disordered behaviors, malnutrition, anxiety, and/or may be a function of the ED itself. This article reviews the most common oral and gastrointestinal manifestations of EDs and describes emergent complications such as acute gastric dilation and superior mesenteric artery syndrome. It is important for providers to recognize complications associated with EDs to provide the best treatment possible.

Keywords
Gastrointestinal complications, oral complications, eating disorders, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa

Eating disorders (EDs) are psychiatric disorders characterized by a persistent disturbance in eating behaviors. Medical complications commonly arise from these mental health disorders ranging in presentation from physical discomfort requiring supportive care to life-threatening injuries requiring emergent surgical intervention. Impairment of gastrointestinal (GI) function and structure is a common feature of EDs and usually is the result of physical binging or purging activities and potential malnutrition and micronutrient deficiencies.[1,2] In 2013, the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*)[3] was published. The *DSM-5* allowed for more distinct ED diagnoses to be made.[4] This edition clarified the diagnosis of anorexia nervosa (AN), providing more inclusive criteria to recognize the impact of EDs on males, on prepubertal females, and across

Table 1. Summary of the Diagnostic Criteria, Development, and Prevalence of Feeding and Eating Disorders[3]

Diagnosis	Diagnostic Criteria	Development and Prevalence
Pica	• Persistent eating of nonfood substances for ≥1 month • The behavior is inappropriate for the individual's developmental level • The behavior is not part of a cultural or social practice • Can be associated with other mental disorders	• Onset can occur at any age, although childhood onset is most common • Prevalence is unclear; prevalence increases with severity of intellectual disability
Rumination	• Repeated regurgitation of food for ≥1 month • Not attributable to another medical condition • Not occurring exclusively during the course of another eating disorder • Can be associated with other mental disorders	• Onset can occur at any age; can be episodic or continuous • Prevalence is unclear; more prevalent in individuals with intellectual disability
Avoidant/restrictive food intake disorder	• Eating/feeding disturbance such as lack of interest, fear of adverse consequences of eating (eg, choking), and avoidance of foods with certain sensory characteristics leading to: – Significant weight loss or failure to gain weight – Significant nutritional deficiency – Dependence on oral or enteral supplements – Poor psychosocial functioning • Not better explained by lack of food or a cultural practice • Not occurring exclusively during the course of another eating disorder • Not due to another medical or psychological disorder	• Onset is most common in infancy or early childhood; may persist into adulthood • Prevalence is unclear
Anorexia nervosa Subtypes: – Restricting – Binge eating/ purging	• Restriction of energy intake leading to significantly low body weight for developmental trajectory • Persistent behaviors to avoid weight gain and intense fear of gaining weight or becoming fat • Disturbed body image perception – Restricting subtype is not associated with purging – Binge eating/purging subtype may or may not involve binge eating (eating large amounts of food in a discrete period of time with a sense of loss of control); purging occurs through self-induced vomiting or misuse of laxatives, diuretics, enemas, or excessive exercise	• Onset is typically in adolescence or young adulthood; often associated with stressful life event • 12-month prevalence among young females is ~0.4%. Less common in males than in females
Bulimia nervosa	• Recurrent episodes of binge eating • Recurrent inappropriate compensatory measures such as vomiting, laxatives, fasting, or excessive exercise to prevent weight gain • Binge-purge cycles occur on average at least 1×/week for 3 months • Self-evaluation is influenced by body shape and weight • The behavior is not exclusively during episodes of bulimia nervosa	• Onset is typically in adolescence or young adulthood; frequently preceded by an episode of dieting • 12-month prevalence among young females is 1%-1.5%. Peaks in older adolescence and young adulthood. Less common in males than in females
Binge eating disorder	• Recurrent episodes of binge eating • Binges associated with ≥3 of the following: – Eating more rapidly than usual – Eating until uncomfortably full – Eating large amounts when not hungry – Eating alone because embarrassed – Feeling disgusted, depressed, or guilty afterwards • Marked distress regarding binges • Recurrent episodes at least 1×/week for 3 months • Not associated with inappropriate compensatory behaviors as in bulimia nervosa or not exclusively during the course of bulimia nervosa or anorexia nervosa	• Common in adolescents and young adults, but onset and development are unknown; dieting frequently occurs after development of binge eating disorder • 12-month prevalence among adult females is 1.6% and among adult males is 0.8%
Other specified feeding or eating disorder	• Atypical anorexia nervosa: all criteria for anorexia nervosa met except lowest weight is within or above normal range • Bulimia nervosa of low frequency or limited duration • Binge eating disorder of low frequency or limited duration • Purging disorder: recurrent purging without binge eating • Night eating syndrome: recurrent episodes of night eating causing significant distress or functional impairment	
Unspecified feeding or eating disorder	• Symptoms cause significant distress or functional impairment but do not meet the full criteria of any of the aforementioned disorders	

Adapted from American Psychiatric Association.[3]

the weight spectrum. Binge eating disorder (BED) was also added as a formal diagnosis, pica and rumination disorders were clarified, and the new avoidant/restrictive food intake disorder (ARFID) was described (see Table 1 for a summary of ED diagnoses). Poorly categorized EDs were placed under the terms of *other specified feeding or eating disorder* and *unspecified feeding or eating disorder*, but the changes allowed for fewer patients to need such undefined diagnoses.[3] Having specific diagnostic criteria allows patients and providers to understand the potential impact of an ED on the body.

To describe the oral and GI complications associated with EDs, we elected to focus specifically on AN, bulimia nervosa (BN), and ARFID because of their significant morbidity as a result of malnutrition, micronutrient deficiency, and binging or purging. AN is defined by an intense fear of gaining weight, body dysmorphia, and poor nutritional intake, leading to significantly low weight. In the *DSM-5*, amenorrhea and a body mass index (BMI) cutoff were removed as diagnostic criteria.[3] BN manifests as recurrent episodes of binge eating and inappropriate compensatory measures such as self-induced vomiting, laxative use, or diuretic use.[3] Patients with AN also may use the same compensatory measures to control their weight, in addition to limited caloric intake in the binge eating or purging subtype. ARFID often presents in early childhood and is characterized by avoidance of foods due to lack of interest in them, displeasure of certain sensory characteristics, or fear of an adverse event when eating.[3] Many patients with ARFID report GI complaints such as reflux, abdominal pain, nausea, and constipation, and cite these as reasons for continued food avoidance.[5,6] Avoidant or restrictive feeding behaviors can lead to malnutrition, dependence on supplemental nutrition, and/or poor psychosocial functioning.[3] The purpose of this article is to review the oral and gastrointestinal manifestations of EDs (specifically AN, BN, and ARFID), so providers can detect concerning signs for an undiagnosed ED when patients present with GI symptoms, identify common findings and urgent complications associated with EDs (Table 2), and recognize when further workup and treatment are necessary.

Oral Manifestations

The oral manifestations of EDs include damage to the teeth, oral mucosa, and salivary glands. These changes are largely due to chronic acid exposure from self-induced vomiting, unhealthy eating and drinking habits, and oral hygiene practices.[7,8] Nutritional deficiencies such as low vitamin B and C also can cause oral injury.[9] There are many changes in the oral cavity, and it is crucial to recognize them during ED diagnosis and monitoring.

Dental Complications

Dental erosion is a significant health problem in patients with EDs, especially AN and BN. A systematic review found that, overall, the odds of dental erosion were 5 times higher in patients with EDs compared with patients without EDs.[10] Although the odds were greatest for patients who reported self-induced vomiting, dental erosion was still a significant complication for patients who denied vomiting. Erosion is most commonly seen on the palatal side of the anterior maxillary teeth, as this side is exposed most directly to acid.[11] The erosion seen in chronic acid exposure results in uniform, polished dental surfaces, unlike changes from mechanical wear.[12,13] In addition to dental erosion, patients with EDs are at higher risk for gingivitis,[8,14] dental caries and tooth decay,[8,10] and tooth hypersensitivity.[8]

Many behaviors can cause dental complications. Self-induced vomiting leads to chronic, repeated acid exposure to the teeth.[15] Patients who report any self-induced vomiting[10,16] are at higher risk of dental changes, and this risk increases with higher frequency of vomiting.[8,17] Patients with EDs also have different toothbrushing habits than the healthy population, such as increased daily brushing and brushing just after vomiting, both of which may promote dental erosion and damage.[7] These patients also have different diets than healthy controls.[11,13,18,19] Patients with AN and BN often have higher consumption of acidic, carbonated, or caffeinated beverages, sweetened foods, and chewing gum, possibly to support excessive exercise, improve energy, and/or suppress appetite.[11,13,18] Patients with ARFID also have diets higher in refined carbohydrates and added sugars when compared with healthy controls, which is a risk factor for dental damage and caries.[19] Providers should look for signs of dental erosion or decay when suspecting an ED in a patient or routinely monitoring a patient with an ED, and refer these patients for regular dental examinations.

Other Oral Complications

There are many immediate oral examination findings to be aware of when monitoring ED behaviors. Various case-control studies and case series have shown that patients with EDs are more likely than patients without EDs to have dry lips[17] and inflammation at the corners of the mouth, known as angular cheilitis.[8,17,20] Angular cheilitis is commonly seen in patients who practice purging behaviors,[8] and could also be a sign of common micronutrient deficiencies found in patients with EDs, such as the B vitamins, iron, folic acid, and zinc.[9,21,22]

Parotid gland enlargement is also a well-known feature of BN and can be the presenting sign of a purging disorder.[23,24] The etiology is theorized to be changes to the

Table 2. Outpatient Vs Urgent/Emergent Management of Oral and Gastrointestinal Manifestations of Eating Disorders[2]

System	Appropriate for Outpatient Management	Requires Urgent/Emergent Management
Dental	• Dental changes (eg, caries, erosion, decay) • Gingivitis	
Oral	• Angular cheilitis • Parotid or salivary gland hypertrophy • Mucosal changes (eg, erythema, edema, petechiae, mild bleeding)	• Necrotizing sialometaplasia • Hemorrhagic gingivitis
Esophagus	• Gastroesophageal reflux • Barrett esophagus • Esophageal dysmotility • Mallory-Weiss tears • Disorders of gut-brain interaction	• Esophageal carcinomas • Unintentional foreign body ingestion • Esophageal rupture (Boerhaave syndrome)
Stomach	• Gastroparesis • Disorders of gut-brain interaction	• Acute gastric dilation
Small and large intestines	• Constipation • Diarrhea • Fecal incontinence • Irritable bowel syndrome • Disorders of gut-brain interaction	• Superior mesenteric artery syndrome • Necrotizing colitis
Pancreas	• Pancreatic atrophy	• Acute pancreatitis
Liver	• Mildly elevated transaminases	• Markedly elevated transaminases • Liver failure

Adapted from Bern et al.[2]

parasympathetic innervation of the glands and/or chronic stimulation from vomiting.[23] Although parotid swelling can be seen within days after a binge-purge episode and can be a persistent finding in chronic purging,[25] it is an infrequent sign in general. Even on magnetic resonance imaging, parotid hypertrophy is only seen in a minority of patients with BN.[26]

In addition to parotid gland enlargement, there can be other salivary gland changes in patients with EDs, including hyposalivation and dry mouth, which can exacerbate or cause dental disease.[8,10,13,17] The most alarming complication is necrotizing sialometaplasia. Necrotizing sialometaplasia is an oral ulcer caused by local trauma, leading to mucosal swelling[27] and ischemia.[28] In many case studies, manipulation of fingers to bring about purging[27,29] can induce trauma to the hard palate leading to necrotizing sialometaplasia.[30] Necrotizing sialometaplasia is a benign, self-limiting, necrotizing process of the minor salivary glands, usually on the hard palate.[28] However, its appearance is commonly mistaken for malignancy,[29] and should still be evaluated if found.

Oral pathologic changes are commonly seen in the setting of chronic micronutrient deficiencies. Patients with ARFID are at particularly high risk given the long-standing nature of the restrictive diet, often limited to highly processed foods and carbohydrates, which are not fortified with minerals and vitamins.[19,31] Oral candidiasis is seen frequently in the elderly population experiencing undernutrition,[32] but is inconsistently found in smaller studies of patients with EDs.[8,20] Mucosal erythema and edema, angular cheilitis, gingival erythema, and dental caries can present in the setting of chronic vitamin B deficiencies.[9] Similarly, vitamin C deficiency—essential in collagen synthesis and wound healing—can present with mucosal petechiae, gingival bleeding, and hemorrhagic gingivitis.[9] Lastly, atrophic glossitis can be caused by significant deficiencies in many vital nutrients, such as the B vitamins, vitamin E, folic acid, iron, and zinc.[33]

However, dental and oral manifestations of EDs may be missed if patients are not seen regularly for dental visits. One study found that 75% of patients with purging behaviors knew of the risk of dental erosion, but just over half saw a dentist regularly, and only 15% were referred to a dentist after their ED diagnosis.[34] Also, most adolescents (83.3%) with a high severity of BED and/or BN risk behaviors did not undergo routine dental examinations.[35]

Earlier diagnosis of EDs is associated with lower dental caries and fewer oral lesions,[20] and given the many dental and oral findings associated with EDs, it is imperative to regularly refer patients with EDs for dental visits.

Gastrointestinal Manifestations

There are many GI manifestations of EDs. These manifestations can occur throughout the GI tract and in the liver and pancreas. Most studies assessing the effects of EDs on the GI system were conducted on patients with severe malnutrition from AN, or with vomiting and laxative abuse, which can be seen in AN as well as in BN. Assessing patients for the origins of their concerns can help providers identify and rectify a problem. However, the pathophysiology of GI manifestations is not always clear. Presenting symptoms are sometimes a function of the ED, a result of malnutrition or other ED behaviors, and prolonged or worsened by continued food avoidance in an attempt to avoid GI discomfort. This section focuses on common GI findings associated with EDs, as well as the emergent and dangerous complications to be aware of.

Esophagus

Dysphagia, regurgitation, heartburn, and emesis can be common complaints in patients with EDs. Distinguishing whether the etiology is a result of underlying GI pathology, the ED itself, or a disorder of the gut-brain interaction can be challenging. Underlying GI disorders can include achalasia, esophageal inflammation, stricture, motility disorders, and gastroesophageal reflux disease. However, patients with AN, BN, and BED can similarly present with dyspepsia and vomiting. Furthermore, repeated, self-induced vomiting may in itself injure the esophageal mucosa, increasing the risk of esophageal inflammation and even dysplasia.[36] Understanding the spectrum of presentation of esophageal disorders can enable providers to better delineate when diagnostic intervention is indicated.

The symptoms of gastroesophageal reflux disease in patients with EDs are regularly attributed to repeated, self-induced vomiting, yet the relationship is still unclear.[36] Acid exposure is not limited to patients who purge, and binge eating itself, a common behavior associated with various EDs, may be an independent risk factor for gastroesophageal reflux disease.[37] Repeated acid exposure can be associated with the development of Barrett esophagus, whereby the esophageal squamous epithelium is replaced by GI columnar epithelium, which is more susceptible to malignancy.[38] Barrett esophagus is associated with a 30-fold increased risk of developing esophageal carcinoma over the general population.[39] It has been hypothesized that self-induced vomiting may be associated with the development of Barrett esophagus, but a lack of data to date prevents drawing a definitive conclusion.[40,41] There are a few case reports of patients with BN presenting with worsening epigastric pain and reflux, and ultimately being diagnosed with esophageal adenocarcinoma.[41,42] Patients with EDs also may be at higher risk of esophageal squamous cell carcinoma compared with the general population.[43] A large study of over 3600 patients who had previously been hospitalized for an ED found a 6-fold higher risk of developing esophageal squamous cell carcinoma. Although all of the patients with cancer had a history of AN, potential confounding factors included high rates of tobacco and alcohol use and chronic nutritional deficiency.[43] Providers should consider endoscopic evaluation of patients with an ED who have persistent symptoms of dyspepsia or vomiting given the potential risk of esophageal malignancy.

The literature supports the presence of normal esophageal motility in the majority of patients with EDs.[44,45] Nickl and colleagues found normal esophageal motility in a group of young adults with stable BN and AN who reported purging, even in those who complained of dysphagia and/or odynophagia.[44] Benini and colleagues recruited 24 female patients admitted to an inpatient ED facility for AN and found that, despite most patients complaining of regurgitation, heartburn, and dysphagia, all but 1 patient (with a nutcracker esophagus) had normal findings on esophageal manometry.[45] In addition, there was no correlation between motility and severity of symptoms. Furthermore, symptoms still persisted despite nutritional rehabilitation, suggesting that there may not always be an identifiable organic cause for these esophageal symptoms, which may be a function of the disease itself.[45]

Physical trauma to the esophagus may occur due to repeated and forceful vomiting. Hematemesis can be seen in patients who purge, usually caused by Mallory-Weiss tears in the esophagus.[25,46] However, patients sometimes use tools to incite the gag reflex, and cases of accidental ingestion have been documented. Patients have presented with ingestion of spoons[47] or forks[48] requiring endoscopic removal. Such ingestion requires urgent intervention, which should be considered when patients with an ED present with sudden-onset dysphagia or respiratory distress. Lastly, there are a few documented cases of esophageal rupture (Boerhaave syndrome) in patients with EDs and purging.[49,50] Patients may present with vomiting, chest pain, shortness of breath, and subcutaneous emphysema.[49,51] Although rare, Boerhaave syndrome is associated with a high mortality rate and must be diagnosed quickly for rapid surgical intervention. Because the clinical symptoms are not always clear, critical diagnostic tools include a chest radiograph showing pneumomediastinum and a contrast esophagogram showing the perforation.[51]

Stomach

Patients with EDs often present with complaints including nausea, abdominal pain, and early satiety.[52,53] These symptoms may be a physical result of malnutrition and/or a psychological function of the ED.[1,54] Gastroparesis is a syndrome of delayed gastric emptying that includes symptoms of nausea and vomiting, bloating, early satiety, and abdominal pain, and is commonly seen in the setting of malnutrition and EDs.[52,55] A systematic review by Norris and colleagues found a multitude of case studies describing significantly delayed gastric emptying in patients with AN; gastroparesis was more prevalent with longer duration and severity of malnutrition.[1] Many studies indicate that gastroparesis is likely a result of malnutrition and can improve with nutritional rehabilitation.[1,56]

The complaints of early satiety, nausea, and abdominal pain may, however, be a disorder of gut-brain interaction. Symptoms consistent with gastroparesis are not always associated with measurable physiologic changes,[52] and can persist despite improved gastric emptying times.[55] Although these symptoms can resolve with nutritional rehabilitation, patients with EDs who have anxiety and depression are more likely to struggle with persistent GI symptoms despite weight improvement when compared with patients with EDs without anxiety and depression.[54] In addition, patients with ARFID frequently report abdominal pain, nausea, vomiting, and anxiety.[57,58] Although these symptoms can mimic gastroparesis, significant delays in gastric motility are inconsistently found.[59] Murray and colleagues surveyed 288 adult patients who were referred to academic centers for symptoms of gastroparesis.[59] They discovered that 39.9% of patients met the cutoff criteria for clinically significant ARFID symptoms. Although symptom severity was positively associated with severity of ARFID symptoms, there was no association between patient-reported symptoms of gastroparesis and measured gastric emptying times.[59]

Ultimately, both the mechanism of and treatment for gastroparesis in patients with EDs are still poorly understood and likely multifactorial. Malnutrition, vomiting, laxative abuse, and a central stress response may lead to autonomic dysfunction, changes to the gastric smooth muscle, and other metabolic and hormonal effects on gastric motility.[60-63] Treatment for the complaints associated with gastroparesis requires a multidisciplinary approach. Nutritional rehabilitation will often lead to symptom improvement, and addressing weight restoration can be both a diagnostic and therapeutic tool. Moreover, helping patients recognize the prevalent association of disorders of gut-brain interaction[64] allows providers to stress the importance of mental health therapy in conjunction with dietary intake.

Lastly, acute gastric dilation can be seen in both AN and BN following a binging episode, or as a result of an obstruction from superior mesenteric artery (SMA) syndrome. This complication can have serious consequences such as ischemia, necrosis, and gastric perforation.[65-67] Cases have been reported in which patients with binge or purge behaviors presented with severe, acute-onset abdominal pain and markedly dilated stomachs after a large binge, requiring emergent intervention for decompression.[65,66] One patient even experienced gastric perforation from the acute dilation.[67] Presentation of this complication ranged from 1 hour to the next morning after a binge.[65-67] Although rare, acute gastric dilation is a medical emergency that cannot be managed as an outpatient.

Small and Large Intestines

Patients with EDs frequently report general GI discomfort, and some present with irritable bowel syndrome or disorders of defecation, constipation, or fecal incontinence.[52,68] There are also some uncommon, but urgent, complications such as SMA syndrome[69-72] and necrotizing colitis.[73] Furthermore, newer data suggest that intestinal dysbiosis can be associated with AN and may be an important area to explore.[74,75]

Symptoms of irritable bowel syndrome are very common in patients who have EDs.[76-78] Boyd and colleagues surveyed 109 patients who were admitted to an inpatient ED care unit with AN, BN, or ED–not otherwise specified.[76] More than half of the patients had symptoms of irritable bowel syndrome, and nearly all (98%) met the criteria for a functional GI disease such as irritable bowel syndrome, bloating, constipation, dysphagia, and anorectal pain. Kessler and colleagues found that the number of irritable bowel syndrome symptoms appears to be associated with the severity of ED symptoms and was independent of BMI.[77]

Constipation is the most common GI symptom in patients with AN[52] and is also frequently reported in BN.[79] Given the relatively new diagnosis of ARFID, the prevalence of constipation among patients with ARFID is not fully known.[57] Patients with both AN and BN can exhibit prolonged gut transit times, manifesting as constipation.[79-81] Malnutrition can be associated with smooth muscle atrophy and delayed gut transit, and a nutrient-balanced diet and weight restoration often normalize transit times in patients with AN.[79-81] However, the sensation of constipation may persist due to pelvic floor dysfunction caused by chronic malnutrition and muscle damage from repeated vomiting and straining.[82] Chiarioni and colleagues used anorectal manometry to demonstrate the presence of pelvic floor dyssynergia in patients with AN and the need for higher pressures to induce an urge

to defecate compared with healthy controls, even after nutritional rehabilitation.[81]

There does not appear to be a direct correlation between constipation and degree of malnutrition. Adults with AN who have normal BMIs still report constipation,[83] and those with BN, despite normal weights, have delayed gut transit times.[79] Longer chronicity of AN is significantly associated with higher rates of constipation, and BMIs of less than 18 are associated with higher rates of constipation and risk of fecal incontinence when compared with patients who have higher BMIs.[83] However, in a study of adolescents admitted for nutritional rehabilitation, patient-reported constipation was not associated with lower BMI.[84]

Disordered gut-brain interaction also impacts the relationship between constipation and an ED. Higher scores on ED behavior or pathology scales are associated with increased reports of defecation complaints.[85,86] Wiklund and colleagues used population-based data in Sweden to show that self-reported constipation and diarrhea at 15 years old were significantly associated with disordered eating behaviors in middle and older adolescence.[85] Murray and colleagues surveyed 279 patients with chronic constipation at a tertiary center and found that 19% of patients had clinically significant, self-reported ED pathology, and GI-specific anxiety fully mediated the relationship between ED severity and constipation severity.[86] Physically, there were no differences in anorectal manometry or colonic transit times between normal-weighted patients with or without clinically significant ED pathology, suggesting that an ED itself is not an independent predictor of constipation.[86] Patients with ED pathology with more GI-specific anxiety reported more severe constipation,[78] highlighting the significance of the interaction between EDs and disorders of gut-brain interaction.[64] Clinicians should consider screening patients with chronic constipation for ED pathology, as their dietary restrictions and disordered behaviors may further perpetuate the presence of constipation.

Constipation is likely multifactorial for patients with EDs. There are physical changes from malnutrition, delayed colonic transit, and pelvic floor dysfunction.[78-81] Anxiety[86] and irritable bowel syndrome are additional factors.[76,77] Understanding the complexity of constipation in this population can help providers better define treatment goals for patients.

Laxatives should be used carefully to treat constipation in patients with suspected EDs. Laxative abuse can be seen in many types of EDs, where patients use laxatives for relief of constipation or bloating, or abuse them for purging and weight loss.[87,88] Excessive use of laxatives can cause dehydration and critical electrolyte abnormalities.[88] Loss of potassium in the stool can cause hypokalemia, which is associated with muscle, renal, and cardiac injury.[88,89] Some laxatives contain magnesium, and excessive use can lead to hypermagnesemia, which can cause neuromuscular injury.[90] However, despite the potential misuse of laxatives, properly addressing constipation is important in ED management because chronic constipation can be associated with severe morbidity such as stercoral colitis and perforation.[91]

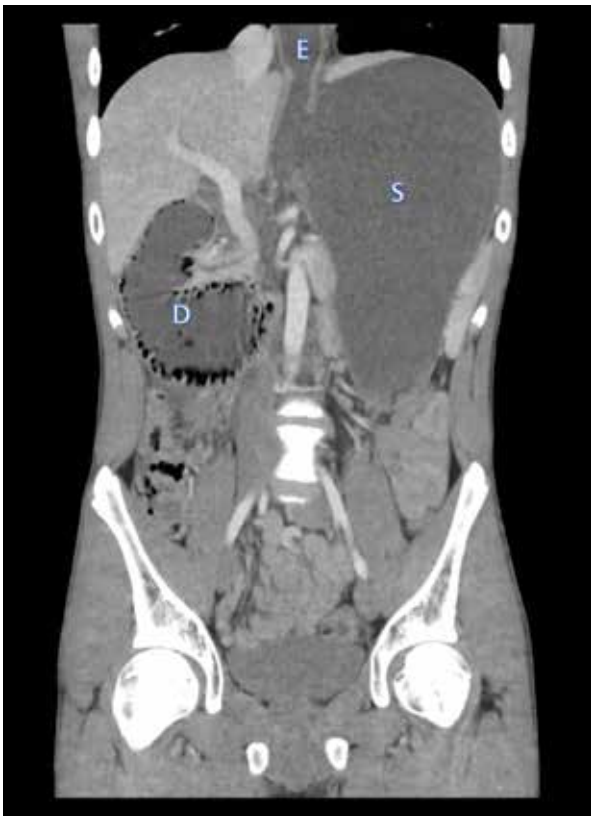

Figure 1. Coronal reformatted image from an abdominal/pelvic computed tomography scan shows the markedly dilated, fluid-filled esophagus (E), stomach (S), and duodenum (D).

SMA syndrome is a rare complication of EDs in which severe malnutrition and loss of fatty tissue leads to compression of the third portion of the duodenum between the aorta and SMA.[92,93] Patients may present with findings consistent with an intestinal obstruction, including postprandial abdominal pain, nausea, and emesis.[70] Sometimes, the symptoms can be persistent,[71] and the postprandial discomfort can lead to further food restriction.[69] Intestinal obstruction caused by SMA syndrome can be life-threatening and should be evaluated immediately, as there are case reports of patients with SMA syndrome and binge eating progressing to acute gastric dilation.[70,72] In our tertiary care hospital, a 16-year-old male patient with severe malnutrition (BMI z-score

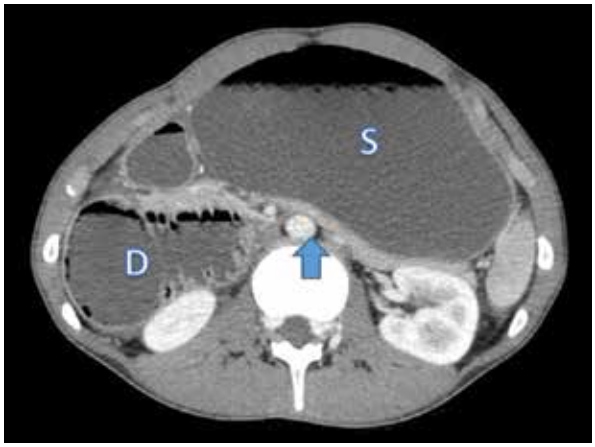

Figure 2. Axial image of a contrast-enhanced abdominal/pelvic computed tomography scan shows the markedly dilated, fluid-filled stomach (S) and the high-grade obstruction of the third portion of the duodenum (D) where it crosses the midline. The aorta is labeled with an arrow.

of -3.39) presented with marked duodenal, gastric, and esophageal dilation secondary to SMA syndrome (Figure 1). He required decompression with a nasogastric tube at a local emergency department prior to admission for nutritional rehabilitation. When SMA syndrome is suspected, it can be confirmed via plain radiographs, a barium upper GI study, computed tomography, or ultrasound.[93] Figure 2 shows a computed tomography image of the patient's intestinal obstruction caused by compression of the third portion of the duodenum between the SMA and abdominal aorta. Treatment and recovery from SMA syndrome are possible with weight restoration and gradually advancing a soft or liquid diet to a full diet.[69,93]

Necrotizing colitis is a very rare complication that may occur in patients with severe malnutrition.[73,94] An early sign may be abdominal pain, which can progress to severe pain and signs of peritonitis. Necrotizing colitis may also present as abdominal pain early in the refeeding process.[73] Potential mechanisms of this complication include starvation-induced hypoperfusion, hypoxic-ischemic injury, dysmotility, and injury to the mucosa.[73]

Recently, there has been research on the role of the intestinal microbiome in the development of an ED and as a consequence of restrictive eating behaviors. Researchers are consistently finding altered gut microbiome profiles, such as less microbial diversity, in malnourished patients with AN compared with healthy controls.[74,95,96] Researchers are also finding a similar relationship with depression in these patient groups.[74,95,96] There are notable alterations to the microbial composition as well. Borgo and colleagues found that, compared with healthy controls, malnourished patients with AN had lower caloric, fat, and carbohydrate intake.[74] Their intestinal microbiome had high concentrations of *Bacteroidetes* and depleted amounts of Firmicutes, the opposite of patients with obesity. Malnourished patients with AN also had lower concentrations of carbohydrate-fermenting genera, which can affect energy absorption from food. Lastly, the researchers found a negative correlation between the number of *Clostridium* spp and anxiety and depression.[74] These findings may be helpful in expanding providers' knowledge of the etiology and management of AN in the future.

Pancreas

Patients with EDs can develop pancreatic atrophy and pancreatitis. Overall, acute pancreatitis in this population is uncommon, and the cases described in the literature are associated with severe malnutrition.[71,97,98] Rigaud and colleagues monitored 41 adults admitted with AN who had a mean BMI of 10.1 (± standard deviation 0.57).[98] Three patients developed acute pancreatitis during refeeding, diagnosed by elevated amylase, lipase, and acute abdominal pain.[98] Importantly, amylase is commonly of salivary origin and may be elevated in patients with self-induced vomiting; measurement of serum lipase and pancreatic isoamylase levels can provide a more sensitive and specific indicator of pancreatic inflammation in patients with EDs.[1,99]

The proposed mechanisms of pancreatic injury in patients with EDs include microlithiasis, ischemia, and structural damage. The pancreas can be injured by fluid shifts during nutritional rehabilitation causing ischemic injury by retrograde flow of duodenal contents due to duodenal dysmotility, or by an unknown function of the ED itself.[100,101] Pancreatic atrophy is commonly associated with malnutrition[102,103] and is worse in patients with EDs who have lower BMIs vs higher BMIs.[104] However, pancreatic atrophy is reversible, and pancreatic size will improve with nutritional rehabilitation.[103,104]

Liver

In patients with EDs, liver injury can range from asymptomatic laboratory elevations to life-threatening liver failure and hypoglycemia.[105-107] Alanine transaminase and aspartate transaminase are commonly elevated in patients with EDs compared with healthy controls,[108] are highest in those with AN over BN or BED, and are negatively correlated with BMI.[106,107,109] Generally, patients with elevated liver enzymes do not have clinical manifestations of liver disease.[109-111] However, studies of patients admitted for medical stabilization and nutritional rehabilitation have found that those with highly elevated liver function tests were more likely to have signs of liver injury,

including high blood urea nitrogen to creatinine ratios and episodes of hypoglycemia.[110,112,113]

The liver injury seen in patients with EDs is likely a result of starvation-induced autophagy[114,115] or hepatic hypoperfusion.[116] In a malnourished state, the liver will increase autophagy to preserve nutrients and protect hepatocytes. However, when the body is chronically and severely malnourished (BMI <13), this can lead to hepatocyte cell death and liver insufficiency.[114,115] Low cardiac output also occurs in malnourished patients with EDs, leading to hepatic hypoperfusion and hypoxic injury.[116] Liver insufficiency in patients with EDs can be severe and lead to hypoglycemic coma and death.[105,114] However, with nutritional rehabilitation, liver injury and its clinical manifestations can resolve.[112,114]

Conclusion

EDs are psychiatric maladies that can lead to physical comorbidities. Patients often present with self-injurious behaviors, including restrictive intake, vomiting, and/or laxative abuse. Impairment to the oral cavity and GI system should be considered in the assessment of these individuals given the potential for serious morbidity. Table 2 summarizes the common and less common, but urgent, presentations of EDs. Given the complexity of the EDs themselves, treatment often requires a multidisciplinary approach, including medicine, nutrition, and mental health support. Medical monitoring for the acute and dangerous complications of severe malnutrition is crucial. Nutritional rehabilitation can lead to improvement and recovery of many oral and GI complications. Lastly, mental health support is crucial for managing the injurious ED behaviors themselves and for recognizing the presence of disorders of gut-brain interaction associated with many GI manifestations. Providers should focus on multidisciplinary treatment methods to manage the medical complications, ensure well-rounded nutritional intake, and support psychological distress for any patient who is struggling with an ED.

Acknowledgements

The authors would like to acknowledge the assistance of Dr Valerie L. Ward, who is an assistant professor at Harvard Medical School and is affiliated with the Department of Radiology at Boston Children's Hospital, both in Boston, Massachusetts.

Funding

This article was supported in part by the Health Resources and Services Administration (HRSA) of the US Department of Health and Human Services (HHS) as part of a MCHB T71MC00009 LEAH training grant. The contents are those of the author(s) and do not necessarily represent the official views of, nor an endorsement by, HRSA, HHS, or the US government. For more information, please visit HRSA.gov.

Disclosures

The authors have no relevant conflicts of interest to disclose.

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