Approach to Meal-Related Nausea and Vomiting

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Abstract: Nausea and vomiting are common symptoms that frequently lead to evaluation in the outpatient and inpatient settings. The pathophysiology of nausea and vomiting is complex, and the list of potential etiologies is vast. Patients with nausea and vomiting frequently report that eating exacerbates symptoms. Noteworthy gastrointestinal causes for meal-related nausea and vomiting include gastroparesis, functional dyspepsia, dumping syndrome, superior mesenteric artery syndrome, and median arcuate ligament syndrome. A number of carefully selected diagnostic tests, utilization of the Rome criteria, and an appreciation for the epidemiology of these various conditions can help the clinician hone in on the underlying cause. Importantly, a properly performed and interpreted gastric emptying study is essential to making an accurate diagnosis of gastroparesis and distinguishing this condition from functional dyspepsia, a common disorder of gut-brain interaction. There are a number of treatment options for nausea and vomiting, and the treatment approach is dependent on the specific cause for the meal-related symptoms. This article examines the approach to meal-related nausea and vomiting by reviewing tests to consider in the diagnostic evaluation of symptoms, followed by a discussion of clinically relevant disorders and disorder-specific treatments.

Keywords
Nausea, vomiting, gastroparesis, functional dyspepsia

Nausea and vomiting are common gastrointestinal symptoms that are nearly universally bothersome to the individuals affected and frequently lead to presentation to clinic, the emergency department, or hospitalization. The etiology of nausea and vomiting is complex and multifactorial, and can include both gastrointestinal and nongastrointestinal causes. In many cases, patients report that the act of eating will cause or exacerbate symptoms. Thus, the purpose of this article is to provide the clinician with a guide to approach meal-related nausea and vomiting specifically. After a review of epidemiology and pathophysiology of nausea and vomiting, common causes of meal-related symptoms will be examined and different treatments will be discussed. The goal of this article is not to provide a comprehensive review of nausea and vomiting, but rather a focused and practical guide

for the common clinical scenario whereby symptoms of nausea and vomiting are strongly associated with eating.

Epidemiology and Impact of Nausea and Vomiting

Accurately characterizing the incidence and prevalence of nausea and vomiting is difficult, as these symptoms develop because of multiple etiologies. Persistent nausea and vomiting, regardless of the cause, are common symptoms requiring medical evaluation. Analysis of data from the Nationwide Emergency Department Sample, as well as both the National Ambulatory Medical Care Survey and National Hospital Ambulatory Medical Care Survey, found that more than 5 million visits occur annually in the United States for symptoms of nausea and vomiting, making them the second most common gastrointestinal symptoms leading to an office or emergency department visit.[1] In a telephone survey of 21,128 US adults, 7% reported nausea and vomiting in the prior 3 months.[2] A population-based, cross-sectional telephone survey of 5000 Korean adults (age 20-60 years) found that nausea was present 1 day per week or more in 1.6%.[3] Nearly 1 in 6 Americans have food poisoning each year, a common cause of acute nausea and vomiting.[4]

The impact of nausea and vomiting can be evaluated by reviewing the effects on quality of life for individuals and also by assessing the economic impact to the health care system. Recurrent nausea and vomiting, from any cause, can dramatically alter home, work, and social life. For example, women with hyperemesis gravidarum are 3 to 6 times more likely to report low health-related quality of life than women with less severe symptoms.[5] Patients with celiac artery compression syndrome reported missing 22 days of school per year (adolescents) and 10 days of work per year (adults) owing to their symptoms.[6] In a study of oncology patients undergoing chemotherapy, 90% reported that nausea and vomiting imposed a significant negative impact on daily functioning.[7] Quality of life in patients with gastroparesis (GP) was significantly reduced and analogous to that of patients with chronic kidney disease and depression.[8]

The profound negative socioeconomic impact of nausea and vomiting has been measured in a number of studies. One investigation found that 8.6 million hours per year of paid employment in England and Wales are lost through pregnancy sickness symptoms, including nausea and vomiting.[9] A Swedish study found that 28% of all sick leave is related to nausea of pregnancy.[10] In the telephone survey of US adults noted previously, nausea and vomiting led to an average of 6.6 workdays, 9.0 leisure days, and 19.7 household days missed during the previous 3 months.[2] A retrospective cohort study of 19,139 patients found that the estimated mean costs of chemotherapy-induced nausea and vomiting were $5299 for the first cycle of chemotherapy alone.[11] Finally, the GP quality-of-life study found that symptoms of nausea and vomiting lowered annual income by 28.5% and resulted in disability in 11%, highlighting the negative impact of these symptoms on both patients and the health care system.[8]

Pathophysiology of Nausea and Vomiting

Nausea is a vague, unpleasant sense of unease with the feeling that vomiting might occur. Vomiting is the forceful ejection of gastric contents from the mouth. These 2 symptoms frequently coexist, although some patients vomit without preceding nausea. No single pathway or nucleus is responsible for symptoms of nausea and vomiting. Rather, multiple interrelated neural pathways, nuclei, and neurotransmitters (eg, histamine, dopamine, serotonin, norepinephrine, acetylcholine, substance P, neurokinin-1, cortisol, beta-endorphin, and vasopressin) are involved in the generation of these symptoms.[12] This complex interplay helps to explain the diverse etiology of nausea and vomiting, and also clarifies why one treatment is unlikely to improve symptoms in all individuals. Symptoms of nausea and vomiting develop owing to stimulation of the emetic center (the vomiting center). This is a collection of closely linked nuclei located in the dorsolateral reticular formation of the medulla.[13] Afferent pathways arise from the gastrointestinal tract, oropharynx, heart, musculoskeletal system, vestibular system, chemoreceptor trigger zone, and cerebral cortex. These pathways synapse on the solitary nucleus and then travel to the emetic center.[14] It has been proposed that mild stimulation of these pathways leads to nausea, whereas more intense stimulation leads to vomiting. It is likely that differences in the degree of nausea and vomiting represent individual variations in sensory thresholds, which may reflect underlying traits of anxiety, anticipation, adaptation, and resilience, although this has not been formally tested. The physical act of vomiting involves a series of carefully orchestrated events involving descending (efferent) pathways and the gastrointestinal tract, diaphragm, abdominal wall muscles, and oropharynx.[15,16] In brief, jejunal and duodenal retrograde contractions move material into the stomach, antral contractions stop and the stomach relaxes, pyloric tone increases, the lower esophageal sphincter relaxes, abdominal wall muscles and the diaphragm contract, and material is propelled upward into the mouth to be ejected. During the final step, respiration briefly ceases, the glottis and vocal cords close, and the soft palate rises, all to prevent aspiration.[14-16]

Gastrointestinal causes of chronic nausea and vom-

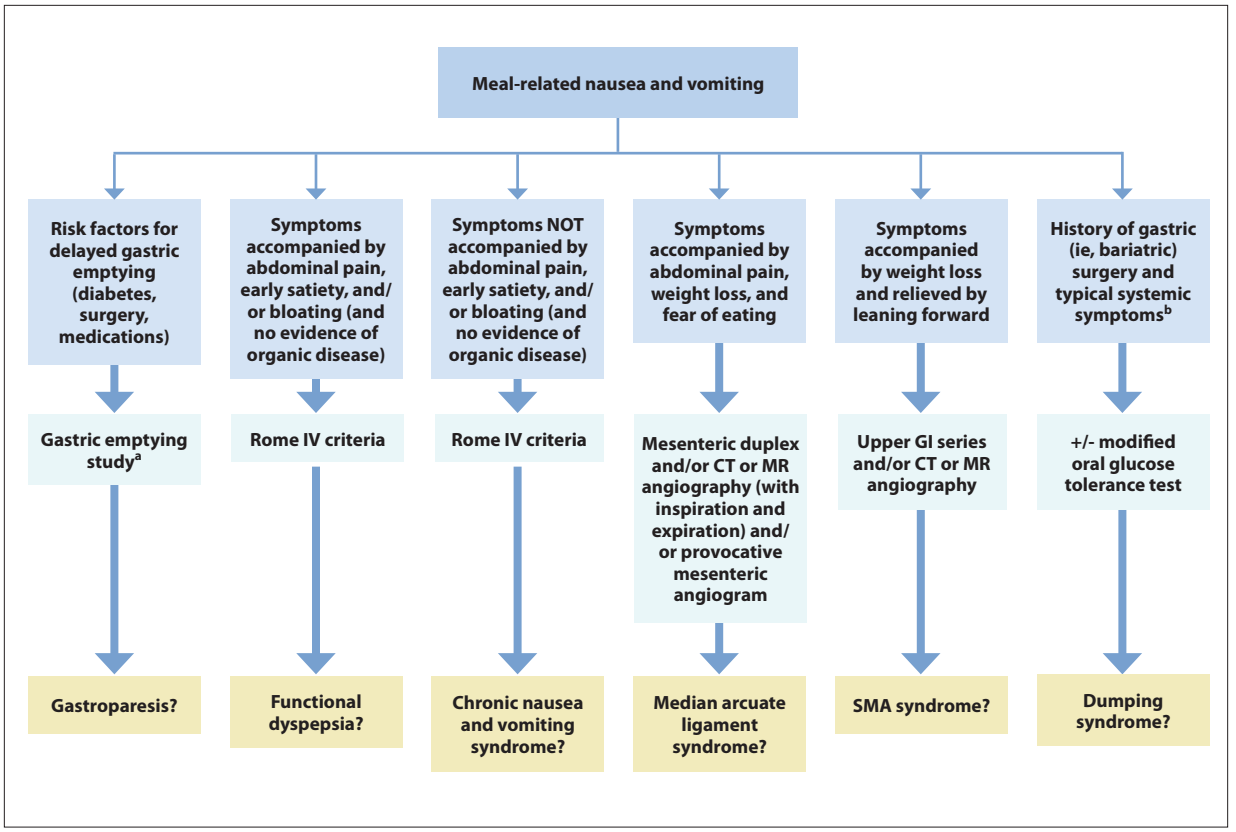

Figure. Proposed diagnostic approach to meal-related nausea and vomiting.

CT, computed tomography; GI, gastrointestinal; MR, magnetic resonance; SMA, superior mesenteric artery.

[a]To enhance diagnostic accuracy, the gastric emptying study should be performed according to guidelines (eg, solid test meal, 4-hour study, discontinuation of medications that may affect gastric emptying, fasting blood glucose <275 mg/dL prior to test).

[b]Systemic symptoms of dumping syndrome include heart palpitations, tachycardia, fatigue, flushing, pallor, lightheadedness, the urge to lie down, hypoglycemia, and hypotension.

iting include GP, cyclic vomiting syndrome, cannabinoid hyperemesis syndrome, rapid gastric emptying, functional dyspepsia (FD), median arcuate ligament syndrome (MALS), superior mesenteric artery (SMA) syndrome, gastric outlet obstruction, peptic ulcer disease, and intestinal pseudo-obstruction.[17] The pathophysiology of GP warrants mention, as it is a common reason for gastroenterology referral. Reduction in the number of interstitial cells of Cajal, injury to the enteric nervous system, nitric oxide dysregulation, vagal nerve injury, and alterations in the levels of gut hormones (eg, motilin, ghrelin, cholecystokinin, gastrin, glucagon-like peptide [GLP]) may all contribute to the generation of gastric dysrhythmias, ineffective antral contractions, delayed gastric emptying, and resulting symptoms of nausea and vomiting.[18-20] Although somewhat counterintuitive, rapid gastric emptying may also cause symptoms of nausea and vomiting. Impaired fundic accommodation (ie, after antireflux surgery), vagal nerve injury, medications, and abnormalities in hormones that impair gastric emptying (eg, glucagon, GLP-1, pancreatic polypeptide, amylin, leptin) may all contribute to the development of rapid gastric emptying.[21]

Diagnostic Evaluation

Diagnostic evaluation of meal-related nausea and vomiting encompasses a range of testing modalities aimed at identifying underlying abnormal gastrointestinal pathophysiology. The Figure contains a proposed diagnostic algorithm for the evaluation of meal-related nausea and vomiting.

Esophagogastroduodenoscopy (Upper Endoscopy)

Esophagogastroduodenoscopy (EGD) is indicated in patients presenting with pertinent physical examination findings or alarm features such as recurrent vomiting, gastrointestinal bleeding, unintentional weight loss, or

family history of inflammatory bowel disease or malignancy.[22] Upper endoscopy allows for direct visualization of the esophageal, gastric, and duodenal mucosa, enabling the identification of lesions and facilitating tissue biopsy for histopathologic diagnosis.[23] Potential etiologies identified via EGD include peptic ulcer disease, gastric outlet obstruction, *Helicobacter pylori* infection, and celiac disease.[17]

Upper Gastrointestinal Radiographic Series and Small Bowel Follow-Through

Upper gastrointestinal radiographic series and small bowel follow-through are valuable in detecting mucosal lesions as well as SMA syndrome in patients with meal-related nausea and vomiting. Small bowel follow-through aids in identifying high-grade obstructions and can also provide information regarding small bowel transit time, contributing to the diagnostic workup.[17,23]

Mesenteric Ultrasound

Mesenteric ultrasound plays a role in identifying stenosis of the celiac artery, SMA, and inferior mesenteric artery by assessing the velocity of blood flow through the mesenteric vessels. Thus, mesenteric ultrasound is particularly useful in cases of suspected mesenteric ischemia or MALS and can be used as a screening tool, as it is safe, noninvasive, relatively inexpensive, and generally well tolerated.[24,25]

Computed Tomography or Magnetic Resonance Enterography

Computed tomography (CT) or magnetic resonance (MR) enterography is useful for identifying intestinal obstruction or strictures, which can be seen in patients with inflammatory bowel disease or prior surgery or radiation. Additionally, CT or MR enterography may also detect extraluminal compression by abdominal or pelvic masses. MR imaging is preferred in younger patients or when repeat imaging is likely, owing to its lack of ionizing radiation.[26]

Gastric Emptying Scintigraphy

Gastric emptying scintigraphy (GES) remains the gold standard for assessing gastric emptying. This diagnostic method requires participants to consume a standardized meal (traditionally, 4 ounces of liquid egg whites infused with 0.5-1 mCi Technetium 99m sulfur colloid, 2 slices of white bread, 30 grams of strawberry jam, and water), ideally within 10 minutes.[27] Following ingestion, patients undergo gastric scintigraphy, during which the residual volume of the meal within the stomach is measured through evaluation of anterior and posterior images at baseline, 1, 2, 3, and 4 hours postprandially.[27] Guidelines encourage a number of protocol measures that are essential to ensuring diagnostic accuracy, including conducting a full 4-hour scan, withholding opiates and other medications that influence the gastric emptying rate (ie, prokinetic and anticholinergic agents), and ensuring that blood glucose levels are controlled at the time of testing (fasting blood glucose <275 mg/dL).[28] Failure to comply with these guidelines can lead to misdiagnosis of GP, as will be discussed in the following section.[29,30]

Specific Disorders and Treatments

Gastroparesis

GP is defined by upper gastrointestinal symptoms (nausea, vomiting, abdominal pain, early satiety, and/or bloating) in the setting of an objectively measured delay in gastric emptying and in the absence of mechanical obstruction.[30] Because delayed gastric emptying is the defining feature of GP, the act of eating is inherently problematic for patients with this condition; as such, GP is frequently considered in the evaluation of meal-related nausea and vomiting. However, it is important to recognize that GP itself is relatively uncommon. A recent systematic review of 13 epidemiologic studies of patients with GP involving mostly US databases or registries identified a prevalence ranging from 13.8 to 267.7 per 100,000 adults.[31] The etiology of GP is diverse, as the condition can occur as a complication of diabetes mellitus, surgery (eg, fundoplication, hiatal hernia repair, lung transplant), medications (eg, GLP-1 receptor agonists, opioids, anticholinergic agents), and ischemia as well as a variety of neurologic disorders, connective tissue disorders, and infections; GP can also be idiopathic in nature.[32] Whereas idiopathic GP was once thought to be the most common presentation of GP, updated epidemiologic data suggest that the majority (57.4%) of patients with GP experience it as a complication of diabetes.[33] The pathophysiology of GP is diverse and includes decreased gastric fundic tone, antroduodenal dyscoordination, gastric dysrhythmias, and abnormal duodenal feedback, as well as pyloric dysfunction in addition to delayed gastric emptying.[34,35]

Importantly, GP cannot be diagnosed without objective findings of delayed gastric emptying, which is most commonly assessed by GES, as mentioned; gastric emptying can also be measured with a breath test using ^{13}C-Spirulina.[36] However, many centers do not adhere to national guidelines when conducting GES, which can lead to misdiagnosis.[37] Updated guidelines by the American College of Gastroenterology (ACG) specify the importance of utilizing a solid meal to measure gastric emptying, ideally for 4 hours.[30] Notably, a recent retrospective study involving 339 patients referred to a tertiary referral center for evaluation of GP found that 80.5% of patients ultimately received alternative diagnoses after

Table. Disorder-Specific Treatments for Meal-Related Nausea and Vomiting

Medication/intervention	Oral dose
Gastroparesis/chronic nausea and vomiting syndrome	
Metoclopramide	5-10 mg three to four times daily
Domperidone[a]	10 mg three to four times daily
Ondansetron[b]	4-8 mg three times daily
Prochlorperazine[b]	5-10 mg four times daily
Chlorpromazine[b]	10-25 mg four times daily
Scopolamine[b]	1.5 mg every 3 days (patch)
Aprepitant[b]	80 mg daily
Tradipitant[b]	85 mg twice daily
Dronabinol[b]	5-10 mg three times daily
Gastric peroral endoscopic myotomy	Not applicable
Gastric electrical stimulation[c]	Not applicable
Functional dyspepsia/chronic nausea and vomiting syndrome	
Mirtazapine[b]	7.5-45 mg daily
Buspirone[b]	15-90 mg daily (divided into twice-daily or thrice-daily dosing)
Dumping syndrome	
Acarbose	25-100 mg three times daily
Octreotide[b]	50 mcg three times daily (subcutaneous); 20 mg once monthly (intramuscular)
Median arcuate ligament syndrome	
Celiac plexus block	Not applicable
Surgical release of median arcuate ligament	Not applicable
Superior mesenteric artery syndrome	
Weight gain (may require enteral or parenteral nutrition)	Not applicable
Gastrojejunostomy	Not applicable

[a]Only available in the United States via US Food and Drug Administration investigational drug protocol.
[b]Off-label use.
[c]Approved as a humanitarian use device for medically refractory diabetic and idiopathic gastroparesis.

tertiary evaluation.[29] The lack of a properly performed gastric emptying study was identified as a significant factor leading to misdiagnosis of GP, as only 6.8% of patients were known to have undergone a 4-hour study that utilized a standard test meal of radiolabeled eggs.[29] It should also be mentioned that while less than 90% gastric emptying (>10% retention) of a solid meal at the 4-hour mark on GES has traditionally been used as a cutoff to define GP, updated ACG guidelines suggest that this cutoff may need to be reconsidered, as mild delays in gastric emptying are commonly found in patients with FD, which will be discussed in the following section.[30] In summary, proper performance and interpretation of GES is critical to making an accurate diagnosis of GP.

Treatment of GP includes dietary therapy (frequent, small volume, low-fat, low-fiber meals; small particle diet), prokinetic therapy (eg, metoclopramide, domperidone, erythromycin, prucalopride), antiemetic therapy (eg, ondansetron, promethazine, aprepitant), behavioral therapy, and interventions such as gastric electrical stimulation and pyloromyotomy (see the Table for a list of disorder-specific treatments).[30] Notably, only 1 medication (metoclopramide) is currently approved by the US Food and Drug Administration for treatment of GP. However, a recently developed intervention, gastric peroral endoscopic myotomy (G-POEM), has demonstrated efficacy for the treatment of GP and is becoming increasingly utilized in clinical practice, particularly in patients with medically refractory symptoms of nausea and vomiting related to GP.[30,38] Importantly, data supporting G-POEM as a treatment for GP had historically involved only noncontrolled studies. However, a recent randomized,

sham-controlled trial involving 41 patients with severe GP demonstrated significant improvement in symptoms and gastric emptying 6 months after G-POEM compared with a sham procedure for the first time.[39] Although these results are encouraging, additional large sham-controlled trials, ideally with long-term follow-up, are still needed.

Functional Dyspepsia

FD is a common disorder of gut-brain interaction (DGBI) characterized by symptoms of epigastric pain or pressure, early satiety, postprandial fullness, bloating, nausea, and vomiting. The Rome IV criteria further categorize FD into 2 subgroups: epigastric pain syndrome and postprandial distress syndrome (PDS).[40] Notably, FD patients with PDS, in particular, experience a significant relationship between eating and symptoms. Thus, in patients with meal-related nausea, FD should be a consideration, especially given its prevalence. In contrast with GP as described previously, the worldwide prevalence of FD has been estimated to be 7% to 10% and around 12% in the United States alone.[41,42] The etiology of FD is diverse and incompletely understood. Psychological factors, medications (eg, anti-inflammatory agents), tobacco use, disturbances in the gut microbiome, female sex, and gastrointestinal infections (namely *H pylori*) have been identified as risk factors.[43-45] The pathophysiology of FD is complex. Mild delays in gastric emptying, impaired gastric accommodation, visceral and central hypersensitivity, abnormal duodenogastric feedback, alterations in the microbiome, microscopic inflammation in the duodenum, and increased intestinal permeability have all been implicated as potential causative mechanisms.[46-50] Furthermore, it should be mentioned that although GP and FD were once viewed as distinct disorders, there is a growing consensus among experts in the field that these disorders should be considered a part of the same spectrum of gastric sensorimotor dysfunction given that they share similar symptoms (ie, nausea, early satiety, abdominal pain), some pathophysiologic mechanisms (ie, mildly delayed gastric emptying in patients with FD), and some treatments (ie, prokinetics, antiemetics, neuromodulators).[47,51] Additionally, in a recent trial conducted by the National Institutes of Health Gastroparesis Clinical Research Consortium involving 944 patients, 42% of patients initially diagnosed with GP were found to have normal gastric emptying at the conclusion of the 48-week study, whereas 37% of patients with normal gastric emptying transitioned to a diagnosis of GP with findings of delayed gastric emptying at the conclusion of the study.[51] The marked transition between diagnoses of GP and FD among patients in this landmark trial provides further evidence of the significant overlap that exists between these 2 disorders.

FD can be diagnosed by using the Rome IV criteria to be certain that typical symptoms are present and by performing limited diagnostic tests (eg, upper endoscopy in appropriate patients) to help exclude an organic disorder.[40,52] As mentioned, patients with the PDS subtype of FD report postprandial fullness and/or early satiation at least 3 days per week. Nausea can accompany symptoms of FD, but the presence of significant vomiting should prompt the clinician to consider other diagnoses. Notably, chronic nausea and vomiting syndrome (CNVS) is another DGBI that is characterized by nausea and/or vomiting at least once per week; a relative lack of abdominal pain, early satiety, and bloating help distinguish CNVS from FD.[40] Additionally, a relationship between eating and symptoms is not required to make a diagnosis of CNVS; hence, this disorder will not be discussed at length in this article.

Treatment of FD includes eradication of *H pylori*, proton pump inhibitor therapy, or treatment with tricyclic antidepressants or prokinetic agents.[53] Other neuromodulators, such as mirtazapine and buspirone, are also used in practice, particularly for patients with meal-related symptoms.[54-56] For patients who do not respond to drug therapy, guidelines recommend consideration of psychological therapies, such as cognitive behavioral therapy and hypnotherapy.[52] Virtual reality has also recently been shown to improve symptoms of FD in a small randomized, double-blind, sham-controlled trial.[57]

Dumping Syndrome

Dumping syndrome (DS) presents with postprandial systemic and gastrointestinal symptoms, commonly following foregut surgeries such as Roux-en-Y gastric bypass, vagotomy with pyloroplasty, sleeve gastrectomy, esophagectomy, and Nissen fundoplication.[58,59] Gastrointestinal symptoms include borborygmi, epigastric pain, bloating, nausea, and diarrhea, whereas systemic symptoms include heart palpitations, tachycardia, fatigue, flushing, pallor, lightheadedness, the urge to lie down, hypoglycemia, and hypotension. These symptoms arise from rapid fluid shifts owing to food osmolality, resulting from accelerated transit of ingested contents to the small intestine, leading to abdominal bloating, distension, hyperinsulinemia mediated by exaggerated GLP-1 response, and release of vasoactive gastrointestinal hormones.[59]

Early DS typically occurs 1 hour postprandially, whereas late DS occurs 1 to 3 hours after meal ingestion.[58-62] The modified oral glucose tolerance test aids diagnosis, identifying early DS by heart rate elevation over 10 bpm or 3% hematocrit rise within 30 minutes postprandially, and late DS by postprandial hypoglycemia. Combining oral glucose tolerance test and validated

questionnaires such as the Sigstad Score and Arts Dumping Questionnaire enhances diagnostic sensitivity.[59,63] Initial DS treatment involves dietary modifications with small, frequent meals, avoiding simple carbohydrates and premeal liquids. Acarbose can prevent carbohydrate cleavage in refractory cases.[59,60,62] Somatostatin analogues such as octreotide reduce splanchnic vasodilation in patients unresponsive to diet and acarbose.[62,64] Surgical or endoscopic revisions, such as transoral outlet reduction post–Roux-en-Y gastric bypass, may be necessary if pharmacologic interventions fail.[65-67]

Superior Mesenteric Artery Syndrome

SMA syndrome, first described in 1842 by von Rokitansky and characterized in 1927 by Wilkie, presents as a rare cause of meal-related nausea and vomiting.[68,69] It occurs when the third part of the duodenum is compressed externally between an acute angle formed by the SMA and the aorta. This compression obstructs duodenal transit, leading to postprandial symptoms such as nausea, vomiting, distension, and epigastric pain; symptoms can frequently be alleviated by bending forward or lying in a knee-to-chest position.[70] The vascular angle change typically follows rapid weight loss, reducing retroperitoneal fat that normally separates the SMA and aorta. This change can then perpetuate symptoms, leading to further weight loss and exacerbation of SMA syndrome.[70,71] Diagnosis of SMA syndrome is based on symptoms and radiologic evidence of duodenal obstruction by the SMA. This can be assessed with an upper gastrointestinal radiographic series showing an abrupt compression of the third portion of the duodenum with resulting dilation of the proximal duodenum, with or without gastric dilation.[70] Historically, CT angiography has been considered the standard to diagnose SMA syndrome, as this modality can assess the aortomesenteric angle (normal >25 degrees) and distance (normal >8 mm), as well as detect evidence of duodenal obstruction.[70-73] Similar to CT angiography, MR angiography can also be performed, and mesenteric ultrasound has recently emerged as a modality for measuring the aortomesenteric distance with sensitivity similar to CT.[74] Initial management includes gastric decompression and enteral or parenteral nutrition to rebuild retroperitoneal fat.[75] Gastrojejunostomy is considered if conservative measures fail.[76]

Median Arcuate Ligament Syndrome

Arising anteriorly from the aorta, the celiac artery passes through the diaphragm at the level of the twelfth thoracic vertebra. The artery lies underneath the median arcuate ligament; this connects the 2 medial borders of the diaphragm and is surrounded by the celiac plexus and the celiac ganglia. If the median arcuate ligament is displaced inferiorly or if the celiac artery is displaced superiorly from its typical position, compression of the artery may occur, leading to symptoms labeled MALS. First reported in 1965,[77] celiac artery compression may lead to symptoms of abdominal pain, nausea, vomiting, and weight loss. Symptoms may worsen during expiration as the diaphragm moves upward, stretching the diaphragmatic crura, and exacerbating compression of the artery. Physical examination may reveal tenderness in the right upper quadrant, whereas auscultation in a quiet room may uncover a bruit or thrill. The diagnosis of this uncommon disorder is controversial, in part because compression of the celiac artery has been identified in more than 10% of an asymptomatic population.[78] Symptomatic celiac artery compression (MALS) is more commonly identified in thin women between the ages of 30 and 50 years.[79] Anatomic/structural causes should be ruled out by EGD; hepatobiliary causes can be excluded with appropriate laboratory tests and an ultrasound. GES can identify delayed gastric emptying. In the appropriate patient, a mesenteric ultrasound should be the first test performed, as it is safe, noninvasive, and has high yield.[80] An expiratory peak velocity of greater than 200 cm/s supports the diagnosis of MALS.[81] However, this needs to be confirmed with either a CT angiogram or MR angiogram. In many centers, a definitive diagnosis is made with an angiogram and provocative maneuvers (eg, injection of papaverine, celiac blockade). Treatment involves laparoscopic surgical release of the median arcuate ligament; concomitant celiac plexus neurolysis is often performed. Fifty percent to 80% of patients note an improvement in symptoms.[82,83]

Conclusion

Nausea and vomiting are common, bothersome symptoms that frequently lead to evaluation in the clinic and hospital setting. The differential diagnosis for nausea and vomiting is vast, and it is often helpful to first separate gastrointestinal causes from nongastrointestinal ones. Because eating can be a trigger for symptoms, it is also helpful for the clinician to recognize and understand the conditions associated with meal-related nausea and vomiting specifically, including GP, FD, DS, SMA syndrome, and MALS. These conditions can often be distinguished by differences in presentation and results of diagnostic testing. A careful selection of diagnostic tests, utilization of the Rome criteria (to make an accurate diagnosis of DGBIs such as FD), and an appreciation for the epidemiology of the aforementioned conditions can further aid the clinician in making an accurate diagnosis. Notably, if GES is performed, it is important to do so according to guidelines and to interpret the results properly to make an accurate diagnosis of GP as well as to distinguish the

condition from FD. Fortunately, there are a number of medications and interventions for nausea and vomiting within the treatment armamentarium, and it is important for the clinician to select an appropriate treatment based on the specific cause for the patient's meal-related symptoms.

Disclosures

The authors have no relevant conflicts of interest to disclose.

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