His Upper GI Tract

5

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5.1 Introduction

The upper gastrointestinal (GI) tract extends from the mouth to the esophagus and then down through the stomach to the duodenum and ends at the terminal ileum. Problems in the upper GI tract are a common source of patient complaints. In the United States, 45 % of respondents to one survey reported one or more upper GI symptoms in the previous 3 months [1]. Among men, the most common symptoms are heartburn, dysphagia, early satiety, and bloating (in descending order). These symptoms are associated with significant impact in terms of both missed work and leisure days as well as physician visits [1, 2].

Patients often present with a combination of symptoms which can make diagnosis difficult. Careful history taking is key in determining where to start the diagnostic workup and initial therapeutic treatments. It is important to note whether these symptoms are acute or chronic and how they relate to food ingestion and time of day. Dietary habits, alleviating (or worsening) factors, current medications and supplements, and the patient's previous surgical history are also critical.

Since there is no shortage of disease-specific resources available in textbooks and online, we organized this chapter by common presenting symptoms. We broke down the symptoms into five broad categories: heartburn and reflux, upper abdominal pain and discomfort, esophageal dysphagia, chronic nausea and vomiting, and bleeding. Within each category, we discuss common diseases, suggest initial

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D.E. Azagury, M.D. (⊠) Assistant Professor of Surgery, Stanford University School of Medicine, 300 Pasteur Drive, Stanford, CA 94305, USA e-mail: dazagury@stanford.edu diagnostic studies and therapies, note important risk factors, and review potential reasons for specialist referrals. Keep in mind that many patients will have symptoms from more than one category, so the workup needs to be tailored to individual cases.

5.2 Heartburn and Reflux

Heartburn (defined as burning chest pain occurring after eating) and regurgitation (the presence of refluxed gastric contents and acid into the mouth or hypopharynx) are among some of the most common problems reported by patients. While an initial diagnosis of gastroesophageal reflux disease (GERD) is often appropriate, non-GI etiologies should always be assessed and ruled out first. Specifically, causes such as angina often carry similar risk factors and patient profiles as GERD (e.g., obesity).

5.2.1 Gastroesophageal Reflux Disease

GERD is a chronic disorder that occurs when acid (or other GI contents, such as bile) flows backward into the esophagus. While a small amount of reflux is normal, heartburn is considered troublesome when mild symptoms occur two or more times per week or severe symptoms occur at least once per week [3]. Surveys of Western countries suggest that 8–27 % of the population experience heartburn or acid regurgitation on a weekly basis, and nearly half of these individuals have been suffering from these symptoms for 5 or more years [4]. Asian countries seem to have a lower prevalence ranging from 3 to 5 %.

While the most common symptoms of GERD are heartburn and regurgitation, other presenting symptoms may include chest pain, globus (the perception of a lump in the throat not related to swallowing), nausea, nighttime or chronic cough, hoarseness, or bitter or sour tastes in the morning. The diagnosis of GERD is usually clinical though the same symptoms are also seen in diseases such as esophagitis, peptic ulcer disease, biliary tract disease, coronary artery disease, and esophageal dysmotility disorders.

When the diagnosis of GERD is suspected, it is reasonable to start acid suppression treatment with an H2 blocker or a proton pump inhibitor (PPI). Patients can also be encouraged and counseled on weight loss (if overweight or obese), elevating the head of the bed to minimize nighttime symptoms, and eating meals 2–3 h before bedtime. Dietary experimentation can also be helpful.

Keep in mind that improvement of symptoms after starting treatment does not definitively make the diagnosis. About half of patients without GERD (as confirmed by endoscopy and other testing) will still improve on PPI treatment [5]. Clinical suspicion for other diseases and the presence of alarm symptoms or risk factors should generally prompt further workup. Some indications for further testing (generally starting with upper endoscopy) are listed in Table 5.1.

Table 5.1 Indications for	• Failure of twice-daily H2/PPI treatment after 4–8 weeks
upper endoscopy [6]	Alarm symptoms
	 Pain with swallowing or difficulty swallowing
	– Bleeding
	– Anemia
	 Involuntary weight loss or weight loss >10 %
	 Previous upper GI malignancy
	 Previously documented peptic ulcer disease
	– Lymphadenopathy
	 Abdominal mass
	History of erosive esophagitis
	• Men >50 years old with one of the following:
	 GERD symptoms for 5 or more years
	 Risk factors of Barrett's esophagus and esophageal adenocarcinoma
	Nocturnal reflux
	Hiatal hernia
	Overweight or obese
	Tobacco use
	Intra-abdominal distribution of fat

Ambulatory 24-h pH monitoring is typically used in the setting of normal or equivocal endoscopic findings with refractory reflux symptoms to confirm diagnosis. A barium swallow and a manometry study will often be requested prior to a surgical procedure and are useful to assess anatomy and/or contraindications to anti-reflux surgery.

Since the development of acid suppression therapy, the number of operations performed for GERD has decreased dramatically. Nonetheless, anti-reflux operations can be a very effective treatment, particularly for medically refractory GERD. Other indications for anti-reflux surgery are listed in Table 5.2. Endoscopic therapies and minimally invasive surgical therapies offer a good alternative for patients who fail to respond to PPI therapy. Laparoscopic fundoplication surgery remains the gold standard of these therapies. In this operation, the stomach is mobilized and wrapped around the esophagus to reinforce the lower esophageal sphincter (Fig. 5.1) [8]. It provides long-standing symptom relief with very low complication rates and short hospital stays (typically 1-3days). Surgery can often be very durable up to 20 years after the initial operation, though a small subset of patients will still require acid suppression therapy or surgical revision [9]. Because of the association between obesity and reflux, morbidly obese patients will often suffer from GERD. These patients should be referred to a bariatric surgeon as laparoscopic Roux-en-Y gastric bypass is very effective in reducing GERD symptoms in conjunction with significant and longlasting weight loss [10–12].

Table 5.2 Indications for anti-reflux surgery [7]	Failed medical management
	 Inadequate symptom control
	 Severe regurgitation not controlled with acid suppression
	 Medication side effects
	Opt for surgery despite successful medical management
	 Quality of life
	 Lifelong need for medication intake
	 Expense of medications
	Complications of GERD
	 Severe esophagitis
	 Barrett's esophagus^a
	Extra-esophageal manifestations
	– Asthma
	– Hoarseness
	– Cough
	 Chest pain
	– Aspiration
	^a Strong indication when symptomatic. However, while patients with asymptomatic Barrett's reportedly regress to a greater

"Strong indication when symptomatic. However, while patients with asymptomatic Barrett's reportedly regress to a greater degree after surgery, there is no reduction in esophageal adenocarcinoma rates

5.3 Upper Abdominal Pain or Discomfort

Chronic or recurrent upper abdominal (epigastric) pain or discomfort is referred to as dyspepsia. It is often accompanied by other symptoms such as heartburn, reflux, early satiety, a sensation of fullness, bloating, belching, and nausea [13]. The most common organic causes of dyspepsia are peptic ulcer disease, GERD, gastric malignancy, and nonsteroidal anti-inflammatory drug (NSAID)-induced dyspepsia. However, the diagnostic workup for dyspepsia is often negative for any identifiable diseases leading to functional or idiopathic dyspepsia as a diagnosis of exclusion.

In patients younger than 55 years old without alarm symptoms or other risk factors (Table 5.1), it is reasonable to begin with either empiric PPI treatment or *Helicobacter pylori* testing. The latter is preferred in areas where the prevalence of *H. pylori* is greater than 10 % [13]. Starting with either of these approaches appears to be equally cost effective [14]. Treatment failure, age > 55, or alarm symptoms all warrant an upper endoscopy.

5.3.1 Peptic Ulcer Disease

Peptic ulcer disease (PUD) encompasses ulcers occurring in either the stomach or the duodenum. Pain from these ulcers is often described as burning or gnawing hunger. Classical teaching describes gastric ulcer pain occurring shortly after

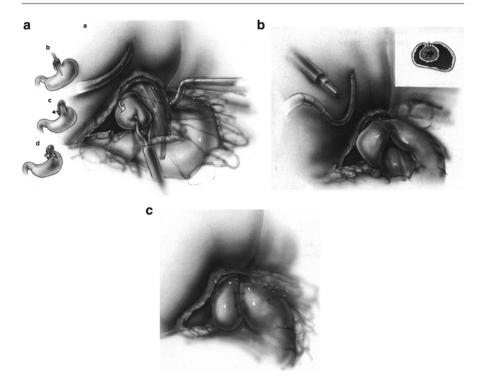


Fig. 5.1 Illustrations from "Laparoscopic Nissen Fundoplication" in *Chassin's Operative Strategy in General Surgery*, 2013, pp. 203–213, Ed. Carol E.H. Scott-Conner. With permission of Springer Science+Business Media. (a) In a Nissen fundoplication, the mobilized stomach is brought behind the esophagus. (b) Sutures are then used to bring the fundus over the esophagus. The fundoplication should come together without any tension. At least one of the sutures will also incorporate the muscular layer of the esophagus to fix the fundoplication in place. (c) The completed fundoplication should be loose enough to allow an instrument to be passed between the wrap and the esophagus. Overly tight wraps can lead to dysphagia

meals, whereas duodenal ulcer pain occurs several hours afterward due to acid secretion in the absence of food. Practically speaking however, once PUD is suspected, then upper endoscopy will confirm or rule out the diagnosis and allow biopsies to be taken for *H. pylori* and malignancy testing. Examples of normal endoscopic anatomy are shown in Fig. 5.2 [15], while benign and malignant ulcers are shown in Fig. 5.3 [16, 17].

The goal of medical management is to heal the ulcer and to treat *H. pylori* if present. Eradication of *H. pylori* reduces ulcer recurrence and complications, although up to 20 % of patients will develop recurrent ulcers [18]. Traditional "triple" therapy with a PPI (twice-daily dosing except for esomeprazole which is once daily), amoxicillin (1 g twice daily), and clarithromycin (500 mg twice daily) for 10–14 days is the recommended first-line treatment for most patients [19, 20]. Metronidazole (500 mg twice daily) can be substituted for amoxicillin in patients with penicillin allergies. However, resistance levels to clarithromycin appear to be rising in many

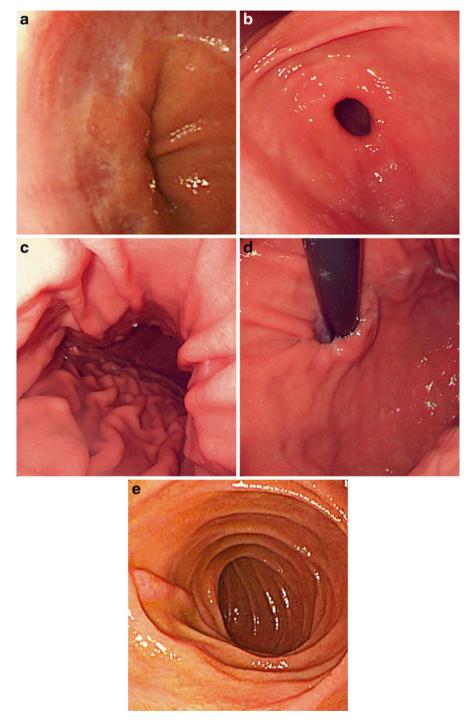


Fig. 5.2 Examples of normal upper endoscopy findings. From Clinical Gastrointestinal Endoscopy: A Comprehensive Atlas, 2014, ed. Hoon Jai Chun, Suk-Kyun Yang, Myung-Gyu Choi. With permission of Springer Science + Business Media. (a) Normal gastroesophageal junction. (b) Antrum and pylorus. (c) Body of the stomach. (d) Retroflex view of the cardia. (e) Second portion of the duodenum



Fig. 5.3 Examples of benign and malignant peptic ulcer disease. Figure (**a**), (**b**), and (**c**) from Clinical Gastrointestinal Endoscopy: A Comprehensive Atlas, 2014, ed. Hoon Jai Chun, Suk-Kyun Yang, Myung-Gyu Choi. With Permission of Springer Science + Business Media. Figure (**d**) and (**e**) from Gastric Cancer, Follow-up endoscopy for benign-appearing gastric ulcers has no additive value in detecting malignancy, 2014, Gielisse EAR, Kuyvenhoven JP, Fig. 1c, d. With permission from the Japanese Gastric Cancer Association, Dr. Eric Gielisse, and Springer Science + Business Media. (**a**) Benign well-circumscribed and deep penetrating ulcer. (**b**) Benign ulcer with a clean base and a regular shape without exudate. (**c**) Well-healed ulcer. (**d**) A 15-mm malignant ulcer with a clean base but an irregular border. (**e**) A 3-cm malignant ulcer with a dirty base as well as an elevated and irregular border

countries leading to treatment failure. In these high-resistance areas or with patients who have failed previous therapy, "quadruple" therapy with a PPI, metronidazole 250 mg and tetracycline 500 mg four times daily, and bismuth (525 mg four times daily) is the recommended regimen. Alternatively, a levofloxacin-based "triple" therapy consisting of a PPI, amoxicillin (1 g twice daily), and levofloxacin (500 mg once daily) can be used.

H. pylori eradication should be confirmed using urea breath testing or stool antigen testing [20]. Patients should also stop NSAID use, alcohol intake, and smoking if possible. For uncomplicated duodenal ulcers, PPI treatment for 4 weeks is usually sufficient and no further testing is necessary [21]. Gastric ulcers should be treated with 8 weeks of PPIs; refractory ulcers should generally receive a twice-daily regimen for another 6–8 weeks. Long-term maintenance therapy with PPIs is generally reserved for high-risk patients with a history of complications (e.g., bleeding, perforation, gastric outlet obstruction) and recurrent or refractory ulcers and those with giant or fibrosed ulcers [20].

The role of follow-up endoscopy in documenting ulcer resolution and allowing for repeat biopsies of concerning lesions remains under some debate. For lesions with equivocal or concerning histology, repeat endoscopy is indicated. However, for patients with initially benign-appearing ulcers (both in endoscopic appearance and histological examination), the rate of malignancy found on follow-up endoscopy is 0–4 % [17, 22, 23]. While earlier detection of cancer may confer some survival benefit, the magnitude of that benefit is not yet well known and follow-up endoscopy is probably not cost effective [23, 24]. Nonetheless, overutilization of follow-up endoscopy, particularly in low-risk patients, appears to be common [25].

Refractory or recurrent ulcers can occur for a wide variety of reasons including persistent *H. pylori* infection, NSAID use, underlying malignancy, or acid hypersecretion (e.g., Zollinger-Ellison syndrome). Identifying and treating the underlying etiology is important to prevent life-threatening complications such as bleeding, perforation, and obstruction [26]. The overall mortality from PUD complications has decreased over time, but is still 3 % for those requiring hospitalization and 11 % for patients undergoing surgery.

If medical therapy is not effective or there is concern about malignancy or the development of complications such as gastric outlet obstruction, then referral to a surgeon is warranted. Surgical options include decreasing acid secretion through vagotomies, removal of the acid-secretion portion of the stomach, excision of ulcers, and gastric drainage procedures to improve emptying.

5.3.2 Biliary Tract Disease

Gallstones are found in approximately 8–10 % of men in the United States and Europe, although that number can be as high as 30 % in certain communities [27]. Most patients are asymptomatic and will not require treatment. The most common symptom is epigastric pain lasting for at least 30 min [28]. Contrary to the classic description of biliary colic, the pain is often steady and constant. Atypical

symptoms such as chest pain, belching, early satiety, bloating, burning epigastric or chest pain, and nausea and vomiting may also be seen in patients with gallstones [28].

Patients with symptomatic cholelithiasis will often have an unremarkable physical examination and laboratory tests. The presence of a Murphy's sign (pain on deep inspiration while palpating just below the liver edge) is concerning for cholecystitis. In the presence of fevers, chills, and/or leukocytosis, acute cholecystitis should be considered, and the patient should be referred urgently to the emergency department and a surgeon.

Other diagnoses that should be considered include choledocholithiasis, sphincter of Oddi dysfunction, functional gallbladder disorders (biliary pain in the absence of stones or sludge), GERD, PUD, hepatitis, esophageal disorders, ischemic heart disease, pancreatitis, irritable bowel syndrome, and urinary tract infections or stones. In general, abnormal liver or pancreatic tests in the presence of gallstones should prompt a rapid referral for further evaluation.

The most sensitive and specific test for biliary tract disease is a right upper quadrant ultrasound. If the study is negative but gallstones are still suspected, then a repeat ultrasound after several weeks is reasonable. If both ultrasound studies are negative, then referral to specialists is advisable. Further tests may include upper endoscopy, endoscopic ultrasound, endoscopic retrograde cholangiopancreatography (ERCP), cholescintigraphy (99mTc-hepato-iminodiacetic acid or "HIDA" scans), and esophageal manometry.

Asymptomatic patients with gallstones do not need a prophylactic cholecystectomy as most will not develop symptoms [29]. However, those with symptoms should undergo cholecystectomy because the risk of recurrent symptoms is 30–50 %. Outpatient laparoscopic cholecystectomy is well tolerated by most patients with low rates of serious complications. Cost-effectiveness studies suggest that medical management is actually associated with increased cost and patient disability, added ERCP procedures, and the possible development of gallbladder cancer [30].

Medical management with ursodiol (ursodeoxycholic acid) remains an uncommon treatment for gallstones. It can be an option in select patients with mild or infrequent episodes of biliary pain, biliary sludge or cholesterol gallstones <20 mm in diameter, and a patent cystic duct [31]. In this population, 90 % will have complete dissolution of small stones after 6 months of treatment [32]. The recommended treatment dose is 8–10 mg/kg/day taken at bedtime [31]. Patients should undergo a follow-up ultrasound after 6–12 months of treatment. Patients who have minimal to no decrease in stone size will most likely never dissolve their stones. Once the stones or sludge disappear on ultrasound, patients can transition to low-dose maintenance therapy of 300 mg/day. Maintenance therapy decreases the recurrence of gallstones and future complications such as gallstone pancreatitis [33, 34]. However, medical treatment requires significant patient compliance and is burdened by an extremely high recurrence rate: nearly half of these patients will develop gallstones again after 5 years and up to three-quarters at 12 years [35].

5.4 Esophageal Dysphagia

Esophageal dysphagia (difficulty swallowing) is an alarm symptom which should prompt a careful workup. Unlike oropharyngeal disorders which typically result in symptoms immediately upon food intake, esophageal disorders generally occur a few seconds after swallowing. Patients will often describe a sensation of having food stuck in their chest behind the sternum. The types of foods that cause the dysphagia can help narrow the list of possible diagnoses. Patients reporting problems with both liquids and solids initially are more likely to have an esophageal dysmotility disorder, whereas progressive dysphagia starting with solids and then later with liquids is consistent with mechanical obstruction. Other important questions to ask include whether the patient has a history of radiation therapy to the head and/or neck, smoking or alcohol use, dry mouth or eyes, referred pain, changes in speech, halitosis, food regurgitation, or pain with swallowing.

The workup for esophageal dysphagia generally begins with upper endoscopy [36]. However, if there is a suspicion for a proximal disorder (e.g., Zenker's diverticulum or previous radiation therapy to the neck, a barium swallow can help map out the anatomy. Treatment will vary with the diagnosis, but generally requires consultation with specialists.

5.4.1 Mechanical Obstruction

Mechanical obstruction of the esophagus can be caused by problems within the esophagus or by external compression. The most common disorder is food bolus impaction which is typically seen in the elderly or those with peptic strictures. However, there is an increasing association over time between food bolus impaction and eosinophilic esophagitis (EoE) [37]. EoE is an allergic inflammatory disorder which most commonly presents as dysphagia in adults [38]. First-line treatment starts with dietary modification to identify and avoid inciting allergens and topical corticosteroids.

Benign esophageal strictures can also obstruct the passage of food. They are most commonly associated with GERD, and in these patients, PPI treatment can improve the dysphagia symptoms and decreases the need for repeat balloon dilation [39]. Strictures can also be due to pills, radiation, infection, or lymphocytic esophagitis.

Another source of obstructions is esophageal rings and webs [36]. Esophageal rings are either mucosal (most common) or muscular rings located in the distal esophagus. Esophageal webs are thin mucosal webs commonly found in the cervical esophagus. Barium esophagrams are more sensitive than endoscopy in diagnosing both and treatment is typically esophageal balloon dilation.

Finally, both benign and malignant masses of the esophagus and stomach can obstruct the passage of food. In North America and Europe, the most common esophageal malignancy is adenocarcinoma, whereas squamous cell carcinoma is the predominant malignancy in the rest of the world [40]. The major risk factors for adenocarcinoma are GERD, smoking, and obesity. Alcohol, especially in conjunction with smoking, is the major risk factor for squamous cell carcinoma.

5.4.2 Esophageal Dysmotility

When a dysmotility disorder is suspected or if the upper endoscopy and barium fluoroscopy are unrevealing, the test of choice is esophageal manometry. Some disorders such as achalasia or diffuse esophageal spasm have associated radiological findings ("bird's beak" and "corkscrew," respectively). However, not all patients have the typical radiological findings, and manometry is still needed to establish the diagnosis. Novel therapies such as peroral endoscopic myotomy (POEM) for achalasia can now be performed completely endoscopically with extremely effective functional results [41].

Esophageal dysmotility can also be due to systemic diseases such as scleroderma, systemic lupus erythematosus, rheumatoid arthritis, or mixed connectivetissue disorders [42]. Other disorders to consider include Chagas' disease, chronic idiopathic intestinal pseudo-obstruction, diabetes mellitus, amyloidosis, alcoholism, and multiple sclerosis.

5.4.3 Functional Dysphagia

In order for a patient to be diagnosed with a functional dysphagia, symptoms must persist for at least 3 months, and the symptoms should be present at least 6 months before the diagnosis is made [43]. In addition, GERD, esophageal dysmotility, and structural and metabolic disorders must be excluded.

5.5 Chronic Nausea and Vomiting

Nausea (the feeling of sickness marked by an urge to vomit) and vomiting (the expulsion of stomach contents through the mouth) are caused by a variety of disorders. This section will focus on chronic nausea and vomiting as most acute episodes are self-limiting (e.g., gastroenteritis), easily treated by stopping the inciting cause (e.g., medications), or controllable with anti-nausea medications. The diagnostic workup for chronic disorders should take place in parallel with treatment of the complications and sequela of nausea and vomiting [44]. Patients with chronic vomiting may be dehydrated or have electrolyte imbalances and acid-base disturbances. While there are a wide variety of disorders which cause nausea and vomiting, questions regarding the onset and timing of symptoms, the characteristics of the vomitus, the presence or absence of associated abdominal pain, and other associated symptoms and findings can help elucidate the diagnosis (see Table 5.3) [45].

Upper endoscopy is generally the first step in diagnosing chronic nausea and vomiting. Other tests to consider include CT scans or upper GI fluoroscopy, endocrine workup, gastric motility studies, and a psychological evaluation. A negative workup after these studies suggests a functional disorder.

Туре	Diagnoses
Gastrointestinal	GERD
	PUD
	Biliary tract disease
	Irritable bowel syndrome
	Hepatitis
	Pancreatic disease
	Irritable bowel syndrome
	Inflammatory bowel disease
	Chronic intestinal pseudo-obstruction
Obstructive	Esophageal disorders
	Malignancy
	Volvulus
Decreased gastric emptying	Gastroparesis
	Gastric outlet obstruction
	Malignancy
Medications	Antibiotics
	Opioids
	Chemotherapy
	Antiarrhythmics
	Anticonvulsants
Neurogenic	Intracranial neoplasm
	Head injury
	Stroke
	Infection
	Migraines
	Seizures
	Vestibular disease
Psychiatric	Anxiety
	Depression
	Conversion disorder

 Table 5.3
 A partial list of etiologies causing chronic nausea and vomiting

Adapted from Scorza et al. [45]

5.5.1 Gastroparesis

Gastroparesis is delayed gastric emptying in the absence of mechanical obstruction. The most common causes of gastroparesis are idiopathic, diabetic, and postsurgical, but a variety of other diseases such as Parkinson's, scleroderma, and mesenteric ischemia can be responsible [46]. In addition to nausea and vomiting, patients may also complain of bloating, early satiety, and abdominal pain. Many patients with idiopathic gastroparesis will have functional disorders such as functional dyspepsia (86 %) and irritable bowel syndrome (65 %) [47]. Idiopathic gastroparesis can also

occur after a systemic viral illness. Postsurgical gastroparesis is rare but may occur after abdominal surgery (usually in the upper abdomen) [48]. Diabetic gastroparesis is found in 5 % of type 1 diabetics and 1 % of type 2 diabetics [49].

A scintigraphic gastric emptying study where patients eat a radiolabeled eggwhite meal is the gold-standard diagnostic test [50]. Patients retaining >90 % at 1 h, >60 % at 2 h, and >10 % of the meal at 4 h are generally considered to have delayed gastric emptying. Alternative forms of testing include wireless capsule motility testing and breath testing.

The first-line management of gastroparesis is dietary modification, nutritional support, and glycemic control [46]. Patients should try eating small and frequent meals and avoid fat and nondigestible fibers (e.g., fruits, vegetables). Those unable to tolerate solids can try pureed foods. Patients should avoid alcohol, smoking, and carbonated beverages. Any caloric, vitamin, or mineral deficiencies should be corrected. Good glycemic control can also improve symptoms. If these interventions are not enough, then a prokinetic drug (typically metoclopramide) is the next step. However, caution should be exercised and patients starting metoclopramide need to discontinue therapy if they develop extrapyramidal side effects such as tardive dyskinesia and dystonia and be counseled on these symptoms. Alternatives include domperidone and erythromycin. Finally, those with refractory or severe disease may consider gastric electrical stimulation using an implanted pacemaker [46]. A recent meta-analysis suggests that gastric electrical stimulation can improve both symptoms and gastric emptying, but further studies are still needed [51].

5.6 Bleeding

When patients present with upper GI bleeding, the most important step is to determine the acuity and severity. The most common symptom prompting a workup is melena (tarry black stools) [52]. Melena or coffee-ground emesis is more consistent with chronic or resolved bleeding, whereas frankly bloody emesis is associated with ongoing moderate to severe bleeding. Frankly bloody stool is more consistent with lower GI bleeding but can be seen in patients with massive upper GI bleeding.

For patients who are hemodynamically stable, the history and physical may suggest a cause for the bleeding. A history of liver disease may suggest variceal bleeding, while a history of smoking, alcohol abuse, or peptic ulcer disease raises concerns about malignancy. Laboratory studies are reasonable but often inconclusive, as patients with acute bleeding will still have a hemoglobin level that is unchanged because the overall concentration in the blood is unchanged. However, microcytic anemia may suggest a chronicity to the disease. A blood urea nitrogen to serum creatinine ratio \geq 36 suggests that the bleeding is occurring in the upper rather than lower GI tract [53].

For most patients, the diagnostic workup begins with upper endoscopy followed by a colonoscopy if needed. In patients without variceal bleeding, the most common finding is an ulcer (33 %) followed by mucosal erosion (19 %), although as many as 17 % of patients will have normal study results [52]. Other causes include

esophagitis, Mallory–Weiss syndrome, angiodysplasia, neoplasms, Cameron's erosions, and Dieulafoy's lesions. Repeat endoscopy may be necessary to make the diagnosis.

In the past, nasogastric lavage has been used as a diagnostic tool prior to upper endoscopy for determining whether there was ongoing bleeding. However, a recent study suggests that the use of nasogastric lavage does not change mortality, length of hospitalization, or the units of blood transfused [54].

If upper endoscopy and colonoscopy are not diagnostic, the next step is wireless capsule endoscopy [55]. Capsule endoscopy is more likely to identify a source of bleeding than push enteroscopy, small bowel radiographic series, and colonoscopy with ileoscopy [56]. In patients under the age of 40 years, small bowel bleeding is typically from neoplasms (e.g., lymphoma, carcinoids, and adenocarcinoma), Meckel's diverticulum, Dieulafoy's lesion, and Crohn's disease [55]. Patients older than 40 years typically bleed from vascular lesions (e.g., angiectasis) or NSAID-related disease. Celiac disease can present at any age.

5.7 Conclusion

Many men will develop upper GI tract symptoms over the course of their lifetime. A careful history and physical examination can narrow the range of possible etiologies. Watch out for alarm symptoms which should prompt further workup. Medical management will be successful for most disorders but patients with refractory or recurrent symptoms should be referred to specialists.

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