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of UWL and is often more pronounced in men. More intense forms of mental illness such as paranoid disorders may lead to delusions about food and cause weight loss. Alcoholism can be a significant source of weight loss and malnutrition.

Elderly persons living in poverty may have to choose whether to purchase food or use the money for other expenses, including medica tions. Screening questions can probe whether patients have run out of food or whether they routinely purchase less than they need. Institu tionalization is an independent risk factor, as up to 30–50% of nursing home patients have inadequate food intake. Medications can cause anorexia, nausea, vomiting, gastrointestinal distress, diarrhea, dry mouth, and changes in taste. This is particularly an issue in the elderly, many of whom take five or more medications. PART 2 Cardinal Manifestations and Presentation of Diseases ■ ■ASSESSMENT The four major manifestations of UWL are (1) anorexia (loss of appetite), (2) sarcopenia (loss of muscle mass), (3) cachexia (a syndrome that combines weight loss, loss of muscle and adipose tissue, anorexia, and weakness), and (4) dehydration. The current obesity epidemic adds complexity, as excess adipose tissue can mask the development of sarcopenia and delay awareness of the development of cachexia. If it is not possible to measure weight directly, a change in clothing size, cor roboration of weight loss by a relative or friend, and a numeric estimate of weight loss provided by the patient are suggestive of true weight loss. Initial assessment includes a comprehensive history and physical, a complete blood count, tests of liver enzyme levels, C-reactive protein, erythrocyte sedimentation rate, renal function studies, thyroid function tests, chest radiography, and an abdominal ultrasound (Table 50-2). Age-, sex-, and risk factor–specific cancer screening tests, such as fecal occult blood, colonoscopy, or mammography, should be performed (Chap. 75). Patients at risk should have HIV testing. All elderly patients with weight loss should undergo screen ing for dementia and depression by using instruments such as the Mini-Mental State Examination and the Geriatric Depression Scale, respectively (Chap. 489). The Mini Nutritional Assessment (www. mna-elderly.com) and the Nutrition Screening Initiative (http://www. ncbi.nlm.nih.gov/pmc/articles/PMC1694757/) are also available for the TABLE 50-2 Assessment and Testing for Involuntary Weight Loss Indications Laboratory 5% weight loss in 6 mo Complete blood count Comprehensive electrolyte and metabolic panel, including liver and renal function tests Body mass index <21 Thyroid function tests 25% of food left uneaten after 7 d Erythrocyte sedimentation rate Change in fit of clothing C-reactive protein Change in appetite, smell, or taste Ferritin Abdominal pain, nausea, vomiting, diarrhea, constipation, dysphagia HIV testing, if indicated Assessment Radiology Complete physical examination, including dental evaluation Chest

x-ray Abdominal ultrasound Medication review Recommended cancer screening Mini-Mental State Examinationa Mini-Nutritional Assessmenta Nutrition Screening Initiativea Simplified Nutritional Assessment Questionnairea Observation of eatinga Activities of daily livinga Instrumental activities of daily livinga aMay be more specific to assess weight loss in the elderly.

nutritional assessment of elderly patients. Almost all patients with a malignancy and >90% of those with other organic diseases have at least one laboratory abnormality. In patients presenting with substan tial UWL, major organic and malignant diseases are unlikely when a baseline evaluation is completely normal. Careful follow-up rather than additional undirected testing is advised because the prognosis of weight loss of undetermined cause is generally favorable. TREATMENT Unintentional Weight Loss The first priority in managing weight loss is to identify and treat the underlying causes. Treatment of underlying metabolic, social, psychiatric, dental, infectious, or other systemic disorders may be sufficient to restore weight and functional status gradually. Medications that cause nausea or anorexia should be withdrawn or changed, if possible. For those with unexplained UWL, oral nutri tional supplements such as high-energy drinks sometimes reverse weight loss. Advising patients to choose appealing foods and to consume supplements between meals rather than with a meal may help minimize appetite suppression and facilitate increased over all intake. Orexigenic, anabolic, and anticytokine agents are not generally recommended. In selected patients, the antidepressant mirtazapine significantly increases body weight, body fat mass, and leptin concentration. However, side effects, including dizziness, fatigue, and somnolence, occur in about 10% of patients. Patients with wasting conditions who can comply with an appropriate exer cise program gain muscle protein mass, strength, and endurance and may be more capable of performing ADLs. ■ ■FURTHER READING Gaddey HL, Holder KK: Unintentional weight loss in older adults. Am Fam Physician 104:34, 2021. McMinn J et al: Investigation and management of unintentional weight loss in older adults. BMJ 342:d1732, 2011. Nicholson BD et al: Prioritising primary care patients with unexpected weight loss for cancer investigation. BMJ 370:m2651, 2020. Perera LAM et al: Approach to patients with unintentional weight loss. Med Clin North Am 105:175, 2021. Wong CJ: Involuntary weight loss. Med Clin North Am 98:625, 2014. Loren Laine

Gastrointestinal

Bleeding Gastrointestinal bleeding (GIB) presents as either overt or occult bleeding. Overt GIB is manifested by hematemesis, vomitus of red blood or “coffee-grounds” material; melena, black, tarry stool; and/or hematochezia, passage of red or maroon blood from the rectum. In the absence of overt bleeding, occult GIB may present with symptoms of blood loss or anemia such as lightheadedness, syncope, angina, or dyspnea; with iron-deficiency anemia; or a positive fecal occult blood test on colorectal cancer screening. GIB is also categorized by the site of bleeding as upper, from the esophagus, stomach, or duodenum; lower, from the colon; small intestinal; or obscure GIB if the source is unclear. GIB is the most common gastrointestinal condition leading to hos pitalization in the United States, accounting for ~530,000 admissions annually and a case fatality of ~2%. Patients generally die from decom pensation of other underlying illnesses rather than exsanguination.

■ ■SOURCES OF GASTROINTESTINAL BLEEDING Upper Gastrointestinal Sources of Bleeding • PEPTIC ULCERS Peptic ulcers are the most common cause of upper GIB (UGIB), accounting for

~50% of UGIB hospitalizations. Features of an ulcer at endoscopy provide important prognostic information that guides subsequent management decisions (Fig. 51-1). Approximately 20% of patients with bleeding ulcers have the highest-risk findings of active bleeding or a nonbleeding visible vessel; one-third of such patients have further bleeding that requires urgent surgery if they are treated conservatively. These patients benefit from endoscopic therapy such as bipolar electrocoagulation, heater probe, injection therapy (e.g., absolute alcohol, 1:10,000 epinephrine), clips, and/or topical hemostatic powder with reductions in bleeding, hospital stay, mortality, and costs. In contrast, patients with clean-based ulcers have rates of serious rebleeding approaching zero. If stable with no other reason for hospitalization, such patients may be discharged home after endoscopy. Randomized controlled trials document that high-dose, proton pump inhibitor (PPI), given to reduce intragastric acid and thereby enhance clot stability, decreases further bleeding and mortality in patients with high-risk ulcers (active bleeding, nonbleeding visible ves sel, adherent clot) when given after endoscopic therapy. Meta-analysis of randomized trials indicates that outcomes are comparable with highdose PPIs given as a constant infusion or intermittently. Patients with lower-risk findings (flat pigmented spot, clean base) do not require endoscopic therapy and receive standard doses of oral PPI. Approximately 10–50% of patients with bleeding ulcers rebleed within the next year if no preventive strategies are employed. Pre vention of recurrent bleeding focuses on the three main factors in ulcer pathogenesis, Helicobacter pylori, nonsteroidal anti-inflammatory drugs (NSAIDs), and acid. Eradication of H. pylori in patients with bleeding ulcers decreases rebleeding rates to <5%. If a bleeding ulcer develops in a patient taking NSAIDs, the NSAIDs should be discon tinued. If NSAIDs must be given, a cyclooxygenase-2 selective NSAID plus a PPI is recommended, based on results of a randomized trial. Patients with established cardiovascular disease who develop bleeding Endoscopic Diagnosis Ulcer Erosions Flat pigmented spot Active bleeding or visible vessel Endoscopic Features Adherent clot May consider endoscopic therapy No endoscopic therapy Endoscopic Therapy Endoscopic therapy Medical Therapy High-dose PPI therapya High-dose PPI therapya Once-daily PPI therapy Once-daily PPI therapy Clear liquids for ~2 days Clear liquids for ~2 days Clear liquids for ~1 day Regular diet Dietc Hospital Stayd Hospitalize 3 days Hospitalize ~1–2 days Discharge after endoscopy Hospitalize 3 days FIGURE 51-1 Suggested algorithm for patients with acute upper gastrointestinal bleeding (hematemesis, melena) based on endoscopic findings. aIntravenous bolus (80 mg) followed by infusion (8 mg/h) or by intermittent oral or intravenous doses (e.g., 40 mg 2–4 times per day) for 3 days. bIntravenous 50 μg bolus followed by 50 μg/h infusion for 2–5 days. cDiet after endoscopy, assuming no nausea or vomiting. dDuration after endoscopy assuming patient stable without further bleeding or concurrent medical conditions requiring hospitalization. PPI, proton pump inhibitor.

ulcers while taking low-dose aspirin for secondary prevention should not discontinue aspirin and, if aspirin is held, should restart aspi rin once hemostasis is confirmed. A randomized trial showed that immediate reinstitution of aspirin was associated with a lower 8-week mortality compared to not restarting aspirin (1% vs 13%; hazard ratio, 0.2; 95% CI, 0.1–0.6). In contrast, aspirin should be discontinued in patients taking aspirin for primary prevention of cardiovascular events who develop UGIB. Patients with bleeding ulcers unrelated to H. pylori or NSAIDs should remain on PPI therapy indefinitely given a 42% inci dence of rebleeding at 7 years without protective therapy. Peptic ulcers are discussed in Chap. 335.

Gastrointestinal Bleeding CHAPTER 51 MALLORY-WEISS TEARS Mallory-Weiss tears account for ~2–10% of UGIB hospitalizations. The classic history is vomiting, retching, or coughing preceding hematemesis. Bleeding from these tears, which are usually on the gastric side of the gastroesophageal junction, stops spontaneously in ~80–90% of patients and recurs in only 0–10%. Endoscopic therapy is indicated for actively bleeding Mallory-Weiss tears. Mallory-Weiss tears are discussed in Chap. 334. ESOPHAGEAL VARICES The proportion of UGIB hospitalizations due to varices varies widely from ~2–40%, depending on the popula tion. Patients with variceal hemorrhage have poorer outcomes than patients with other sources of UGIB. Esophageal varices are treated with endoscopic ligation and an IV vasoactive medication (octreotide, somatostatin, vapreotide, terlipressin) for 2–5 days. Combination of endoscopic and medical therapy is superior to either therapy alone in decreasing rebleeding. Over the long term, treatment with nonselective beta blockers plus endoscopic ligation is recommended because the combination is more effective than either alone in reduction of recur rent esophageal variceal bleeding. Transjugular intrahepatic portosys temic shunt (TIPS) is recommended in patients who have persistent or recurrent bleeding despite endoscopic and medical therapy. TIPS also is suggested for acute variceal bleeding in patients with advanced liver disease (Child-Pugh class B with score 8–9 and active bleeding at endoscopy, Child-Pugh class C with score 10–13), because randomized trials show significant decreases in rebleeding and mortality compared with standard endoscopic and medical therapy. Esophageal Varices Mallory-Weiss Tear Clean base Active bleeding No active bleeding No endoscopic therapy No endoscopic therapy No endoscopic therapy Endoscopic ligation Endoscopic therapy Antiemetic if ongoing nausea Antiemetic if ongoing nausea Vasoactive drug (e.g., octreotideb) + antibiotic (e.g., ceftriaxone) Once-daily PPI therapy Clear liquids for ~2 days Clear liquids for ~1 day Regular diet Regular diet Hospitalize ~3–5 days Hospitalize ~1–2 days Discharge after endoscopy Discharge after endoscopy

Portal hypertension is also responsible for bleeding from gastric varices, varices in the small and large intestine, and portal hypertensive gastropathy and enterocolopathy. Bleeding gastric varices are treated with endoscopic injection of tissue adhesive (e.g., n-butyl cyanoacry late), TIPS, or retrograde transvenous obliteration.

EROSIVE DISEASE Erosions are endoscopically visualized breaks that are confined to the mucosa and do not cause major bleeding because arteries and veins are not present in the mucosa. Erosions in the esophagus, stomach, or duodenum commonly cause mild UGIB, with erosive esophagitis (primarily due to gastroesophageal reflux disease), gastritis, and duodenitis accounting for up to perhaps ~30% of UGIB hospitalizations. The most important cause of gastric and duodenal erosions is NSAID use: up to ~50% of patients who chronically ingest NSAIDs may have gastric erosions. Other potential causes of gastric erosions include alcohol intake, H. pylori infection, and stress-related mucosal injury. PART 2 Cardinal Manifestations and Presentation of Diseases Stress-related gastric mucosal injury occurs only in extremely ill patients, such as those with serious trauma, major surgery, burns cov ering more than one-third of the body surface area, major intracranial disease, or severe medical illness. Severe bleeding should not develop unless ulceration occurs. The mortality rate in these patients is high because of their serious underlying illnesses. The incidence of bleeding from stress-related gastric mucosal injury has decreased dramatically in recent years, most likely due to better care of critically ill patients. Meta-analysis of randomized trials indicates prophylaxis with PPIs reduces clinically important GIB more than no prophylaxis or H2-receptor antagonists without impacting mortality or risk of infection (e.g.,

pneumonia, Clostridioides difficile). A guideline suggested PPI prophylaxis in critically ill patients at high risk (≥4%) of bleeding, defined as mechanical ventilation without enteral nutrition, portal hypertension, cirrhosis, platelets <50 × 109/L, international nor malized ratio >1.5, or two of the following: mechanical ventilation with enteral nutrition, acute kidney injury, sepsis, or shock. OTHER CAUSES Less common causes of UGIB include neoplasms, vascular ectasias (including hereditary hemorrhagic telangiectasias and gastric antral vascular ectasia), Dieulafoy's lesion (in which an aberrant vessel in the mucosa bleeds from a pinpoint mucosal defect), prolapse gastropathy (prolapse of proximal stomach into esophagus with retch ing), aortoenteric fistulas, and hemobilia or hemosuccus pancreaticus (bleeding from the bile duct or pancreatic duct). Small-Intestinal Sources of Bleeding Patients without a source of GIB identified on upper endoscopy and colonoscopy were previ ously labeled as having obscure GIB. With the advent of improved diagnostic modalities, ~75% of GIB previously labeled obscure is now estimated to originate in the small intestine beyond the extent of a standard upper endoscopic exam. Small-intestinal GIB may account for ~5% of GIB cases. The most common causes in adults include vascular ectasias, neoplasm (e.g., gastrointestinal stromal tumor, car cinoid, adenocarcinoma, lymphoma, metastases), and NSAID-induced erosions and ulcers. Meckel's diverticulum is the most common cause of significant small-intestinal GIB in children, decreasing in frequency as a cause of bleeding with age. Other less common causes of smallintestinal GIB include Crohn's disease, infection, ischemia, vasculitis, small-bowel varices, diverticula, intussusception, Dieulafoy's lesions, aortoenteric fistulas, and duplication cysts. Small-intestinal vascular ectasias are treated initially with endo scopic therapy, based on observational studies suggesting short-term efficacy. However, rebleeding is common, with pooled rebleeding rates of ~45% over a mean follow-up of ~2 years in systematic reviews, lead ing guidelines to suggest medical therapy if further bleeding occurs after endoscopic therapy. The best available evidence supports use of thalidomide, with a multicenter double-blind randomized trial show ing marked reductions in bleeding episodes, transfusions, and hospi talizations. Monthly intramuscular injection of octreotide long-acting release also is suggested based on observational studies and a small open-label randomized trial. Other isolated lesions, such as tumors, generally require surgical resection.

Colonic Sources of Bleeding Hemorrhoids are probably the most common cause of lower GIB (LGIB); anal fissures also cause minor bleeding and pain. If these local anal processes, which rarely require hospitalization, are excluded, the most common cause of LGIB in adults is diverticulosis. Other causes include vascular ectasias (especially in the proximal colon of patients >70 years), neoplasms (primarily adenocarcinoma), colitis (ischemic, infectious, Crohn's or ulcerative colitis, NSAID-induced colitis or ulcers), postpolypectomy bleeding, and radiation proctopathy. Rarer causes include solitary rec tal ulcer syndrome, varices (most commonly rectal), lymphoid nodular hyperplasia, vasculitis, trauma, and aortocolic fistulas. In children and adolescents, the most common colonic causes of significant GIB are inflammatory bowel disease and juvenile polyps. Diverticular bleeding is abrupt in onset, usually painless, sometimes massive, and often from the right colon; chronic or occult bleeding is not characteristic. Case series from the United States and Europe suggest colonic diverticula stop bleeding spontaneously in ≥90% of patients, with rebleeding on long-term follow-up as low as ~15% over 4–5 years. Rebleeding is substantially higher in reports from Asia. Case series suggest endoscopic therapy may decrease recurrent bleeding in the uncommon case when colonoscopy identifies the specific bleeding diverticulum. When diverticular bleeding is found at angiography, transcatheter arterial embolization by superselective technique pre vents further bleeding in most patients. Segmental surgical resection

is recommended for refractory diverticular bleeding. Bleeding from colonic vascular ectasias may be overt or occult; it tends to be chronic and only occasionally hemodynamically signifi cant. Endoscopic hemostatic therapy may be used in the treatment of vascular ectasias, as well as discrete bleeding ulcers and postpolyp ectomy bleeding. Transcatheter arterial embolization also may be attempted for persistent bleeding from vascular ectasias and other lesions, although rebleeding is higher in nondiverticular LGIB at ~45%. Surgical therapy is generally required for major persistent or recur rent bleeding from colonic sources that cannot be treated medically, endoscopically, or angiographically. Patients with Heyde's syndrome (bleeding vascular ectasias and aortic stenosis) appear to benefit from aortic valve replacement. APPROACH TO THE PATIENT Gastrointestinal Bleeding INITIAL ASSESSMENT Measurement of the heart rate and blood pressure is the best way to initially assess a patient with GIB. Clinically significant bleeding leads to postural changes in heart rate or blood pressure, tachycar dia, and, finally, recumbent hypotension. In contrast, hemoglobin does not fall immediately with acute GIB, due to proportionate reductions in plasma and red cell volumes ("people bleed whole blood"). Thus, hemoglobin may be normal or only minimally decreased at initial presentation of a severe bleeding episode. As extravascular fluid enters the vascular space to restore volume, the hemoglobin falls, but this process may take up to 72 h. Transfusion is recommended when the hemoglobin drops below 7 g/dL, based on a large randomized trial showing this restrictive transfusion strategy decreases rebleeding and death in acute UGIB compared with a transfusion threshold of 9 g/dL. Patients with slow, chronic GIB may have very low hemoglobin values despite normal blood pressure and heart rate. With the development of iron-deficiency anemia, the mean corpuscular volume is low and red-blood-cell distribution width is increased. DIFFERENTIATION OF UGIB FROM LGIB Hematemesis indicates an UGIB source. Melena indicates blood has been present in the gastrointestinal (GI) tract for ≥14 h and as long as 3–5 days. The more proximal the bleeding site, the more likely melena will occur. Hematochezia usually represents a lower GI source of bleeding, although an upper GI lesion may bleed so briskly that blood transits the bowel before melena develops. When

hematochezia is the presenting symptom of UGIB, it is associated with hemodynamic instability and dropping hemoglobin. Bleeding lesions of the small bowel may present as melena or hematochezia. Other clues to UGIB include hyperactive bowel sounds and elevated blood urea nitrogen (due to volume depletion and blood proteins absorbed in the small intestine). A nonbloody nasogastric aspirate may be seen in ~15% of patients with UGIB who present with clinically serious hemato chezia. A bile-stained appearance does not exclude UGIB because reports of bile in the aspirate are incorrect in ~50% of cases. Testing of aspirates that are not grossly bloody for occult blood is not useful. EVALUATION AND MANAGEMENT OF UGIB (FIG. 51-1) Initial Risk Assessment Baseline characteristics predictive of rebleed ing and death include hemodynamic compromise (tachycardia or hypotension), increasing age, and comorbidities. Risk assessment tools may be used to identify patients with very low risk. Discharge from the emergency room with outpatient management is suggested for patients with a Glasgow-Blatchford score (possible range 0–23, Table 51-1) of 0–1 because only ~1% of patients who require transfusion, require hemostatic intervention, or die have a score of 0–1. Pre-Endoscopic Medications PPI infusion may be considered at presentation; it modestly decreases need for endoscopic therapy due to a reduction in high-risk ulcer stigmata (e.g., active bleeding) but does not improve clinical outcomes such as further bleeding or death. The promotility agent erythromycin, 250 mg intravenously ~30–90 min before endoscopy, is suggested to improve visualization at endoscopy, thereby reducing the need for repeat endoscopy and hospital stay. Cirrhotic patients presenting with UGIB should be given an

antibiotic (e.g., ceftriaxone) and IV vasoactive medication (e.g., octreotide) upon presentation. Antibiotics decrease bacterial infections, rebleeding, and mortality, and vasoactive medications improve control of bleeding in the 12 h after presentation. Endoscopy Upper endoscopy should be performed within 24 h in most patients hospitalized with UGIB whether they have clinical No Hemodynamic Instability Hemodynamic Instability Colonoscopya Site identified, bleeding persists Site not identified Site identified, bleeding stops Angiography Bleeding persists Surgery FIGURE 51-2 Suggested algorithm for patients with acute lower gastrointestinal (GI) bleeding (hematochezia). aFor patients <35 years old with minor bleeding (e.g., blood on toilet paper), normal blood pressure and heart rate, normal hemoglobin and iron panel, and no family history of colorectal cancer, some would suggest flexible sigmoidoscopy is adequate.

TABLE 51-1 Glasgow-Blatchford Score RISK FACTORS AT ADMISSION SCORE Blood urea nitrogen (mg/dL) 18.2 to <22.4

22.4 to <28.0

28.0 to <70.0

≥70.0

Hemoglobin (g/dL) 12.0 to <13.0 (men); 10.0 to <12.0 (women)

 10.0 to <12.0 (men)

<10.0

Systolic blood pressure (mmHg) 100–109

90–99

<90

Heart rate (beats per minute) ≥100

Melena

Syncope

Hepatic disease

Cardiac failure

features predicting low or high risk of further bleeding and death. Even in high-risk patients, more urgent endoscopy (performed within 6–12 h of gastroenterology consultation) does not improve clinical outcomes, and some observational studies suggest increased mortality with endoscopy within 6–12 h in high-risk patients. Early endoscopy in low-risk patients (e.g., hemodynamically stable without severe comorbidities) identifies low-risk findings (e.g., clean-based ulcers, erosions, nonbleeding Mallory-Weiss tears) that allow discharge in ≥40% of patients, thereby reducing

hospital stay Upper endoscopy No upper GI source Able to prep Too unstable to prep No Extravasation CT angiography Able to prep Extravasation Angiography Bleeding persists Instability persists Workup for small intestinal/obscure bleeding site Surgery (with intraoperative endoscopy if site has not been identified)

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