



FIGURE 345-27 Mallory-Weiss tear at the gastroesophageal junction.

unable to swallow water; endoscopy is generally the best initial test in such patients, because endoscopic removal of the obstructing material is usually possible, and the presence of an underlying esophageal pathology can often be determined. Radiographs of the chest and neck should be considered before endoscopy in patients with fever, obstruction for ≥ 24 h, or ingestion of a sharp object, such as a fishbone. Radiographic contrast studies interfere with subsequent endoscopy and are not advisable in most patients with a clinical picture of esophageal obstruction. Sips of a carbonated beverage, sublingual nifedipine or nitrates, or intravenous glucagon may resolve an esophageal food impaction, but in most patients, an underlying web, ring, or stricture is present and endoscopic removal of the obstructing food bolus is necessary.

Gastric Outlet Obstruction Obstruction of the gastric outlet is commonly caused by gastric, duodenal, or pancreatic malignancy or chronic peptic ulceration with stenosis of the pylorus (Fig. 345-32). Patients vomit partially digested food many hours after eating. Gastric decompression with a nasogastric tube and subsequent lavage for removal of retained material is the first step in treatment. The diagnosis can then be confirmed with a saline load test, if desired. Endoscopy is useful for diagnosis and treatment. Patients with benign pyloric stenosis may be treated with endoscopic balloon dilatation of the pylorus, and a course of endoscopic dilatation results in long-term relief of symptoms in about 50% of patients. Malignant gastric outlet obstruction can be relieved with endoscopically placed expandable stents in patients with inoperable malignancy (Fig. 345-33).

Colonic Obstruction and Pseudoobstruction These both present with abdominal distention and discomfort; tympany; and a dilated, air-filled

colon on plain abdominal radiography. The radiographic appearance can be characteristic of a particular condition, such as sigmoid volvulus (Fig. 345-34). Both structural obstruction and pseudoobstruction may lead to colonic perforation if left untreated. Acute colonic pseudoobstruction is a form of colonic ileus that is usually attributable to electrolyte disorders, narcotic and anticholinergic medications, immobility (as after surgery), and retroperitoneal hemorrhage or mass. Multiple causative factors are often present. Colonoscopy, water-soluble contrast enema, or CT may be used to assess for an obstructing lesion and differentiate obstruction from pseudoobstruction. One of these diagnostic studies should be strongly considered if the patient does not have clear risk factors for pseudoobstruction, if radiographs do not show air in the rectum, or if the patient fails to improve when underlying causes of pseudoobstruction have been addressed. The risk of cecal perforation in pseudoobstruction rises when the cecal diameter exceeds 12 cm, and decompression of the colon may be achieved using intravenous neostigmine or via colonoscopic decompression (Fig. 345-35). Most patients should receive a trial of conservative therapy (with correction of electrolyte disorders, removal of offending medications, and increased mobilization) before undergoing an invasive decompressive procedure for colonic pseudoobstruction.

Colonic obstruction is an indication for urgent intervention. In the past, emergent diverting colostomy was usually performed with a subsequent second operation after bowel preparation to treat the underlying cause of obstruction. Colonoscopic placement of an expandable stent is now a widely used alternative that can relieve malignant colonic obstruction without emergency surgery and permit bowel preparation for an elective one-stage operation (Fig. 345-36, see Video 346e-15).

ACUTE BILIARY OBSTRUCTION

The steady, severe pain that occurs when a gallstone acutely obstructs the common bile duct often brings patients to a hospital. The diagnosis of a ductal stone is suspected when the patient is jaundiced or when serum liver tests or pancreatic enzyme levels are elevated; it is confirmed by EUS, magnetic resonance cholangiography (MRCP), or direct cholangiography (performed endoscopically, percutaneously, or during surgery). ERCP is currently the primary means of diagnosing and treating common bile duct stones in most hospitals in the United States (Figs. 345-11 and 345-12).

Bile Duct Imaging Whereas transabdominal ultrasound diagnoses only a minority of bile duct stones, MRCP and EUS are $>90\%$ accurate and have an important role in diagnosis. Examples of these modalities are shown in Fig. 345-37.

If the suspicion for a bile duct stone is high and urgent treatment is required (as in a patient with obstructive jaundice and biliary sepsis), ERCP is the procedure of choice, because it remains the gold standard for diagnosis and allows for immediate treatment (see Video 346e-16). If a persistent bile duct stone is relatively unlikely (as in a patient with gallstone pancreatitis), ERCP may be supplanted by less invasive imaging techniques, such as EUS, MRCP, or intraoperative cholangiography performed during cholecystectomy, sparing patients the risk and discomfort of ERCP.

Ascending Cholangitis Charcot's triad of jaundice, abdominal pain, and fever is present in about 70% of patients with ascending cholangitis and biliary sepsis. These patients are managed initially with fluid resuscitation and intravenous antibiotics. Abdominal ultrasound is often performed to assess for gallbladder stones and bile duct dilation. However, the bile duct may not be dilated early in the course of acute biliary obstruction. Medical management usually improves the patient's clinical status, providing a window of approximately 24 h during which biliary drainage should be established, typically by ERCP. Undue delay



FIGURE 345-28 Gastrointestinal vascular ectasias. **A.** Gastric antral vascular ectasia ("watermelon stomach") characterized by stripes of prominent flat or raised vascular ectasias. **B.** Cecal vascular ectasias. **C.** Radiation-induced vascular ectasias of the rectum in a patient previously treated for prostate cancer.