TABLE 33-2 COMMON SOURCES OF ACUTE GASTROINTESTINAL HEMORRHAGE

SOURCE	ASSOCIATED CLINICAL FEATURES	TREATMENTS
UPPER GASTROINTESTINAL TRACT		
Esophagitis	Heartburn, dysphagia, odynophagia	Medication* Antireflux surgery or procedures
Esophageal cancer	Progressive dysphagia, weight loss	Chemoradiotherapy, surgery Palliative endoscopy procedures
Gastritis, gastric ulcer	Aspirin, NSAID use	Withdraw NSAIDs
Duodenitis, duodenal ulcer	Abdominal pain, dyspepsia, *Helicobacter pylori* infection	Medication† Endoscopic therapy for acute bleeding
Gastric cancer	Early satiety, weight loss, abdominal pain	Surgery, chemotherapy
Esophagogastric varices	History of CLD Stigmata of CLD on examination	Variceal banding, sclerotherapy Vasopressin, octreotide TIPS or decompressive surgery
Mallory-Weiss tear	History of retching before hematemesis	Supportive (usually self-limited) Endoscopic therapy
LOWER GASTROINTESTINAL TRACT		
Infection	History of exposure, diarrhea, fever	Supportive, antibiotics
Inflammatory bowel diseases	History of colitis, diarrhea, abdominal pain, fever	Steroids, 5-ASA, immunotherapy Surgery if unresponsive to medication
Diverticula	Painless hematochezia	Supportive Surgery for recurrent disease
Angiodysplasia	Painless hematochezia Often in ascending colon Commonly involves stomach and small bowel as well	Endoscopic therapy Supportive Surgery for localized disease
Colon cancer	Change in bowel habit, anemia, weight loss	Surgery
Colon polyp	Usually asymptomatic	Endoscopic or surgical removal
Ischemic colitis	Typically elderly patients History of vascular disease May produce abdominal pain	Supportive (self-limited)
Meckel's diverticulum	Painless hematochezia in young patient Located at distal ileum	Surgery
Hemorrhoids	Rectal bleeding associated with bowel movement	Supportive Surgery, banding

CLD, Chronic liver disease; NSAIDs, nonsteroidal anti-inflammatory drugs; TIPS, transjugular intrahepatic shunt; 5-ASA, 5-aminosalicylic acid compounds.

*Proton pump inhibitors or histamine-2 receptor antagonists.

†Proton pump inhibitors or histamine-2 receptor antagonists in the absence of *H. pylori* infection; various combinations of antibiotics, proton pump inhibitors, and bismuth products in the presence of *H. pylori* infection.

4. Other associated symptoms, including fever, urgency or tenesmus, recent change in bowel habits, and weight loss
5. Current or recent medication use, particularly nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin, which may predispose to ulceration or gastritis (see Chapter 37), anticoagulants, and alcohol. Many over-the-counter products may contain aspirin or NSAIDs.
6. Relevant past medical and surgical history, including a history of prior GI bleeding, abdominal surgery (prior abdominal aorta repair should raise suspicion for an aortoenteric fistula), radiation therapy (radiation proctitis), major organ diseases (including cardiopulmonary, hepatic, or renal disease), inflammatory bowel diseases, and recent polypectomy (post-polypectomy bleeding).

The physical examination must include an assessment of vital signs, cardiac and pulmonary examinations, and abdominal and digital rectal examinations. The initial laboratory examination should include a complete blood cell count, blood typing and cross-matching, and measurements of serum electrolytes, blood urea nitrogen, creatinine, and coagulation factors. The first hematocrit measurement may not reflect the degree of blood loss, but it will decrease gradually to a stable level over 24 to 48 hours.

The initial disposition of the patient must also be considered. Patients older than 60 years of age, those with severe blood loss or continued bleeding (as reflected by a significant decrease in hematocrit or postural changes in blood pressure or pulse rate), and those with significant comorbid illness are at the greatest risk for complications of GI hemorrhage and are best managed in an intensive care setting until stabilized.

Identification of the Bleeding Source

In 80% to 90% of cases, acute GI hemorrhage resolves spontaneously without recurrence. Nevertheless, it is prudent to localize the bleeding source, especially in those with significant bleeding or comorbidities. Proper identification allows for direct treatment if the bleeding does not spontaneously resolve and for recognition of those patient who are at risk for further bleeding. For example, in a patient with a bleeding gastric or duodenal ulcer, acid suppression with an intravenous proton pump inhibitor may maximize clot stability and enhance platelet aggregation. Proton pump inhibitors, in combination with appropriate endoscopic management, decrease the risks for ulcer rebleeding, need for urgent surgery, and death. Direct visualization of the bleeding site by endoscopy can alter patient management.

Classification systems, such as the Forrest Ulcer Description or the Rockall Scoring System, rely heavily on endoscopic criteria for rebleeding risk stratification. Various stigmata of hemorrhage may be identified within the ulcer crater. Stigmata that carry a high risk for rebleeding include active bleeding (Forrest 1) and the visible presence of a pigmented protuberance (artery) within