41
Lower Gastrointestinal Hemorrhage

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Key Concepts

- Common etiologies of lower gastrointestinal hemorrhage include diverticular disease, angioectasia, ischemic colitis, and neoplasm.
- The primary consideration in managing the patient with acute lower gastrointestinal hemorrhage is ensuring adequate volume resuscitation.
- Patients presenting with massive lower gastrointestinal bleeding should be evaluated for upper gastrointestinal and anorectal sources via gastric lavage and anoscopy/proctoscopy.
- Screening for active bleeding via CT angiography or ^{99m}Tc-RBC scan increases the likelihood of identifying active bleeding on mesenteric angiography.
- An active bleeding source seen on mesenteric angiography can often be managed with superselective transcatheter embolization.
- The patient with a self-limited major lower gastrointestinal hemorrhage that has stopped should undergo colonoscopy for further evaluation after a mechanical bowel prep.
- In certain circumstances, colonoscopy for the evaluation of active lower gastrointestinal bleeding may be considered; if active bleeding is encountered, therapeutic options include clipping, injection, and argon plasma coagulation.
- The unstable patient with uncontrolled, unlocalized lower gastrointestinal hemorrhage should undergo a total abdominal colectomy, in most cases with an ileostomy.
- The patient with ongoing or recurrent hemorrhage from a localized lower gastrointestinal source may be managed with a targeted, segmental resection.
- Clinical pathways and predictive models may help better guide the management of patients with acute lower gastrointestinal hemorrhage, limiting unnecessary admissions and optimizing the use of resources.

Introduction

Lower gastrointestinal bleeding (LGIB) refers to the passage of visible blood from the rectum and classically originates from a source distal to the ligament of Treitz. This distressing condition challenges both the clinician and patient, as LGIB may potentially arise from anywhere along a large anatomic distribution, may result from an array of pathologic conditions, can vary widely in severity, and frequently stops spontaneously prior to definitive diagnosis. In fact, no definitive source is found in approximately 10% of all cases of LGIB [1–3].

Descriptions reported by patients and witnesses can offer a spectrum of qualifiers in regard to the volume, color, associated symptoms, and hemodynamic consequences. The patient and family often experience significant stress and emotion by the sight of any significant quantity of blood passing from the rectum and likely experience an understandable sense of urgency to seek rapid medical evaluation and treatment. Thus, it is not uncommon for patients to present to emergency departments with less serious degrees of rectal bleeding. In fact, a report from an urban medical center reviewed over 1100 patients admitted for LGIB and found that over 20% of their hospitalized patients ultimately were identified to have a diagnosis of hemorrhoids [4]. The financial burden of LGIB per hospitalization ranges from $9700 to $11,800 [5, 6]. Clinicians bear the burden of determining which cases represent potentially life-threatening bleeding that mandates hospitalization and utilization of critical and costly resources.

The purpose of this chapter is to review the scope of the problem of LGIB, to identify underlying causes and their clinical presentation, and to help surgeons and other clinicians develop a rational approach to the diagnosis and treatment of patients experiencing LGIB.

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Epidemiology

LGIB represents a broad clinical entity of varied severity and etiologies, and obtaining accurate epidemiologic data represents a formidable endeavor. Outpatient office visits provide one measure of the prevalence of LGIB, with over 1.7 million office visits in the United States for rectal bleeding occurring in 2009 [7]. Hospitalization data presumably reflects more serious LGIB, and in a review of a large hospital administrative database, admissions for all gastrointestinal bleeding [upper gastrointestinal bleeding (UGIB) and LGIB] were found to occur in approximately 97 cases per 100,000 persons, with 60.6 cases/100,000 persons due to UGIB and 35.7 cases/100,000 persons for LGIB [8]. This report assessed trends from 2001 to 2009 and revealed that the incidence of UGIB dramatically decreased (78.4–60.6 cases/100,000 persons), while cases for LGIB decreased as well but to a lesser extent (41.8–35.7 cases/100,000 persons). A study sponsored by the Agency for Healthcare Research and Quality queried the Nationwide Inpatient Sample Database and also noted a decreasing trend in the incidence of UGI bleeding (decreased by 14%), while during the time period of 1998–2006, the incidence of LGIB actually increased by 8% [9]. In this study, LGIB attributable to diverticulosis decreased by 7%, while anorectal hemorrhage increased by 41%. In Spain, a study involving ten academic hospitals between 1996 and 2005 found a similar decrease in UGIB (87/100,000–47/100,000), while LGIB increased from 20/100,000 to 33/100,000 [10]. Lastly, a recent prospective and population-based study from Iceland reported the highest incidence of LGIB at 87/100,000 persons, which equaled the incidence of UGIB [2].

While LGIB affects both the young and the old, the incidence of LGIB increases dramatically with age. Laine et al. reported that the incidence of LGIB for patients age <65 was 9.8/100,000, while individuals age >65 were found to have an incidence of 127.7/100,000 [8]. This phenomenon is likely explained by the simple fact that many of the conditions responsible for LGIB, such as diverticulosis coli and angioectasia, increase in incidence with age. Gender differences have not been consistently found in studies with regard to LGIB, and a recent survey of the Nationwide Inpatient Sample Database did not identify any difference in diverticular bleeding among men and women [11]. However, in this study, race was examined and found to be a significant factor with African Americans experiencing a higher prevalence of diverticular bleeding (34.4/100,000 persons) than Caucasians (20.3/100,000). Racial demographic data have not been consistently reported among most of the large database epidemiologic studies [7, 8, 12].

Etiologies of LGIB

Benign Anorectal Causes: Hemorrhoidal Bleeding and Fissures

Hemorrhoids and anal fissures commonly are associated with the appearance of bright red blood with bowel movements. The latter is commonly differentiated by the presence of pain during and after evacuation. Blood may be reported on the toilet paper with wiping, on the stool, or in the toilet bowel itself. In some cases, patients with bleeding internal hemorrhoids describe dripping of blood, or even streaming of blood, into the toilet bowl. Hemorrhoidal bleeding accounts for a substantial number of hospitalizations, representing 5–20% of all admissions for LGIB [1, 2, 4, 12]. Chronic bleeding from hemorrhoids over time also may result in iron deficiency anemia [13]. Hemorrhoids and anal fissures are generally not a likely cause of massive lower GI hemorrhage, although persistent bleeding hemorrhoids may require urgent operative intervention on occasion [4].

Diverticulosis Coli

Diverticulosis coli represents an acquired outpouching of the mucosa through the muscular layers of the colonic wall adjacent to penetrating vessels, the vasa recta. Diverticulosis increases with age; roughly 60% of individuals will develop diverticula by the age of 80, although it is estimated that perhaps only 15% will develop actual bleeding as a complication [14, 15].

Though the majority of patients with diverticulosis are not likely to experience clinically significant bleeding, diverticulosis is generally felt to represent the most common cause of LGIB not of anorectal etiology, accounting for 30–65% of cases [2, 4, 14–17]. In terms of severe hemorrhage, diverticular bleeding is certainly recognized as the most likely etiology [18, 19]. The theory behind diverticular bleeding describes the erosion of vasa recta through the mucosa at the neck or at the dome of the diverticulum [20]. Risk factors that predispose to diverticular bleeding include the use of nonsteroidal anti-inflammatory drugs (NSAIDs), hypertension, and anticoagulant use [1, 19, 21].

The diagnosis of a diverticular bleed is often considered presumptive, noting the presence of diverticulosis on colonoscopy without any other definitive bleeding site. Colonoscopy will provide definitive confirmation in the minority of cases (22%); criteria for diagnosis include colonoscopic identification of active bleeding or stigmata of bleeding such as an adherent clot or visible vessel [1]. Most diverticular bleeds present with painless hematochezia, which is often significant in volume. The natural history of

these episodes generally indicates spontaneous cessation in up to 80% of cases [1, 18]. The incidence of recurrent bleeding varies and has been noted to be as high as 40% in one series, though more recent series indicate rates on the order of 10–15% [22–24].

Angioectasia

Angioectasias [also known as angiodysplasia, arteriovenous malformations (AVMs), and vascular ectasias] are dilated, tortuous vascular abnormalities of the submucosa (Figure 41-1). The most widely accepted theory proposes that, with aging, low-grade obstruction of the submucosal veins traversing the colonic muscular layers results in incompetency of the precapillary sphincters, producing a small arteriovenous communication and subsequent dilation [25]. Colonic lesions more commonly occur in the cecum and right side of the colon, tend to be multiple, and are estimated to be the underlying etiology of bleeding in 3–15% of LGIB episodes [26–28].

The clinical presentation of LGIB due to angioectasia varies, and the color of blood has been reported to range from occult blood to melena to painless hematochezia [29]. Historically, angiodysplasia has been characterized by chronic or recurrent LGIB [30–32]. Factors that predispose to bleeding include increased age, comorbid conditions, multiple lesions, and the use of antiplatelet and anticoagulant therapy [33]. Recurrent bleeding is associated with multiple lesions, anticoagulation and antiplatelet therapy, the number of prior bleeding episodes, and rate of bleeding (events/year) [25].

In regards to recurrent bleeding, one must consider that angioectasias tend to be multiple and often involve proximal regions of the intestinal tract that require investigations in addition to colonoscopy. A recent report (which included diagnostic studies of the small bowel such as capsule endoscopy and double balloon enteroscopy) identified angioectasias most commonly in the jejunum (80%), followed by the duodenum (51%), stomach (22.8%), right colon (11.4%), and ileum (5.4%); nearly two-thirds of patients had lesions in multiple locations [2].

Ischemic Colitis

Ischemic injury of the colon occurs as a result of compromised blood flow and may be responsible for up to 16% of cases of LGIB, although most series indicate the incidence to be in the range of 10% [1, 3, 34, 35]. Bleeding typically occurs as a result of reperfusion of an ischemic segment of bowel, with sloughing of the mucosa and varying degrees of ulceration and necrosis (Figure 41-2). Bleeding generally is less severe when compared to diverticular bleeding or that related to angioectasias and, in some cases, may not be part of the clinical presentation at all. There is a spectrum of scenarios that may fall under the category of ischemic injury. Clinically, ischemic injury of the colon may be broadly considered as two distinct entities: (1) the traditional concept of "ischemic colitis" which affects primarily the left colon and is notable for transient and rapidly reversible ischemia and (2) other variants of "colonic ischemia" (CI) which may be due to arterial occlusion, thromboembolic disease, venous occlusion due to mesenteric venous thrombosis, or severe hypotension with a resultant low-flow state, also called nonocclusive mesenteric ischemia (NOMI). The mechanism of interrupted blood supply, the anatomic distribution at risk, and the prognosis vary between the two entities. The latter forms are more typically associated with severe, irreversible ischemic injury, greater risk of necrosis, increased risk of surgical resection, and mortality [36].

"Ischemic colitis" generally refers to a less severe ischemic intestinal injury that tends to be transient and revers-

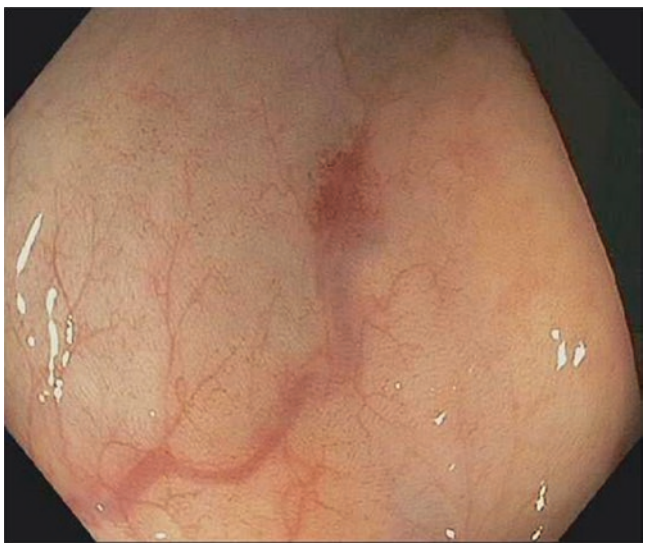

FIGURE 41-1. Angioectasia, seen on colonoscopy.

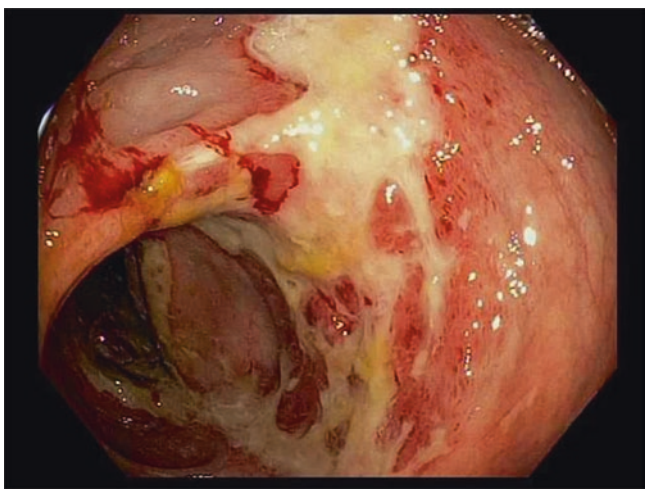

FIGURE 41-2. Ischemic colitis, seen on colonoscopy.

ible. Certain segments of the hindgut appear more vulnerable to this transient interruption of blood flow. These segments classically are referred to as the "watershed" regions: (1) the splenic flexure (Griffith's point), where vessels originating from midgut (superior mesenteric artery distribution) and hindgut (inferior mesenteric artery distribution) communicate via the marginal artery of Drummond, and (2) the rectosigmoid colon (Sudeck's point), where the marginal artery generally is not present and the arterial blood supply is provided by end sigmoidal vessels with less collateral redundancy [37].

Patients with ischemic colitis often present with cramping, abdominal pain, and associated tenderness localized to the left side of the abdomen; they may also experience associated nausea, vomiting, and an urgency to defecate. Typically, patients describe diarrheal stools that become bloody within 24 h of onset and can be either bright red or maroon-colored. Generally, bleeding from ischemic colitis is less severe and blood transfusion is necessary in fewer than 5% of patients. Symptoms generally resolve quickly (within 2–3 days) due to rapid restoration of blood flow, and acute complications requiring surgical intervention occur rarely.

Conversely, patients experiencing acute mesenteric vascular occlusion due to thromboembolism or mesenteric venous thrombosis, or those suffering from profound hypotension requiring vasopressor therapy (NOMI), are at greater risk for severe and irreversible ischemia, bowel necrosis, and need for urgent surgical intervention. Patterns of ischemia tend to be either pancolonic or isolated right colonic ischemia (IRCI) and are more likely to be associated with small bowel ischemia and infarction. Outcomes following surgical resection are remarkable for high mortality rates, ranging from 37 to 47% [38, 39].

Neoplasms of the Large Intestine

As with diverticulosis and vascular ectasias, the incidence of colorectal cancer increases with age [40]. Bleeding from neoplastic lesions of the large intestine generally presents according to anatomic location, with cecal and right-sided lesions more likely to cause occult blood loss, whereas left-sided and rectal lesions tend to present with visible blood per rectum. Acute massive hematochezia due to ulceration of the tumor is rare in the setting of colorectal cancer, and colorectal cancer represents less than 10% of all cases of LGIB requiring hospitalization [2, 4].

Additional Causes of LGIB

The definition of LGIB describes a large anatomic region with multiple additional conditions that may potentially give rise to bleeding, and these are briefly discussed in this section.

Post-polypectomy hemorrhage occurs after less than 1% of colonoscopic polypectomies [41]. However, given the vast numbers of colonoscopies and polypectomies performed annually (1.7 million colon cancer screening colonoscopies in the United States), this may account for up to 8% of all episodes of LGIB [42].

Inflammatory bowel disease (IBD) commonly presents with LGIB. However, severe, massive hemorrhage as the primary symptom prompting hospitalization occurs infrequently and accounts for less than 6% of all patients with a diagnosis of either Crohn's disease or ulcerative colitis [43–45]. Crohn's disease involving the colon and rectum is more likely to be a cause of LGIB as compared with isolated small bowel disease. While IBD more commonly affects the young patient, one must keep in mind the bimodal age distribution of IBD, which should be considered as a potential etiology of LGIB in the older patient as well.

Nonsteroidal anti-inflammatory drugs (NSAIDs) increase the risk of LGIB, especially in patients with diverticular disease. Remarkably, the prevalence of NSAID use among patients experiencing LGIB remains high, reported to be 86% in one series [46]. The association of NSAID use and LGIB may be the result of a specific effect of the medication on the mucosa or alternatively may exacerbate an underlying condition such as diverticulosis. In regard to the former, NSAIDs can cause a type of colitis that can be confused with IBD, characterized by ulcerations and weblike strictures, afflicting primarily the terminal ileum and right colon. Clinically, this can present with massive LGIB and even perforation [47].

Infectious hemorrhagic colitides due to bacterial infection must be considered in the individual experiencing LGIB. Inflammatory diarrhea is characterized by bloody and mucopurulent stool that is often associated with fever, tenesmus, and severe abdominal pain [48]. Common pathogenic bacteria causing inflammatory diarrhea include *Campylobacter*, *Salmonella*, *Shigella*, enteroinvasive and enterohemorrhagic *Escherichia coli*, and *Yersinia* species [49]. In North America, the most common clinically significant strain is *E. coli* O157:H7 [50]. These bacteria naturally occur in the intestines of healthy cattle. Transmission to humans occurs by eating undercooked ground beef or by drinking unpasteurized milk or juice. Consuming food or water contaminated with cow manure or raw ground beef can also lead to infection. The disease can be transmitted from person to person, notably from a child's diapers to their caregivers; additionally, low levels of chlorine in wading/swimming pools can predispose to infection [51, 52]. The infection causes mucosal injury with resulting bloody diarrhea, which is generally self-limited, requiring only supportive care. The most severe consequence of infection is hemolytic-uremic syndrome, characterized by thrombocytopenic thrombotic purpura and renal failure.

HIV-positive patients may experience LGIB from a variety of potential causes. Viral infections may be due to herpes simplex virus, cytomegalovirus, and HIV-related idiopathic proctocolitis. Additionally, one must also consider Kaposi's sarcoma. Sexually transmitted pathogens causing bloody diarrhea include *Chlamydial* species, with *C. trachomatis* infection being the most prevalent among homosexual males. *Neisseria gonorrhoeae* also can produce hemorrhagic proctitis, presenting with bloody mucoid diarrhea [49].

Radiation injury to the large intestine can be either acute (<3 months) or chronic. The incidence of chronic proctitis after pelvic irradiation is approximately 5–20% [53]. Chronic injury results in endarteritis obliterans that leads to neovascularization and telangiectasias, most commonly in the rectum. The incidence of radiation proctitis appears more likely if acute toxicity was observed during the course of treatment [54, 55]. The most common symptom of chronic radiation-induced proctitis remains rectal bleeding, while other associated symptoms include fecal urgency, incontinence, rectal pain, and mucoid discharge. More severe consequences include stricture and necrosis, resulting in potential fistulization to the urethra [56]. Endoscopy represents the diagnostic test of choice for evaluating radiation-induced proctocolitis and should be performed to exclude the possibility of an associated neoplasm of the large intestine, which can be seen in up to 12% of cases [57]. Typical endoscopic findings include telangiectasias, edema, ulceration, necrosis, and stenosis. Biopsy should be performed judiciously due to the known risk of non-healing ulcers and development of fistulae to the urethra and/or bladder [58].

Ulceration of the rectum has been described as a source of LGIB that can be severe and unrelenting, often requiring urgent colonoscopy and intervention. These ulcerations can result from stercoral injury or de novo in acutely ill patients.

Dieulafoy's lesions, most commonly found in the stomach, may be located elsewhere in the gastrointestinal tract 30% of the time, including the colon, the rectum, and the small intestine. These lesions represent a rare cause of GI bleeding (1–2%), though it has been suggested that this may underrepresent the true incidence due to a lack of recognition of the entity [59], as they have been identified via endoscopy and colonoscopy with increasing incidence in recent decades. Characteristic endoscopic findings describe a solitary vessel, histologically normal but large in diameter, protruding through the mucosa without surrounding ulceration [60]. Bleeding can be violent and voluminous and lead to life-threatening hemorrhage. The cause of Dieulafoy's lesions remains uncertain, and the range of clinical presentation includes reports occurring in acutely ill hospitalized patients as well as in newborn infants [59]. Approximately 5% of Dieulafoy's lesions are estimated to occur in the colon and rectum, with the right colon being the most common location [60].

Ectopic varices represent another rare but sinister cause of LGIB. Ectopic (non-esophageal) varices may occur in up to 70% of patients with portal hypertension and cirrhosis. Colorectal varices are well described but fortunately not a common cause of hemorrhage. Rectal varices result from portosystemic shunting and decompression of the inferior mesenteric vein and superior rectal veins via the middle and inferior rectal veins. Rectal varices do not prolapse, tend to be blue-gray in color, and may extend from the rectum superiorly to the squamous epithelium of the anus distally, in distinction to internal hemorrhoids, which may prolapse, tend to be purple in color, and generally do not extend proximally into the rectum [61]. The vessels tend to be serpentine in morphology, submucosal, and extend from the squamous epithelium cranially [62]. Bleeding from varices results from wall tension that is proportional to transmural pressure and the radius of the vessel. Similar to esophageal varices, the major determining factors are vessel size and portal venous pressure [63]. Fortunately, bleeding from rectal varices is rare, occurring in 0.5–3.6% of all cases [64–67]. However, when bleeding occurs, hemorrhage can be massive and life-threatening, requiring urgent intervention. Optimization of medical management remains paramount, including consideration of decompression procedures such as transjugular intrahepatic portosystemic shunt (TIPSS) [68]. Endoscopic techniques, such as injection sclerotherapy, as well as interventional radiology techniques, such as embolization, have been reported to be effective. While band ligation has been described as a treatment modality for esophageal varices, rectal varices less commonly are amenable to ligation technique, with variceal size (>9 mm) being an important predictor of poorer outcome [69].

Obscure bleeding from a small intestinal source has been estimated to account for 5% of LGIB episodes. Previously an anatomic territory that proved difficult to image endoscopically, the small bowel can now be directly visualized using techniques such as device-assisted enteroscopy (balloon and double balloon enteroscopes) and video capsule endoscopy. Such technologies prove far more sensitive than contrast studies or computed tomography and can identify many of the varied diagnoses causing bleeding, such as angioectasias, ulcerations, small bowel tumors, and IBD. In younger patients with LGIB, one must always consider Meckel's diverticulum, especially when bleeding is acute and massive [70]. Radionuclide imaging identifying ectopic gastric mucosa assists in confirming this diagnosis.

Table 41-1 summarizes the distribution of causes of LGIB, as reported in a number of large epidemiologic studies.

TABLE 41-1. Lower gastrointestinal bleeding—distribution of etiologies

Author (Year)	Diverticulosis (%)	Hemorrhoids (%)	Neoplasm (%)	Angioectasia (%)	Ischemic colitis (%)	IBD (%)	Colitides (%)	Ulcers (%)	Post-polypectomy (%)	Small bowel (%)	Radiation (%)	Other (%)	Unknown (%)
Longstreth 1997 [1]	41.6	4.6	9.1	2.7	8.7	2.3	5.0					10	11.9
Strate 2003 [3]	30	12	6	3	10	4	8		7		3	7	9
Velayos 2004 [72]	30	12	6	4		4		4	2	6			11
Strate (NIS) 2008 [12]	33.1	20	21.3	6.0	6.6	4.4							11
Gayer 2009 [4]	37.3	21	11.8	2.3		5.4	10.7 (includes ischemic)			8		6.58	3.45
Hreinsson 2012 [2]	23.3	10.4	10.5	3.1	16	11.7				3.1		11	9.2

Models Predicting Severity of LGIB

Patients presenting with LGIB represent a considerable challenge to healthcare teams and hospital systems, given the heterogeneous nature of causes, spectrum of severity, and often elusive nature with spontaneous cessation of bleeding prior to definitive diagnosis. While occasionally dramatic in presentation, the vast majority of patients with LGIB do not require surgical intervention and experience exceedingly low mortality. Ideally, healthcare teams could better serve patients by employing a model to predict which episodes require hospitalization and help direct rational use of diagnostic testing and intervention. Validated models predicting severity and guiding management for UGIB exist and are widely employed [71]. However, models for predicting LGIB behavior have been much more difficult to develop and validate, given the heterogeneity of the clinical syndrome and the complexity of its clinical presentation.

Velayos et al. prospectively evaluated parameters identified within the first hour of presentation with LGIB to an emergency department and attempted to identify risk factors for adverse outcomes. A total of 448 patients were prospectively followed, and multivariate regression analysis identified three independent risk factors for severe LGIB: initial hematocrit less than 35%, presence of abnormal vital signs 1 h after initial medical evaluation, and gross blood on initial rectal examination. Severe LGIB occurred in 79% of patients with three risk factors, 57% of patients with two risk factors, 17% of patients with one risk factor, and zero patients with no risk factors [72].

Strate et al. also sought also to identify risk factors for severity of LGIB and predict which patients would most benefit from aggressive care and intervention. Multivariable logistic regression analysis of a cohort of 252 patients identified seven independent risk factors for severe LGIB: initial heart rate greater than 100/min, initial systolic blood pressure less than 115 mmHg, syncope, non-tender abdomen, bleeding per rectum during the first 4 h of evaluation, aspirin use, and Charlson Comorbidity Index score of more than 2. Severe LGIB was seen in 84% of patients with more than three risk factors, 43% of patients with one to three risk factors, and 9% of patients with no risk factors [3].

As a follow-up, Strate et al. then prospectively validated their predictive model in a cohort of 275 patients, noting that the number of positive risk factors, calculated within 4 h of presentation, significantly correlated with major clinical outcomes, including surgery, death, blood transfusions, and length of hospital stay. They concluded that the triage of high-risk patients (three or more risk factors) to urgent interventions could be used to improve utilization of resources and quality of care [34]. It should be noted that this model made the assumption that hemorrhage due to ischemic colitis and IBD is generally mild to moderate, so LGIB due to these etiologies was not included in the study group.

Patel et al. prospectively applied an algorithm to the evaluation of patients presenting with uncomplicated rectal bleeding. If the patient's hemoglobin was >13 g/dl, SBP> 115 mmHg, and the patient was not anticoagulated, the patient was discharged with plans for an outpatient flexible sigmoidoscopy within 6 weeks. This algorithm was applied to a series of 57 patients, and potential inpatient admissions were avoided in 35%. Only one discharged patient was readmitted with severe colitis, and avoidable admissions were reduced from 50 to 1.8% [73].

Although predictive models such as these have been developed and validated, it is unclear as to what extent their implementation will impact clinical practice and improve patient outcomes. Certainly, the application of practical and predictive clinical models for the evaluation and management of LGIB will become more relevant to physicians in the future, given the economic and administrative pressures on healthcare systems to demonstrate appropriate resource utilization and cost reduction efforts.

Presentation, Evaluation, and Management

Due to the diversity in underlying etiologies, the presentation of LGIB can range from occult bleeding to life-threatening hemorrhage. Of paramount importance is rapid assessment of the patient's hemodynamic stability. Patients presenting with massive gastrointestinal bleeding and signs of hemodynamic instability, chest pain, shortness of breath, or orthostatic hypotension should immediately have two large-bore intravenous lines placed and undergo rapid volume resuscitation with crystalloid while awaiting labs and availability of cross-matched blood; in extreme circumstances, one may consider transfusion with non cross-matched type O negative blood. Continuous monitoring of vital signs is essential, and a Foley catheter should be placed to monitor urine output.

Placement of a nasogastric tube and gastric lavage is essential. Aspiration of frank blood, clot, or coffee grounds should prompt further investigation for UGIB via upper endoscopy. A bilious aspirate all but excludes an upper gastrointestinal source, while a clear aspirate is indeterminate, as there could be source of bleeding distal to a contacted pylorus.

A thorough history and physical examination should be performed, including an intake of the patient's medications, paying particular attention to NSAIDs, anticoagulants, and antiplatelet agents that may exacerbate bleeding, as well as beta-blockers that may mask the physiologic response to hypovolemia. Pertinent points in the history should include onset and duration of bleeding, volume and frequency of bleeding, color of blood (bright red, maroon, or tarry), and presence or absence of clots. A history of abdominal pain and weight loss may suggest IBD, ischemia, or malignancy, though colorectal cancer rarely presents with massive

hematochezia. The presence of significant pain represents a branch point in the evaluation of the patient with LGIB and should prompt earlier cross-sectional imaging if the patient is hemodynamically stable. Other salient questioning should focus on comorbidities such as cardiovascular, pulmonary, or hepatic disease, the presence or absence of chest pain, shortness of breath, lightheadedness, anorectal pain, and the date and findings of the patient's most recent colonoscopy and/or upper endoscopy. Particular attention should be paid to those who have undergone prior intestinal surgery due to the possibility of an anastomotic ulcer and those who have been previously treated with abdominopelvic radiation, implicating radiation proctitis/colitis/enteritis.

Physical examination should begin with assessment for signs/symptoms of hypovolemic shock. Once the patient's volume status has been assessed and appropriate resuscitation has been initiated, a more focused physical exam should ensue. Abdominal examination should focus on the presence of pain, palpable masses, distention, scars from prior surgeries, and hepatosplenomegaly. Stigmata of chronic liver disease, such as jaundice, caput medusa, or palmar erythema, may suggest variceal bleeding. Visual inspection of the perineum should be performed to evaluate for thrombosed or prolapsing hemorrhoids, anal fissure, or anal masses. Digital rectal examination should be done to assess for the presence of a rectal mass, and anoscopy and/or rigid proctosigmoidoscopy should be performed to evaluate for a distal source of bleeding, such as internal hemorrhoids, proctitis, ulcers, or varices.

Laboratory studies should include a basic chemistry panel, complete blood count, coagulation parameters, and type and cross. Coagulopathies should be corrected via transfusion of blood products and/or factors as appropriate. Patients with cardiovascular disease or those with chest pain or shortness of breath should undergo an electrocardiogram, and if abnormal, cardiac enzymes should be assessed.

Initial volume resuscitation of the hypovolemic patient should include bolus infusion of isotonic crystalloid, such as normal saline or lactated Ringer's solution, aiming to restore normotension. Continued hypotension despite aggressive crystalloid infusion should prompt transfusion of packed red blood cells. Further transfusion should be guided by the patient's hemodynamic response and change in hemoglobin. A hemoglobin transfusion threshold of 9–10 g/dL has traditionally been employed, especially in patients with significant cardiovascular disease. While data regarding a more restrictive pattern of transfusion specifically in patients with LGIB is lacking, a number of studies in patients with UGIB have demonstrated improved outcomes using a more restrictive threshold, as low as 7 g/dL, in low-risk patients [74–76]. For patients requiring transfusion of multiple units of PRBC, concurrent administration of platelets and fresh frozen plasma may prevent dilutional coagulopathy.

Colonoscopy

When patients present with a self-limited LGIB, colonoscopy is the diagnostic modality of choice, identifying either a definitive or presumed source of bleeding in 74–100% of cases [77–83]. The major advantage of colonoscopy is the potential for concurrent diagnosis and therapeutic intervention, even in the absence of active bleeding. Because most bleeding stops spontaneously, colonoscopy is typically performed semi-electively, usually following a mechanical bowel preparation. However, the optimal means of bowel preparation and timing of colonoscopy is often a topic of debate. While the use of a mechanical bowel purge allows for more complete visualization of the colonic mucosa, it also necessitates a delay in performing the procedure. If bleeding has stopped by the time colonoscopy is performed, it is often difficult to know which, if any, of identified abnormalities was responsible, especially when multiple sources, such as diverticula or AVMs, are identified.

A pooled analysis of studies looking at colonoscopy after mechanical bowel preparation for the evaluation of LGIB found a diagnostic yield of 91% [25]. Early colonoscopy has been found to correlate with a shorter length of admission, primarily due to increased yield. Strate and Syngal reported that time to colonoscopy was an independent predictor of length of hospital stay in patients presenting with hematochezia. The absence of visible blood or active bleeding at the time of colonoscopy was also related to a shorted length of stay [84]. A number of other studies have suggested that urgent colonoscopy within 12–24 h of presentation can improve the diagnostic yield [79, 80, 85, 86].

In contrast to "early" colonoscopy is the concept of "urgent" colonoscopy, usually performed within a few hours of the patient's arrival. Jensen et al. studied the role of urgent colonoscopy (within 6–12 h of hospitalization) in patients with hematochezia and known diverticulosis after a 3–4 h mechanical colon purge in two sequential prospective trials. In the first trial, 23% of patients were found to have definitive signs of diverticular bleeding during colonoscopy and were managed medically, not endoscopically. Nearly half of these patients experienced rebleeding, and two-thirds of those that rebled required emergency hemicolectomy. In the second trial, 21% of patients had signs of diverticular hemorrhage at the time of colonoscopy, half of which were found to have active visible bleeding. All patients with signs of diverticular hemorrhage were treated endoscopically, and none had recurrent bleeding or required surgery [79].

Others have reported conflicting data regarding improved outcomes with urgent colonoscopy. Green et al. randomized patients with lower GI bleeding to urgent colonoscopy after a raid purge or a standard care algorithm based on angiographic intervention and expectant colonoscopy. They reported no differences in main outcome measures, including mortality, hospital stay, ICU stay, transfusion requirements, early and late rebleeding, and need for surgery [80]. Laine et al. performed

a prospective randomized trial comparing urgent colonoscopy (within 12 h of presentation) to elective colonoscopy (36–60 h after presentation). Though the trial closed prematurely due to inadequate enrollment and statistical power, the urgent group showed no decrease in diagnostic or therapeutic interventions, number of transfusions, length of stay, or hospital charges [87]. A number of other studies have also reported data indicating that urgent colonoscopy for acute LGIB may not be advantageous [78, 88].

In an effort to minimize the delay needed for mechanical bowel preparation, some have proposed the use of enemas to clear the left colon, though oral bowel preparation has been shown to clearly increase the diagnostic rate for colonoscopy compared with enemas alone [89]. In some instances, rapid enemas can be used prior to immediate colonoscopy after the onset of massive hematochezia to simply help distinguish a right-sided from a left-sided source; if blood is only seen in the left colon, further diagnostics should focus there [90]. Often, an acute bleed will clear the colon distal to the bleed of any solid stool, though there may be significant clot to clear during the procedure. Repaka et al. published a prospective feasibility study looking at urgent colonoscopy without oral bowel preparation aided by water jet pumps and mechanical suction devices (hydroflush colonoscopy). In a series of 13 procedures, a presumed or definitive source of bleeding was seen in all patients, a defined source was identified in 5/13 (38.5%), and endoscopic hemostasis was achieved in four of these. Complete colonoscopy to the cecum was performed in 9/13 (69.2%), and no patients required a repeat colonoscopy due to inadequate preparation [91].

In addition to the increased diagnostic yield, another advantage of early or urgent colonoscopy is the potential for therapeutic intervention if a source is identified. Colonoscopic interventions for cessation of active bleeding include clipping, band ligation, injection of epinephrine or saline, monopolar or bipolar electrocautery, laser coagulation, or argon plasma coagulation (APC). Bleeding diverticula can be treated by submucosal injection of epinephrine (diluted 1:20,000 in saline) in 1-mL aliquots into four quadrants around the base of the diverticulum. This can be done either with or without application of a heater probe applied at a low power setting for 1–2 s, though this does increase the risk of perforation. Early rebleeding rates for injection range from 0 to 35%, with minimal procedure-related complication [79, 80, 92].

Endoscopic clips can also be applied, either alone or as an adjunct to injection. A number of case series and retrospective studies have described successful endoscopic clipping for the management of LGIB of diverticular origin with rebleeding rates ranging from 0 to 21% [93]. Endoscopic clipping of the base of the diverticulum has been reported to have comparable success rates and complication profiles when compared with epinephrine injection [94–96]. Clipping of diverticula located in the right colon, as opposed to the left colon, has been reported to be a predictor of refractory hemorrhage [97].

Alternatively, endoscopic band ligation has been described in the management of diverticular hemorrhage with excellent success rates and low rebleeding rates [98, 99]. Setoyama et al. compared patients treated with endoclips and those treated with EBL and found initial success rates of 100% in both groups; however, rebleeding was seen in only 6% of patients treated with EBL, compared with 33% of those treated with clips [100].

AVMs can be treated either with electrocautery, APC, or laser coagulation. Multiple sessions may be required, and long-term rebleeding rates range from 10 to 39% [32, 101]. A combination of APC and endoscopic clipping has also been reported [102]. The risk of complications, including perforation, ranges from 2 to 7%. One must keep in mind that AVMs in particular are more often located in the thinner-walled right colon, increasing the risk of perforation with any intervention.

A novel means of obtaining endoscopic hemostasis for diffuse bleeding diatheses in the colon, such as radiation- or NSAID-induced colitis, has been described by Kratt et al. who reported the use of Hemospray (Cook Medical, Bloomington, IN), a mineral-based granular powder that absorbs water and induces the clotting cascade. They reported introducing it via a colonoscope throughout the cecum and ascending colon in an elderly patient with NSAID-induced colonopathy, successfully ceasing hemorrhage and avoiding the need for an urgent colectomy [103].

Radionucleotide Scintigraphy

Nuclear scintigraphy has long been utilized as a means of detecting active GI bleeding. Two techniques can be employed for the detection of active GI bleeding—^{99m}Tc-sulfur colloid and ^{99m}Tc-labeled RBCs, the latter of which has been shown to be superior for the detection of GI bleeding [104, 105]. ^{99m}Tc-RBC scanning requires labeling a small sample of the patient's blood with technetium and then injecting it back into the patient's bloodstream, followed by scintigraphic scanning. Active hemorrhage is indicated by extravasation and pooling of the radionucleotide tracer (Figure 41-3).

The procedure is not invasive, carries little risk, and does not require mechanical bowel preparation. Other benefits include its high sensitivity and the slow washout of the tracer, which allows repeat scanning over periods of up to 24 h in instances of intermittent bleeding. This is important to take into consideration, given that rebleeding can be seen in up to 27% of patients after an initial negative ^{99m}Tc-RBC scan [106]. The main drawbacks are that this technique requires some prep time to extract and tag the RBCs (approximately 30 min), and there is no possibility for therapeutic intervention.

Detection of bleeding as slow as 0.04–0.05 cm^3/min has been reported with ^{99m}Tc-RBC scans [107, 108]. The sensitivity

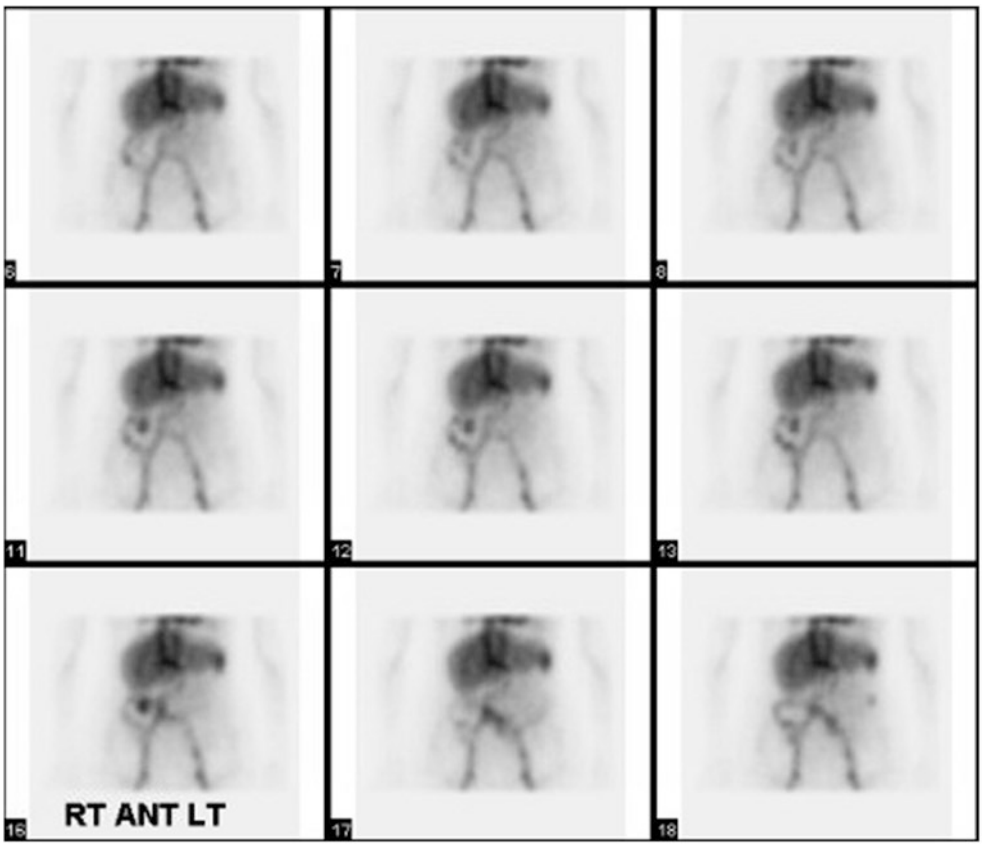

FIGURE 41-3. ^{99m}Tc-RBC scan showing active extravasation in the right lower quadrant. RBC = red blood cell.

is linked to the volume of extravasated RBC at the site of bleeding. While detection of low rates of bleeding is possible, hyperperistalsis may distribute the labeled RBCs over a substantial length of bowel, reducing the sensitivity. Feingold et al. found that patients who were hemodynamically unstable at presentation were more likely to have a positive ^{99m}Tc-RBC scan than those who were hemodynamically stable (62% vs. 21%) [109]. Reported accuracy in detection of the anatomic site of bleeding varies widely (41–94%) [110, 111], mainly because of rapid movement of tracer within the lumen of the bowel due to peristalsis and gravity, as well as difficulty discriminating colon from overlying small bowel. Because of the variability in accurate localization of the anatomic site of bleeding, most algorithms that include ^{99m}Tc-RBC scanning in the evaluation of patients with LGIB use it as a screening study prior to proceeding with mesenteric angiogram rather than as a localizing study. In a retrospective review of 271 angiograms published by Gunderman et al., the use of screening ^{99m}Tc-RBC scans prior to mesenteric angiography improved the diagnostic yield from 22 to 53% [112].

Because of the lack of reliability in determining the actual anatomic site of bleeding, segmental resection based on ^{99m}Tc-RBC scan localization alone is generally not advocated. However, a number of studies have attempted to refute this dogmatic approach. Suzman et al. retrospectively evaluated patients with LGIB who underwent a preoperative ^{99m}Tc-RBC scan and ultimately required surgery; 97.3% had correct localization based on surgical pathology, and only one of 50 patients over the 5-year period of the study required subtotal colectomy because of nonlocalized bleeding [113]. In a similar study, Gutierrez et al. reported an 88% accuracy of ^{99m}Tc-RBC scans in determining the site of bleeding [114]. Despite these reports, the decision to perform a segmental resection based on ^{99m}Tc-RBC scan localization alone should be made only after careful consideration.

Computed Tomography Angiography

Recent advances in computed tomography have led to the development and validation of CT angiography (CTA) techniques. Sixty-four-row CTA allows thinner collimation, faster scanning times, greater anatomic coverage, and better multiplanar reformatted images, greatly expanding its diagnostic role for the evaluation of LGIB [115]. With its widespread availability, CTA has largely supplanted ^{99m}Tc-RBC scanning as the initial means of evaluating most patients presenting with acute LGIB who do not have a contraindication such as renal insufficiency or allergy to contrast dye. Besides the detection of active bleeding, CTA has the added

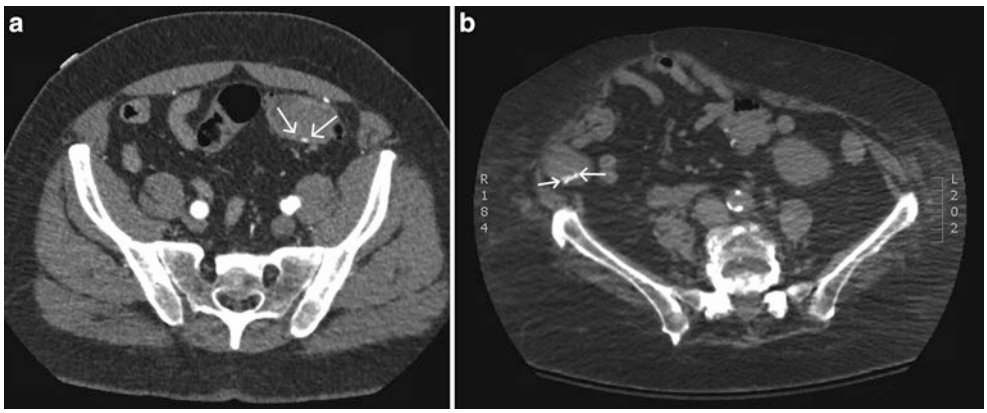

FIGURE 41-4. (**a**) CT angiogram showing active extravasation in the sigmoid colon. (**b**) CT angiogram showing active extravasation in the cecum.

advantages of being able to localize the site of bleeding and identify any coexisting pathology. A positive CTA (Figure 41-4) should prompt further therapeutic efforts, such as angiographic embolization, or if the patient is showing signs of massive hemorrhage, targeted surgical resection of the culprit segment of intestine.

The rate of bleeding able to be detected by CTA has been reported to be as low as 0.3 mL/min [116]. The sensitivity of CTA for localization of a LGIB source is 91–92% when active bleeding is present, though it drops to as low as 45–47% when bleeding is intermittent [117]. In a prospective trial, Ren et al. found that CTA had an accuracy of 90.5% in the detection of active GI bleeding, and treatment planning was correctly established on the basis of CTA findings with an accuracy of 98.4%. Another prospective study comparing the diagnostic performance of CTA with angiography, colonoscopy, and surgical findings reported a sensitivity of 100%, specificity of 96%, positive predictive value (PPV) of 95%, negative predictive value (NPV) of 100%, and accuracy of 93% [118].

A prospective trial published by Obana et al. found that the detection rate of colonic diverticular bleeding by CTA alone was only 15.4%, but jumped to 46.2% when combined with colonoscopy [119]. Nagata and colleagues evaluated rates of detection of a LGIB source comparing early colonoscopy following urgent CTA with early colonoscopy alone and found that the detection rate was higher with colonoscopy following CTA than with colonoscopy alone for vascular lesions (35.7% vs. 20.6%, $p=0.01$), leading to more endoscopic therapies (34.9% vs. 13.4%, $p<0.01$) [120].

A major advantage of CTA in the evaluation of the patient with LGIB is its ready availability and ease with which the study can be performed and rapidly interpreted, leading to earlier and more targeted therapeutic intervention. It is a noninvasive study that does not require mechanical bowel preparation and carries very little risk. The main disadvantage is the small risk of contrast nephropathy, which may limit its use in patients with renal insufficiency.

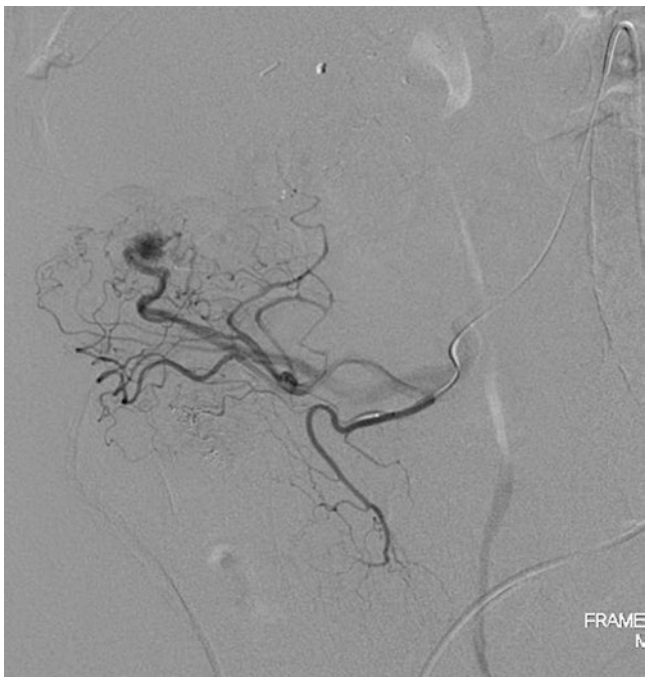

FIGURE 41-5. Mesenteric angiogram showing active extravasation from a branch of the ileocolic artery.

Angiography

Diagnostic Angiography

Diagnostic mesenteric angiography was first reported in the evaluation of hematochezia in 1963 [121]. Despite being an invasive procedure with a number of potential complications, its major advantage is the ability to perform a therapeutic intervention if active bleeding is identified (Figure 41-5). Angiography requires a more rapid rate of bleeding (0.5–1.5 cm^3/min) than nuclear scintigraphy to detect active extravasation. Identification of active bleeding following a positive "screening" ^{99m}Tc-RBC scan or CTA may be hampered by the intermittent nature of most LGIBs and the time

delay between the positive scan and the performance of angiography.

Ng et al. studied the timing of extravasation in a ^{99m}Tc-RBC scan and its role in predicting a positive mesenteric angiogram. They found that an immediate blush had a 75% PPV for active extravasation found on angiography, whereas a delayed (>2 min) blush had an NPV of 93%, suggesting that patients with a delayed blush may not require diagnostic angiography and may instead be observed and evaluated with colonoscopy after a bowel prep [122].

The diagnostic yield of mesenteric angiography depends on patient selection, the timing of the procedure, and the skill of the angiographer [123, 124]. A review by of the literature by Vernava et al. [77] reported diagnostic yields ranging from 40 to 86%. Abbas et al. found that angiography successfully localized bleeding sites in 51% of LGIB episodes; positive localization correlated with hemodynamic instability on arrival, a drop in hemoglobin level ≥50% from previous admission, and a transfusion requirement of ≥5 U of PRBC within 24 h [125].

Tan et al. reviewed patients with LGIB who had a positive CTA followed by mesenteric angiography and found that factors associated with a positive mesenteric angiography included nondiverticular etiology and hemoglobin <10 g/dL; when mesenteric angiography was performed within <150 min of the CTA, it was 2.89 times more likely to identify an active bleeding source [126]. A similar study by Koh et al. also found that mesenteric angiography after a positive CTA was 8.56 times more likely to be positive when performed within 90 min of the CTA [127]. Rasuli et al. reported that older age, ICU admission, and having received 4 U of PRBC over 12 h or 5 U over 24 h were indicators of a positive mesenteric angiogram [128].

For patients with recurrent intermittent LGIB that continue to escape localization despite multiple diagnostic studies, one technique that is often discussed but rarely utilized is provocative angiography, which incorporates the use of heparin, thrombolytics, vasodilators, or some combination of these to induce a bleed that has ceased. Before considering provocative angiography, one must balance the risk of uncontrolled hemorrhage or intracranial hemorrhage against the potential diagnostic and therapeutic benefit. Small series have been reported with low complication rates and diagnostic yields ranging from 29 to 38% [129–131].

The risks associated with diagnostic mesenteric angiography include bleeding, access complications such as vascular injury and pseudoaneurysm, thromboembolic events, and contrast-induced nephropathy. Contraindications include contrast dye allergy and renal insufficiency that might limit the ability to administer intravenous contrast.

Therapeutic Angiography

If a blush or area of obvious extravasation is seen during diagnostic angiography, therapeutic intervention should be attempted. In the 1970s, the technique of vasopressin infusion via a selectively placed mesenteric arterial catheter to induce vasospasm was introduced, and fairly recently, this was the preferred means of intervention. Transcatheter infusion generally begins at 0.2 U/min and may be increased to 0.4 U/min if bleeding persists. Cessation of active bleeding is seen in up to 90% of patients, though the rate of rebleeding upon discontinuation of the infusion approaches 50% [132]. Because of the antidiuretic effect of vasopressin, there is a tendency toward fluid retention and congestive heart failure, so its use in patients with significant cardiac disease becomes somewhat limited, especially considering the significant volume resuscitation many patients with LGIB require.

Vasopressin infusion was previously favored over intravascular embolization due to the high rates of intestinal ischemia and perforation (in as many as 20% of cases) reported with the use of larger catheters, which only allowed for embolization of larger vessels. However, the availability of "microcatheters" now allows for transcatheter superselective embolization of much smaller target vessels with a negligible risk of intestinal ischemia. Success rates with cessation of active arterial bleeding range from 50 to 100% with rebleeding rates of 22–24% [133–136]. Complications such as transmural ischemia and stricture formation, which were more common in the past following embolization of larger segmental vessels, now occur rarely with the use of superselective embolization angiography and are usually asymptomatic. Due to its efficacy and low risk of complications, superselective embolization is now considered by most to be the first-line angiographic therapy for LGIB. Materials used for embolization include microcoils, polyvinyl alcohol particles, and gelfoam.

Tan et al. published a retrospective review of 265 patients undergoing mesenteric angiography for LGIB, of which 32 (12%) underwent superselective embolization. Immediate cessation of bleeding was seen in 31 (97%), though only 20 (63%) were subsequently discharged with no further interventions. Seven patients rebled, and a total of nine required surgery; postembolization ischemia was seen in only one patient (3%). Rebleeding was more likely to occur if the bleeding source was the small bowel or if the presenting hematocrit was <20.0% or platelet count was <140; surgical resection was more likely if the underlying etiology of bleeding was diverticular disease or if the presenting hematocrit was <20.0% [134].

Compared with nuclear scintigraphy as a "screening" test for LGIB, pre-angiography localization of hemorrhage site by CTA has been shown to be more precise and consistent with angiographic findings. Pre-angiography CTA followed by therapeutic angiography typically results in administration of similar cumulative volumes of intravenous contrast when compared to angiography preceded by ^{99m}Tc-RBC, presumably due to pre-angiographic localization of the anatomic site of bleeding. The use of CTA prior to mesenteric angiography has been shown to have no effect on the incidence of contrast-induced nephropathy, given the similar volumes of contrast administration [137].

Localization of Small Bowel Bleeding

When a patient shows signs of ongoing GI bleeding in the face of negative evaluations of both the upper and lower GI tracts, one should consider evaluation for a small bowel source of bleeding. Options include video capsule endoscopy (VCE), double balloon enteroscopy (DBE), radionucleotide Meckel's scan, and, as a last resort, intraoperative push enteroscopy.

VCE and DBE have both been shown to have diagnostic yields in range of 55–65% in patients with hematochezia [117]. One disadvantage of VCE is failure to pass the capsule in instances in which structuring or obstructive disease is present, necessitating further intervention for retrieval. A dissolvable test capsule can be ingested prior to performing the study in an attempt to avoid this. Another disadvantage is that of missed lesions. As VCE generally records 2 frames/s, there have been reports of missed lesions subsequently seen on DBE [138]. Leung et al. randomized patients presenting with GI bleeding and nondiagnostic upper/lower endoscopy to undergo further evaluation via either VCE or angiography and found a higher diagnostic yield with VCE (53.3% vs. 20.0%, $p=0.016$) with no differences in long-term outcomes, including further transfusion, rebleeding, and mortality [139]. Newer devices are able to capture as many as 35 frames/s, which should greatly enhance the diagnostic accuracy. VCE takes significant time to perform and is most commonly utilized to evaluate for an occult source of chronic bleeding, not in the presence of massive LGIB.

DBE is a technically challenging and time-consuming procedure that should only be attempted by a skilled endoscopist who has training and significant experience with the technique. Despite these limitations, DBE compared to VCE has the added benefit of both localization and potential therapeutic intervention if bleeding source is identified. If a site of bleeding is identified but endoscopic intervention is not possible, the endoscopist can mark the site of bleeding with endoclips or tattooing for later identification at the time of possible radiologic or surgical intervention. Similar to VCE, the role of DBE in the setting of acute, massive LGIB is somewhat limited. However, Mönkemüller et al. have reported a series of 17 emergency DBEs for overt obscure GIB in which they successfully identified a source of bleeding in 59% [140].

A Meckel's scan relies on uptake of ^{99m}Tc-pertechnetate in ectopic gastric mucosa within a Meckel's diverticulum that has the potential for GI hemorrhage (but not active GI hemorrhage, for which ^{99m}Tc-RBC scanning would be more appropriate). The procedure is noninvasive, has minimal morbidity, and has both specificity and PPV approaching 100%, though its sensitivity is much lower at 62% [141–143]. Concurrent administration of H-2 blockers has been shown to increase the diagnostic yield.

Surgery

Despite the frequency with which patients present for evaluation of LGIB, the number of patients who require emergency surgery without a preoperatively localized site of bleeding is less than 5% [4]. However, in hemodynamically unstable patients with ongoing LGIB unresponsive to initial resuscitative efforts, emergent surgical intervention is indicated. Also, patients in whom a source of bleeding has been localized but therapeutic efforts are either unsuccessful or not feasible should be considered surgical candidates, as should those with massive transfusion requirements. Six units of PRBC in a 24-h period has traditionally been considered the threshold trigger prompting surgical intervention, though this varies depending on institution and the clinical state of the patient. Bender et al. reported a 45% mortality rate for patients undergoing emergency surgery for LGIB when a total of ten or more units of PRBCs were transfused preoperatively, compared with 7% when less than ten units were transfused [144].

When ongoing LGIB hemorrhage is present and a source cannot be localized despite multiple diagnostic studies or if the patient is too unstable for additional diagnostic studies, the patient should undergo exploratory laparotomy. The small bowel should be thoroughly examined to exclude a Meckel's diverticulum or a palpable mass that could be a source of bleeding. Transillumination of the small bowel may reveal small tumors or angiodysplasia. If the patient is stable, an intraoperative colonoscopy can be performed with luminal lavage and irrigation of sequential segments with proximal compression of the colon. Intraoperative push enteroscopy can also be considered if a colonic source is not identified and there is bright red blood and/or clots in the terminal ileum, though this can be technically challenging and time-consuming.

If a clear source cannot be identified and there is no obvious source in the stomach or small bowel (and an anorectal source has been excluded), the bleeding source is presumed to be colonic. In this scenario, and in the face of ongoing hemodynamic instability or ongoing frank hemorrhage, a total abdominal colectomy should be performed with either an end ileostomy or, in select circumstances, an ileoproctostomy. Generally speaking, most would advocate avoiding an anastomosis, given the indication for emergent laparotomy. If the patient is unstable or on vasopressors, has required multiple transfusions, or is markedly hypoalbuminemic, an end ileostomy is usually the safer option, as it eliminates the risk of anastomotic leak. Furthermore, ongoing bleeding from a source proximal to the colon can be identified quickly and more easily identified endoscopically via an ileoscopy. An ileoproctostomy, while an acceptable choice in properly selected patients, carries with it the risk of anastomotic leak, which can have devastating consequences, especially given that most patients who require emergency surgery for LGIB have numerous preexisting comorbidities; many of these patients may require ongoing use of vasopressors in the immediate post-op period, further compromising the anastomosis. Plummer et al. found a mortality rate of 17% in patients undergoing emergency surgery for unlocalized

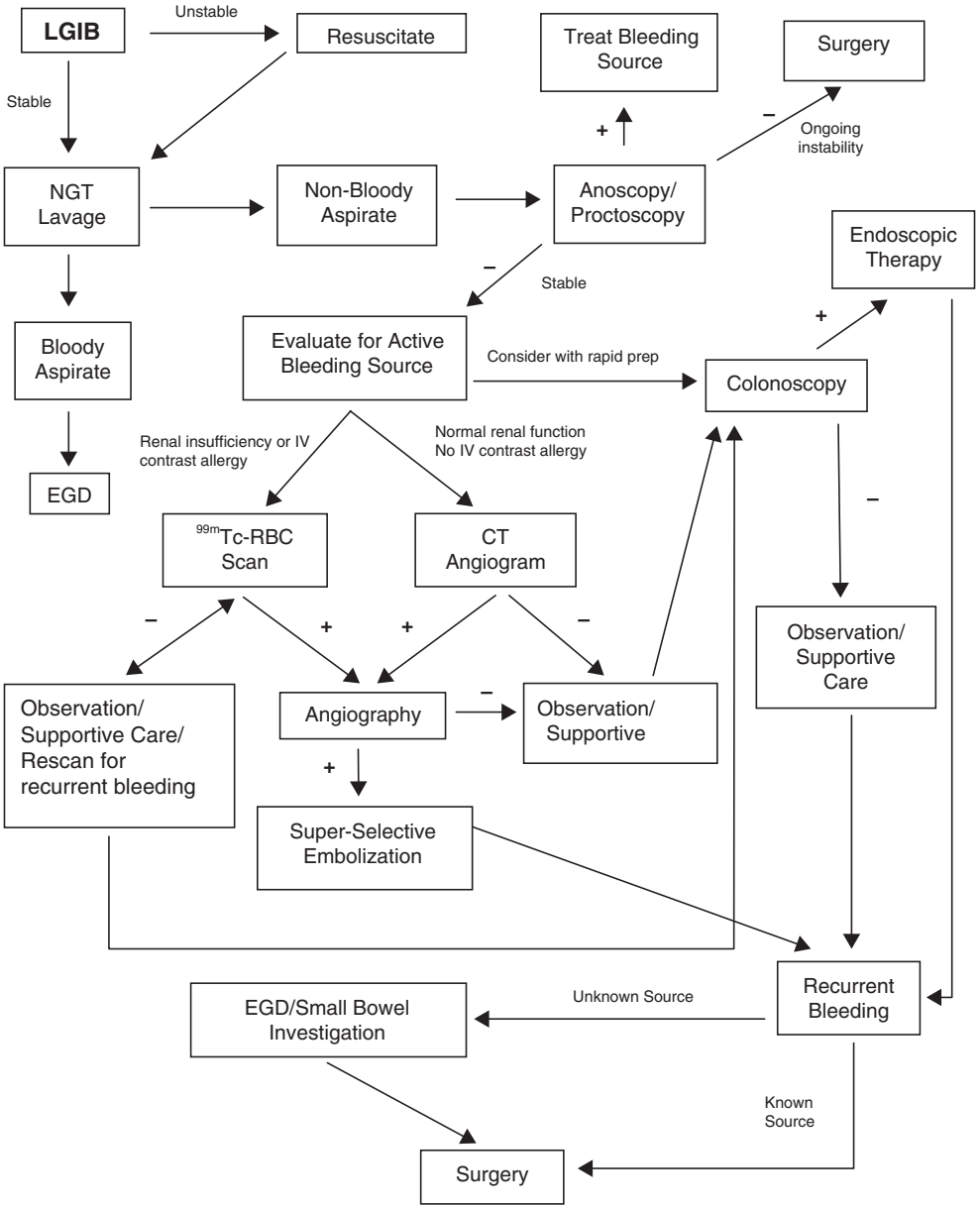

FIGURE 41-6. Algorithm for the evaluation and management of the patient with LGIB (lower gastrointestinal bleeding).

LGIB, with the main contributor being sepsis from an anastomotic leak [145]. Rebleeding rates after total abdominal colectomy generally are less than 5%, and modern advances in postoperative ICU care have reduced postoperative mortality to 2–6% [146–151].

When the bleeding site has been localized but endoscopic or angiographic attempts to control it have failed, a targeted segmental resection is indicated, either with primary anastomosis or a stoma dictated by the patient's clinical condition at the time of surgery. In this scenario, rebleeding rates and mortality are 4–10% and 0–40%, respectively [77, 147–149]. Compared with subtotal colectomy and ileorectal anastomosis, segmental colectomy provides measurable improvements in postoperative morbidity, BM frequency, social restrictions, and overall quality of life [152].

A "blind" segmental resection without preoperative localization should not be performed. The previously held tenet that the majority of GI bleeds are left-sided is no longer felt to be accurate. Blind segmental resections have been shown to have mortality rates ranging from 30 to 57% with rebleeding rates of 33–75% [148, 151, 153, 154].

Summary

LGIB is a commonly encountered condition, with a number of possible etiologies and several options for evaluation. Key points in the management include restoring hemodynamic stability, identifying and localizing ongoing bleeding, and cessation of hemorrhage, either by radiographic or

surgical means. There are a number of options in the evaluation of patients with LGIB, which should be individualized to the experience of the evaluating physician and available resources.

New technologies such as CTA and earlier use of colonoscopy now allow for more rapid and accurate detection of active bleeding; the number of nondiagnostic invasive angiograms has diminished, and pre-angiographic localization of an active bleeding site via CTA helps to facilitate treatment via therapeutic angiography. Advances in interventional techniques and use of microcatheters have improved the efficacy of therapeutic angiography and reduced the risk of postembolic ischemia. Fewer patients are undergoing emergency surgery for nonlocalized bleeding, even fewer are requiring subtotal colectomy for nonlocalized LGIB, and those who do require surgery fare much better than in the past due to improvement in postoperative ICU care.

However, despite the advances and new technologies, LGIB can still present significant diagnostic and therapeutic challenges to the treating physician. Employing a well-defined strategy in the evaluation and management of the patient can help to minimize unnecessary hospital admissions and make the best use of healthcare resources. An algorithm summarizing the evaluation and management of the patient presenting with LGIB is presented in Figure 41-6.

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