ARTICLE IN PRESS
Disease-a-Month ▮ (2018) ▮▮▮–▮▮▮

Contents lists available at ScienceDirect

Disease-a-Month

journal homepage: www.elsevier.com/locate/disamonth

Evaluation and management of lower gastrointestinal bleeding

Aun Raza Shah, MD[a], Vikram Jala, MD[b], Hammad Arshad, MD[c], Mohammad Bilal, MD[b,*]

[a]Darent Valley Hospital, Dartford, United Kingdom
[b]Division of Gastroenterology & Hepatology, University of Texas Medical Branch, 7400 Jones Drive, Apt 724, Galveston, TX 77551
[c]Summit Health Services, Chambersburg, PA

ARTICLE INFO

Keywords:
Lower gastrointestinal bleeding
Hematochezia
Diverticulosis
Diverticular bleeding

ABSTRACT

Lower gastrointestinal bleeding (LGIB) is a common cause of presentation to the emergency department and hospital admissions. The incidence of LGIB increases with age and the most common etiologies are diverticulosis, angiodysplasia, malignancy and anorectal diseases. Foremost modality for evaluation and treatment of LGIB is colonosopy. Other diagnostic tools such as nuclear scintigraphy, computed tomography, angiography and capsule endoscopy are also frequently used in the workup of LGIB. Choice of treatment modality depends on the hemodynamic status of the patient, rate of bleeding, expertise and available resources. We present a comprehensive review of the evaluation and management of LGIB.
© 2018 Elsevier Inc. All rights reserved.

Introduction

Lower gastrointestinal bleeding (LGIB) has been historically differentiated from upper gastrointestinal bleeding (UGIB) as any bleed originating distal to the ligament of Treitz. Clinical

Disclaimers: none.
Sources of support/funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.
*Corresponding author. Tel.: +1 412 660 3624; fax: +1 409 772 3394.
E-mail address: billa17@hotmail.com (M. Bilal).

https://doi.org/10.1016/j.disamonth.2018.02.002
0011-5029/© 2018 Elsevier Inc. All rights reserved.

presentations can vary from minor hematochezia to massive hemorrhage with fatal outcomes. LGIB continues to be a common cause of morbidity and mortality in hospitalized patients, especially amongst the elderly.[1,2] Incidence of LGIB ranges from 20 to 30 per 100,000, surmounting to almost 30% of all episodes of gastrointestinal hemorrhage.[1–3] This incidence continues to rise and is postulated to be even higher than reported as a large proportion of patients do not seek medical attention.[3,4]

Endoscopy, radionuclide scintigraphy, and angiography are the mainstays for evaluation of LGIB. However numerous advancements have been made in recent times to improve diagnostic capabilities including capsule endoscopy, double-balloon enteroscopy, and computed tomographic angiography (CTA). On the other hand, endoscopic interventions, endovascular embolization, and surgery continue to be the cornerstone for treatment. Advancements in endoscopic techniques, interventional angiography and minimally invasive procedures have reduced the need for surgery.[5,6] The development of these modalities has also resulted in less healthcare expenditures as well as improved patient outcomes.[4,7,8]

Mortality rates for LGIB vary.[9] Patients requiring transfusion of greater than 5 units of packed cells or surgery or those with multiple co-morbid conditions or multi-organ system disease are at higher risk for increased morbidity and mortality. Age >60 years, hemodynamic instability, low hematocrit ($<35\%$) and elevated serum creatinine levels have been identified as risk factors for increased morbidity and mortality.[9] However, with aggressive supportive care 75–85% of LGIB will resolve without any complications.[6,10]

Pathophysiology

LGIB can occur due to a wide variety of causes. The most common causes of LGIB include diverticular bleeding, anorectal disorders including hemorrhoids, colorectal malignancies, inflammatory bowel disease, arteriovenous malformations, colitis (ischemic, radiation, and infectious) and iatrogenic (Table).[11]

Retrospective reviews of hospital admission data have consistently showed diverticulosis to be the most common cause of LGIB in adults, followed by anorectal disease, colitis, carcinoma and arteriovenous malformations.[12,13] Gayer et al. evaluated the etiologies of 1112 patients hospitalized with LGIB, and found diverticulosis (33.5%) as the most common cause hospitalizations due to LGIB, followed by hemorrhoids (22.5%), and malignancy (12.7%). They also determined that most common presenting symptom was hematochezia (55.5%) in contrast to melena which was the presenting symptom in only 11% of patients.[14] Studies done by Vernava et al. and Longstreth also yielded similar results.[15,16]

Other less common causes include post-polypectomy bleeding, nonsteroidal anti-inflammatory drug (NSAID) related ulcers, diversion colitis, history of radiation therapy, solitary rectal ulcer, stercoral ulcers, and genetic/iatrogenic bleeding diatheses.[11]

Table
Etiology of lower gastrointestinal bleeding.

Etiology	Frequency (%)
Diverticulosis	23.3
Others	18.9
Ischemic colitis	16.0
IBD	11.7
Hemorrhoids	10.4
Unknown	9.2
Colorectal cancer	7.4
AVMs	3.1

IBD: inflammatory bowel disease; AVMs; arteriovenous malformations.

It should be noted that though LGIB may occur at any age, diseases and presentations are specific for different age groups, requiring diagnostic evaluation to be tailored accordingly. Lower GI hemorrhage in younger population is most commonly caused by Meckel's diverticulum, IBD or bleeding polyps.[17] Whilst with increasing age, diverticulosis and malignancy are the leading causes.[12]

Diverticulosis

In adults diverticular bleed is the leading etiology of clinically significant LGIB, occurring in up to 3–5% of patients with diverticulosis.[12,13,18] It is a widely prevalent condition in western populations where up to 50% of those aged 60 years or more have radiologic evidence of diverticulosis.[12,19,20]

A diverticulum is a mucosal protrusion through the wall of the colon at points of weakness where the penetrating vessel perforate through the muscular layer (propagated by increased intraluminal pressure). The perforating vessels become exposed and scantily protected, making them susceptible to chronic trauma which leads to erosion and bleeding. Bleeding risk increases with advancing age, constipation, low fiber diet and NSAID use.

Although diverticula can be located throughout the colon, about 75% of the diverticula occur in sigmoid and descending colon which are common places for increased pressure.[18] Although diverticula are less common on the right side, proximal colon diverticulosis is responsible for more than 50% of diverticular bleeding.[18,20] Fortunately, 80% of diverticular bleeds resolve spontaneously.[6,12]

Arteriovenous malformations (AVM)

Colonic angiodysplasias are one of the more common vascular malformations found in the gut.[21] These are acquired lesions that become increasingly prevalent beyond the sixth decade of life. Most colonic angiodysplasias develop from chronic, intermittent, low-grade colonic contraction which results in chronic venous congestion and capillary dilation with subsequent formation of AVM.[22] Although angiodysplasias can be found throughout the gastrointestinal tract, the most frequent location is the proximal colon.[1] Massive LGIB is much less common than diverticular bleeding as hemorrhage in angiodysplasia is of venous origin.

Numerous conditions including Von Willebrand disease, aortic stenosis and chronic kidney disease have been linked to colonic angiodysplasias, however causal evidence is lacking. Age-related systemic vascular changes result in an increased incidence of arteriovenous malformations (AVM) with age.[23,24]

Approximately 90% of AVM related bleeding resolve spontaneously; however, re-bleeding is common.[22]

Inflammatory bowel disease

LGIB from inflammatory bowel disease (IBD) is usually small volume does not require hospital admission. It is more common in ulcerative colitis (UC) than Crohn's disease (CD). When it does occur in CD, it tends to be much more common with colonic involvement than with small bowel involvement alone.[25,26]

The histological findings are comparable to those in patients with infectious and ischemic colitis; appearing as friable, erythematous, edematous, and ulcerated mucosa. In severe colitis, inflammation may extend beyond the bowel wall leading to colonic perforation.[27]

Non-IBD colitis

Of all the gastrointestinal tract, the colon is most frequently affected by ischemic insult. Ischemic colitis is a disease seen in the elderly and patients with multiple comorbidities, such as heart failure and arrhythmia increase the risk of ischemic events. Ischemia most commonly involves the

watershed areas, which include the splenic flexure and the rectosigmoid junction. Abdominal pain and bloody diarrhea are the usual presenting complaints but bleeding is rarely large or uncontrolled. Hypotension and vasoconstriction leads to mucosal friability, wall sloughing, edema, and bleeding.[28]

Radiation therapy can cause LGIB by several mechanisms including formation of mucosal telangiectasias, inflammatory changes, colitis and ulceration. Radiation colitis can occur at any time during the treatment or after, however the median reported time is 6 months to 5 years.[29] Atherosclerosis and ongoing chemotherapy increases the risk of bleeding.

Infectious colitis is characterized by fever, dysentery, lower quadrant cramps, and tenesmus. Two mechanisms of bleeding are gut wall invasion or toxin-mediated damage, depending on the organism. Salmonella, Shigella, *Campylobacter jejuni*, *E. coli*, and Yersinia species are the most common culprits causing dysentery worldwide.[30]

Systemic diseases

Vasculitides like polyarteritis nodosa and polyangiitis with granulomatosis can cause LGIB from mucosal injury and vasculitic necrosis.

Human immunodeficiency virus (HIV)-associated pathologies are also an infrequent cause of LGIB. Cytomegalovirus (CMV) colitis and Kaposi sarcoma are amongst the important HIV-associated etiologies.[31]

Colorectal cancer

Colorectal adenocarcinoma is the third most common cancer in the United States and is the predominant cause of neoplastic bleeding. Almost 10% cases of hematochezia in those past the fifth decade are due to colon cancer. Most common presentation is occult bleeding with anemia or low volume recurrent hematochezia.[32,33]

Iatrogenic

Oral anticoagulants, antiplatelet agents, and non-steroidal anti-inflammatory drugs (NSAIDs) can potentiate LGIB and should be used with caution. NSAIDs and Aspirin are the most common culprits of drug-induced bleeding and the risk of hemorrhage increases with increasing doses. These agents can potentiate or exacerbate bleeding from preexisting lesions.[34]

Post-polypectomy bleeding is a potential source of massive hemorrhage and more often arterial. It is reported in almost 1–6% cases of polypectomies and can occur even up to a month after the resection.[35,36]

Other rare but note-worthy etiologies include, drug-induced thrombocytopenias, anticoagulation or post-surgical aorto-colonic fistulas.

Anorectal disease

Benign anorectal diseases such as hemorrhoids, anal fissures, anorectal fistulas are quite common in the general population and can potentially be the underlying pathology of LGIB. Significant hemorrhage is rare however.[28]

Small bowel pathology and miscellaneous

Adenocarcinomas, hemangiomas, polyps, leiomyomas, IBD and Peutz-Jeghers syndrome are some of the small intestine pathologies that may present as LGIB. However more often than not these episodes are concealed bleeds.

LGIB in younger patients is most commonly from a Meckel's diverticulum, inflammatory bowel disease or juvenile polyps.[17] One in five patients with Meckel's diverticula are symptomatic, with the most frequent presentation being hemorrhage or obstruction.[37] Hemorrhagic episodes are usually

self-limited, but may recur from mucosal ulceration.[38] Juvenile polyps are benign hamartomas that can bleed if large enough. These are solitary lesions that can auto-amputate if they outgrow their blood supply. Most common presenting symptom is hemorrhage.

Evaluation

The main modalities employed to evaluate lower gastrointestinal bleeding (LGIB) are colonoscopy, radionuclide scans, and angiography. Other newer techniques such as CTA, video capsule endoscopy (VCE), retrograde enteroscopy, and double-balloon enteroscopy, are useful in the setting of obscure bleeding, depending on the clinical circumstance. The systemic approach to employing these modalities depends on factors such as clinical presentation, hemodynamic status of the patient, rate of bleeding and ease of localization of source of hemorrhage (Fig.).[39]

Colonoscopy is the initial test of choice for most patients with LGIB.[39,40] It can serve to identify the cause and site of bleeding as well as provide an opportunity to intervene and achieve endoscopic hemostasis. Computed tomography (CT) angiography or tagged red blood cell (RBC) scans are useful modalities for unstable patients with ongoing bleeding who are not expected to tolerate colonoscopy.[41]

Evaluation of the whole gastrointestinal tract may be needed to detect uncommon or undiagnosed lesions. Such cases can be investigated further with mesenteric angiography, upper and lower gastrointestinal endoscopy, Meckel scanning, small bowel examination, and enteroclysis.[9,41]

Initial assessment

Initial assessment begins with determination of the hemodynamic status of the patient and the need for urgent resuscitation. All patients presenting to the hospital with presumed LGIB should have 2 large bore intra-venous lines (18 gauge or larger) and/or a central venous catheter placed.

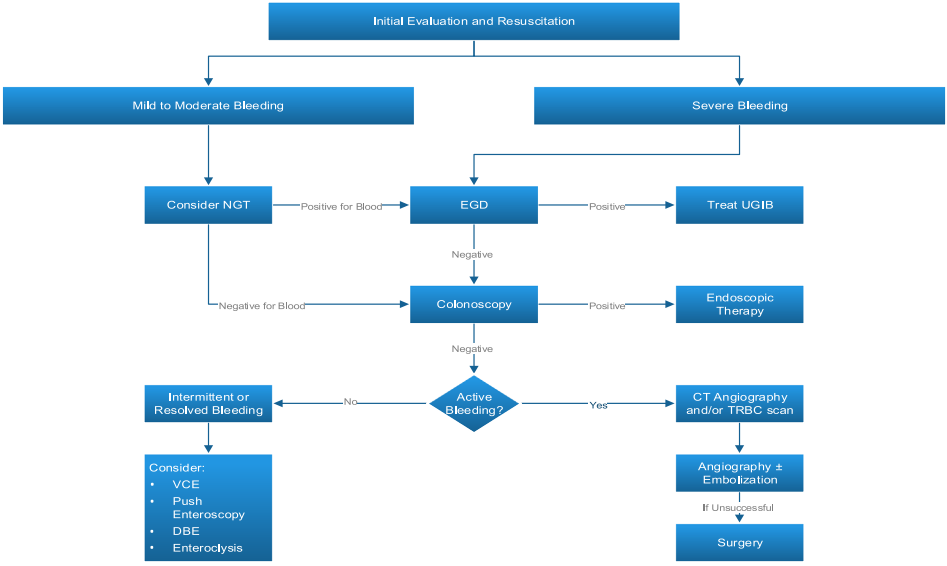

Fig. Management of lower gastrointestinal bleeding. NGT: nasogastric tube; EGD: esophagogastroduodenoscopy; UGIB: upper gastrointestinal bleeding; TRBC: tagged red blood cell; VCE: video capsule endoscopy; DBE: double balloon enteroscopy.

Depending on the clinical severity, intravenous fluids and blood products may be required with consideration of intensive care unit (ICU) admission. In clinically stable patients, a thorough history and pertinent examination must take priority to try and determine the etiology of hemorrhage; as well as formulate a comprehensive management plan based on the clinical findings.[9]

In actively bleeding patients, efforts should be directed at localizing the source of hemorrhage. It is important to note that up to 15% of patient presenting with hematochezia may have an underlying UGIB.[42,43] At the same time digital rectal examination is required to look for anorectal pathologies. Proctoscopy or sigmoidoscopy may be pursued, depending on clinical situation.

Complete blood cell (CBC) count, coagulation profile, basic metabolic profile and liver function tests are essential blood tests in the initial assessment of LGIB.

Colonoscopy

Colonoscopy is the initial test of choice for most patients with LGIB. It can successfully detect the site of LGIB in almost 80% of cases.[15] Colonoscopy also offers the possibility of therapeutic intervention in addition to diagnostic evaluation. Actively bleeding lesions once identified, can be treated with resection, adrenaline injection, laser coagulation, clipping or thermoregulation.[44]

Ideally, colonoscopy is performed after bowel preparation with use of cathartic agents. In cases of active severe bleeding, urgent unprepared hydroflush colonoscopy may be done alternatively.[45] This technique uses a system of high flow endoscopic irrigation combined with suction. This is however, not widely available. Concurrently, a large volume of blood in the gut itself provides powerful cathartic action. It is important to note that bowel preparation does not increase the rate of hemorrhage.

Urgent colonoscopy is the test of choice for patients who are hemodynamically stable. In patients with moderate bleeding, urgent colonoscopy results in better outcomes as compared to CTA with higher diagnostic yield and less complications.[43,44] It is note-worthy however that although urgent colonoscopy possibly gives better diagnostic and therapeutic yield, no evidence has been found that there is a reduction in rates of re-bleeding or surgery.[42] A randomized controlled trial by Green et. al comparing urgent versus elective colonoscopy showed no improvement in the clinical outcome or cost of care with urgent colonoscopy as compared to elective colonoscopy.[44]

The disadvantages of colonoscopy include (1) requirement for bowel preparation which can delay management (2) alternatively unprepared hydroflush colonoscopy requires several liters of water enemas. (3) Need for skilled endoscopist to perform procedure (4) risk for bowel perforation (5) need for sedation.

Plain abdominal X-rays should be obtained prior to colonoscopy if there is any suspicion of perforation or obstruction.

Radiology

Some of the radiological modalities used to evaluate LGIB include spiral CT scanning, multidetector row CT (MDCT) scanning, and computed tomographic angiography (CTA). Spiral CT scanning of the abdomen and pelvis is used when a routine evaluation does not yield the source of active gastrointestinal (GI) bleeding. Three-phase spiral CT using intravenous and oral should be performed. Spiral CT scan as a diagnostic modality for LGIB is safe, convenient, and accurate.[46]

Multidetector row CT (MDCT) scanning is occasionally used in the workup of LGIB. It has a sensitivity of 100% and 88.2% for site and etiology identification as compared to 52.9% with endoscopy for both site and etiology.[47]

Computed tomographic angiography (CTA) is often employed as a modality for evaluation of LGIB being cost-effective, easily available and accurate.[48] Studies report sensitivities ranging from 85.2% to 89% and specificities of 74–96.9%.[48–52] As compared to tagged red blood cell (RBC) scanning, a retrospective study found no significant difference in length of hospital stays, in-hospital mortality, requirement for transfusion or incidence of acute kidney injury. However, CT angiography localized

site of bleeding more accurately than tagged RBC scanning (53% vs 30%).[53] When compared with digital subtraction angiography (DSA), CTA is more accurate and less invasive.[54]

Nuclear scintigraphy

Nuclear scintigraphic imaging or radionuclide scanning include technetium-99 (99 Tc) sulfur colloid, Indium labeled RBC and 99 Tc pertechnetate-labeled red blood cell (TRBC) scans. There is an ongoing debate on the role of nuclear scintigraphy in the evaluation and management of LGIB.[9,55]

Radionuclide scans are a non-invasive and highly sensitive modality (75%) often performed prior to angiography, as scintigraphy can detect bleeding at rates as low as 0.1 mL/min as compared to 1–1.5 mL/min for angiography.[56,57] However, radionuclide scans suffer from low specificity and are thus recommended as primarily screening tools. Furthermore, scintigraphy is only useful if performed during active bleeding.[55]

The TRBC scan is highly sensitive (80%-98%) and is the preferred nuclear scan modality.[58] The advantage over other radionuclide scans is a longer half-life, allowing improved detection of intermittent or slow hemorrhage. Technetium sulfur has a very short half-life and Indium labeled RBCs have a very prolonged half-life, making them less desirable.[59]

Angiography

In patients with active ongoing LGIB, angiography can be performed by sequentially cannulating the superior mesenteric artery followed by the inferior mesenteric artery and then the celiac artery. Higher rates of bleeding (1–1.5 mL/min) are needed to visualize extravasation of contrast media.[60] Angiography can also be used to detect AVMs, varices and diverticula based on opacification and accumulation of contrast. The mean rates of localization of the bleeding site is 40% since many cases of LGIB are intermittent.[61]

Interventional modalities employed with angiography include embolization and vasopressin injection. In one study vasopressin infusion successfully achieved hemostasis in 91% patients but 50% of the patients had re-bleeding.[62] Surgical intervention such as resection or watchful waiting may be pursued after localization of hemorrhage on angiography.

Complications from angiography include arterial thrombosis, embolization or acute kidney injury. Other disadvantages of angiography are low sensitivity and usefulness only if there is active ongoing bleeding.[63]

Small bowel visualization

At times bleeding from the distal small bowel can mimic a LGIB with patients presenting with hematochezia. Small bowel visualization is required in face of negative examinations of the upper and lower gastrointestinal tract and continued hemorrhage. The methods of evaluating the small bowel include video capsule endoscopy (VCE), push enteroscopy, enteroclysis and double-balloon enteroscopy. VCE is the most frequently used test, however no consensus exists on the sequential approach to small bowel visualization.

Video capsule endoscopy (VCE)

VCE permits visualization of almost the entirety of small bowel. However, disadvantages include possible retention of capsule in patients with reduced gut motility or obstruction/strictures.[64]

Push enteroscopy

Push enteroscopy performed with perdiatric colonoscope or enteroscope is able to reach the proximal jejunum. This gives the advantage of therapeutic intervention in addition to localization of bleeding site.

Enteroclysis

Enteroclysis is performed by injecting contrast via a small bowel catheter and thus producing double contrast. However, this is best avoided in cases of acute hemorrhage.

Double-balloon enteroscopy (DBE)

Evaluation of the entire small bowel is possible in patients undergoing DBE with a diagnostic yield of 58% reported in one multicenter study.[65] Advantage over VCE is the ability to perform therapeutic intervention.

Management

The mainstays of management of LGIB are resuscitation, localization of source of hemorrhage and definitive treatment (Fig.).

Clinical outcomes have improved with advancements in available diagnostic and therapeutic modalities. The sequential use of these modalities in management of LGIB depends on the hemodynamic status and rate of hemorrhage. Surgical interventions are rarely required nowadays with the timely application of such modalities.

Resuscitation and initial assessment

As previously discussed, initial assessment should be focused on determining the hemodynamic status of the patient and rate of gastrointestinal bleeding. Initial resuscitation in unstable patients includes insertion of two large-bore IV lines and/or placement of central venous catheters, and infusion of crystalloids. This should be followed by focused history, pertinent physical examinations and blood tests once patient is stabilized.

The American College of Gastroenterology guidelines on management of LGIB recommend the following as part of initial assessment[9]: (i) evaluation and risk stratification, (ii) hemodynamic resuscitation, (iii) management of anticoagulant medications (anticoagulation reversal if INR > 2.5), endoscopic hemostasis may be attempted without reversal of anticoagulation if INR 1.5–2.5, platelet transfusion should be considered if platelet count below 50,000/ml, multidisciplinary approach to balance risk of hemorrhage with risk of thrombotic events when deciding alteration of anticoagulant/ antiplatelet medications).

Patients with ongoing bleeding, with multiple comorbidities or those requiring multiple transfusions or in hemorrhagic shock need admission to intensive care unit and urgent assessment by gastroenterologist/surgeon.

Endoscopic therapies

Colonoscopy with therapeutic intervention is a minimally invasive management option for LGIB, and colonoscopy continues to be the primary diagnostic and therapeutic modality for LGIB. Multiple modalities are available for endoscopic hemostasis which include Nd:YAG laser therapy, argon plasma coagulation, topical formalin application, injection of sclerosing agents, injections of vasoconstrictors, application of metallic clips, bipolar probe coagulation and electrocoagulation.

The American College of Gastroenterology recommends that colonoscopy should be performed within 24 hours in patients with high risk manifested by ongoing active bleeding, and hemodynamic compromise.[9] In routine clinical practice, the rate limiting step to colonoscopy after initial resuscitation is bowel preparation. In patients without signs of active bleeding, non-urgent colonoscopy can be performed after routine split dose bowel preparation with polyethylene glycol (PEG) based solution or equivalent. However, in patients with high risk and signs of active ongoing bleeding, a rapid bowel purge should be done using a nasogastric tube if needed. When colonoscopy is performed, endoscopic hemostasis should be attempted on visible active bleeding sites, visible

non-bleeding vessels or adherent clots. Experts also recommend that epinephrine injection may be used initially in active bleeding site to gain better visualization and control of bleeding. For patients with diverticular bleeding, endoscopic clipping is recommended. Other alternative endotherapeis include band ligation and thermal contact therapy. for diverticular bleeding. Alternatives are band ligation and contact thermal therapy.

Vasopressor therapy

Vasoconstrictive agents decrease blood flow and can enable platelet plug formation in the bleeding vessel. However, these should be used with caution in patients with severe atherosclerotic disease. Studies on intra-arterial infusion of vasopressors demonstrated significant reduction in mesenteric blood flow, but cessation of infusion resulted in recurrent bleeding from rebound increase in blood flow.[62]

Vasopressin is the primary agent used to decrease splanchnic blood flow by vasoconstriction. Hence it is more valuable in arterial bleeding rather than venous/capillary hemorrhage. Other disadvantages include high risk of recurrent bleeding (up to 50%), volume overload, arrhythmias and coronary vasoconstriction.[62]

Embolization

Nowadays, embolization has replaced surgery as the treatment of choice for LGIB refractory to endoscopic therapy or in patients whom colonoscopy cannot be performed or is contraindicated. Once the bleeding vessel has been accurately identified by angiography, treatment options include injection of vasopressors or intra-arterial embolization of vessel. Selective embolization can be used in high risk patients who are poor surgical candidates or can be a bridging procedure to semi-urgent segmental bowel resection once the patient is clinically stable. Microcoils, polyvinyl alcohol, gelatin sponge and oxidized cellulose can be used during arterial catheterization to embolize culprit vessels. Up to 98% success rate with achievement of immediate hemostasis has been reported. Yet embolization is not free of risks.[66–69]

The most significant risk of embolization is bowel ischemia. Various studies have suggested ischemic complication rates as high as 20%.[70,71] However, the advent of super-selective catheterization and use of microcoils has reduced rates of complications significantly. Appropriate use of these techniques has been reported to yield ischemic complication rates as low as 4.5%.[67]

Surgery

Surgical intervention is rarely required for lower gastrointestinal bleeding (LGIB) with the advanced in other less invasive management modalities.

Indications for surgery include ongoing hemorrhage in an unstable patient, recurrent, persistent hemorrhage and massive transfusion requirement.

Unstable patients should be considered for urgent explorative laparotomy following aggressive resuscitation. In patients who are stable, surgery may be an option as segmental bowel resection once site of bleeding has been accurately localized. Intraoperative localization of bleeding site can be facilitated by surgeon-guided enteroscopy or intra-operative colonoscopy. Ongoing, persistent hemorrhage from unidentified source may warrant a subtotal colectomy, however it is much less preferable to segmental colectomy.[9]

Conclusion

LGIB is a spectrum of presentation with high morbidity, mortality and healthcare costs. A systematic, step-wise approach to management of LGIB can help improve patient outcomes and

reduce costs. Fundamental principles of management are: resuscitation and stabilizing patient, localizing source of bleeding, and selecting appropriate therapeutic modality

Hemodynamic status of patient, rate of bleeding, individual expertise and available resources dictate the approach to diagnostic evaluation and selection of therapeutic modality.

References

1. Parker DR, Luo X, Jalbert JJ, Assaf AR. Impact of upper and lower gastrointestinal blood loss on healthcare utilization and costs: a systematic review. *J Med Econ*. 2011;14(3):279–287.
2. Qayed E, Dagar G, Nanchal RS. Lower gastrointestinal hemorrhage. *Crit Care Clin*. 2016;32(2):241–254.
3. Talley NJ, Jones M. Self-reported rectal bleeding in a United States community: prevalence, risk factors, and health care seeking. *Am J Gastroenterol*. 1998;93(11):2179–2183.
4. Whelan CT, Chen C, Kaboli P, Siddique J, Prochaska M, Meltzer DO. Upper versus lower gastrointestinal bleeding: a direct comparison of clinical presentation, outcomes, and resource utilization. *J Hosp Med*. 2010;5(3):141–147.
5. Bokhari M, Vernava AM, Ure T, Longo WE. Diverticular hemorrhage in the elderly—is it well tolerated? *Dis Colon Rectum*. 1996;39(2):191–195.
6. McGuire HH, Jr. Bleeding colonic diverticula. A reappraisal of natural history and management. Ann Surg 1994;220 (5):653-656.
7. Prakash C, Zuckerman GR. Acute small bowel bleeding: a distinct entity with significantly different economic implications compared with GI bleeding from other locations. *Gastrointest Endosc*. 2003;58(3):330–335.
8. Cirocchi R, Grassi V, Cavaliere D, Renzi C, Tabola R, Poli G, et al. New trends in acute management of colonic diverticular bleeding: a systematic review. *Medicine*. 2015;94(44):e1710.
9. Strate LL, Gralnek IM. ACG clinical guideline: management of patients with acute lower gastrointestinal bleeding. *Am J Gastroenterol*. 2016;111(5):755.
10. Farrell JJ, Friedman LS. Gastrointestinal bleeding in the elderly. *Gastroenterol Clin North Am*. 2001;30(2):377–407.
11. Hreinsson JP, Gumundsson S, Kalaitzakis E, Bjornsson ES. Lower gastrointestinal bleeding: incidence, etiology, and outcomes in a population-based setting. *Eur J Gastroenterol Hepatol*. 2013;25(1):37–43.
12. Zuckerman GR, Prakash C. Acute lower intestinal bleeding: part I: clinical presentation and diagnosis. *Gastrointest Endosc*. 1998;48(6):606–617.
13. Reinus JF, Brandt LJ. Vascular ectasias and diverticulosis. Common causes of lower intestinal bleeding. *Gastroenterol Clin North Am*. 1994;23(1):1–20.
14. Gayer C, Chino A, Lucas C, Tokioka S, Yamasaki T, Edelman DA, et al. Acute lower gastrointestinal bleeding in 1,112 patients admitted to an urban emergency medical center. *Surgery*. 2009;146(4):600–606. [discussion 6-7].
15. Vernava AM 3rd, Moore BA, Longo WE, Johnson FE. Lower gastrointestinal bleeding. *Dis Colon Rectum*. 1997;40(7): 846–858.
16. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. *Am J Gastroenterol*. 1997;92(3):419–424.
17. Potter GD, Sellin JH. Lower gastrointestinal bleeding. *Gastroenterol Clin North Am*. 1988;17(2):341–356.
18. Meyers MA, Alonso DR, Gray GF, Baer JW. Pathogenesis of bleeding colonic diverticulosis. *Gastroenterology*. 1976;71(4): 577–583.
19. Wong SK, Ho YH, Leong AP, Seow-Choen F. Clinical behavior of complicated right-sided and left-sided diverticulosis. *Dis Colon Rectum*. 1997;40(3):344–348.
20. Matrana MR, Margolin DA. Epidemiology and pathophysiology of diverticular disease. *Clin Colon Rectal Surg*. 2009;22(3): 141–146.
21. Nishimura N, Mizuno M, Shimodate Y, Doi A, Mouri H, Matsueda K, et al. Risk factors for active bleeding from colonic angiodysplasia confirmed by colonoscopic observation. *Int J Colorectal Dis*. 2016;31(12):1869–1873.
22. Boley SJ, Sammartano R, Adams A, DiBiase A, Kleinhaus S, Sprayregen S. On the nature and etiology of vascular ectasias of the colon. Degenerative lesions of aging. *Gastroenterology*. 1977;72(4 Pt 1):650–660.
23. Batur P, Stewart WJ, Isaacson JH. Increased prevalence of aortic stenosis in patients with arteriovenous malformations of the gastrointestinal tract in Heyde syndrome. *Arch Intern Med*. 2003;163(15):1821–1824.
24. Olearchyk AS. Heyde's syndrome. *J Thorac Cardiovasc Surg*. 1992;103(4):823–824.
25. Robert JR, Sachar DB, Greenstein AJ. Severe gastrointestinal hemorrhage in Crohn's disease. *Ann Surg*. 1991;213(3): 207–211.
26. Robert JH, Sachar DB, Aufses AH Jr, Greenstein AJ. Management of severe hemorrhage in ulcerative colitis. *Am J Surg*. 1990;159(6):550–555.
27. Magro F, Langner C, Driessen A, Ensari A, Geboes K, Mantzaris GJ, et al. European consensus on the histopathology of inflammatory bowel disease. *J Crohn's Colitis*. 2013;7(10):827–851.
28. Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. *Gastrointest Endosc*. 1999;49(2):228–238.
29. Kountouras J, Zavos C. Recent advances in the management of radiation colitis. *World J Gastroenterol*. 2008;14(48): 7289–7301.
30. Papaconstantinou HT, Thomas JS. Bacterial colitis. *Clin Colon Rectal Surg*. 2007;20(1):18–27.
31. Chalasani N, Wilcox CM. Etiology and outcome of lower gastrointestinal bleeding in patients with AIDS. *Am J Gastroenterol*. 1998;93(2):175–178.
32. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2016. *CA Cancer J Clin*. 2016;66(1):7–30.
33. Miller KD, Siegel RL, Lin CC, Mariotto AB, Kramer JL, Rowland JH, et al. Cancer treatment and survivorship statistics, 2016. *CA Cancer J Clin*. 2016;66(4):271–289.

34. Huang ES, Strate LL, Ho WW, Lee SS, Chan AT. Long-term use of aspirin and the risk of gastrointestinal bleeding. *Am J Med*. 2011;124(5):426–433.
35. Kwon MJ, Kim YS, Bae SI, Park YI, Lee KJ, Min JH, et al. Risk factors for delayed post-polypectomy bleeding. *Intest Res*. 2015;13(2):160–165.
36. Rosen L, Bub DS, Reed JF, 3rd, Nastasee SA. Hemorrhage following colonoscopic polypectomy. Dis Colon Rectum. 1993;36 (12):1126-1131.
37. Mackey WC, Dineen P. A fifty year experience with Meckel's diverticulum. *Surg Gynecol Obstet*. 1983;156(1):56–64.
38. Ymaguchi M, Takeuchi S, Awazu S. Meckel's diverticulum. Investigation of 600 patients in Japanese literature. *Am J Surg*. 1978;136(2):247–249.
39. Strate LL, Gralnek IM. ACG clinical guideline: management of patients with acute lower gastrointestinal bleeding. *Am J Gastroenterol*. 2016;111(4):459–474.
40. Peter P, Deyhle P, Brandli H, Krejs GJ, Bron B, Nuesch HJ, et al. Endoscopic diagnosis of acute perianal hemorrhage. *Schweiz Med Wochenschr*. 1976;106(26):880–883.
41. Moss AJ, Tuffaha H, Malik A. Lower GI bleeding: a review of current management, controversies and advances. *Int J Colorectal Dis*. 2016;31(2):175–188.
42. Laine L, Shah A. Randomized trial of urgent vs. elective colonoscopy in patients hospitalized with lower GI bleeding. *Am J Gastroenterol*. 2010;105(12):2636–2641. [quiz 42].
43. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. *Gastroenterology*. 1988;95(6):1569–1574.
44. Green BT, Rockey DC, Portwood G, Tarnasky PR, Guarisco S, Branch MS, et al. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage: a randomized controlled trial. *Am J Gastroenterol*. 2005;100 (11):2395–2402.
45. Wong RC. Immediate unprepared hydroflush colonoscopy for management of severe lower gastrointestinal bleeding. *Gastroenterol Hepatol*. 2013;9(1):31–34.
46. Sabharwal R, Vladica P, Chou R, Law WP. Helical CT in the diagnosis of acute lower gastrointestinal haemorrhage. *Eur J Radiol*. 2006;58(2):273–279.
47. Frattaroli FM, Casciani E, Spoletini D, Polettini E, Nunziale A, Bertini L, et al. Prospective study comparing multi-detector row CT and endoscopy in acute gastrointestinal bleeding. *World J Surg*. 2009;33(10):2209–2217.
48. Jaeckle T, Stuber G, Hoffmann MH, Jeltsch M, Schmitz BL, Aschoff AJ. Detection and localization of acute upper and lower gastrointestinal (GI) bleeding with arterial phase multi-detector row helical CT. *Eur Radiol*. 2008;18(7):1406–1413.
49. Wu LM, Xu JR, Yin Y, Qu XH. Usefulness of CT angiography in diagnosing acute gastrointestinal bleeding: a meta-analysis. *World J Gastroenterol*. 2010;16(31):3957–3963.
50. Garcia-Blazquez V, Vicente-Bartulos A, Olavarria-Delgado A, Plana MN, van der Winden D, Zamora J, et al. Accuracy of CT angiography in the diagnosis of acute gastrointestinal bleeding: systematic review and meta-analysis. *Eur Radiol*. 2013;23 (5):1181–1190.
51. Kim J, Kim YH, Lee KH, Lee YJ, Park JH. Diagnostic performance of CT angiography in patients visiting emergency department with overt gastrointestinal bleeding. *Korean J Radiol*. 2015;16(3):541–549.
52. Huprich JE, Fletcher JG, Alexander JA, Fidler JL, Burton SS, McCullough CH. Obscure gastrointestinal bleeding: evaluation with 64-section multiphase CT enterography—initial experience. *Radiology*. 2008;246(2):562–571.
53. Feuerstein JD, Ketwaroo G, Tewani SK, Cheesman A, Trivella J, Raptopoulos V, et al. Localizing acute lower gastrointestinal hemorrhage: CT angiography versus tagged RBC scintigraphy. *AJR Am J Roentgenol*. 2016;207(3):578–584.
54. Wildgruber M, Wrede CE, Zorger N, Muller-Wille R, Hamer OW, Zeman F, et al. Computed tomography versus digital subtraction angiography for the diagnosis of obscure gastrointestinal bleeding. *Eur J Radiol*. 2017;88:8–14.
55. Gunderman R, Leef J, Ong K, Reba R, Metz C. Scintigraphic screening prior to visceral arteriography in acute lower gastrointestinal bleeding. *J Nucl Med*. 1998;39(6):1081–1083.
56. Alavi A, Dann RW, Baum S, Biery DN. Scintigraphic detection of acute gastrointestinal bleeding. *Radiology*. 1977;124(3): 753–756.
57. Currie GM, Towers PA, Wheat JM. Improved detection and localization of lower gastrointestinal tract hemorrhage by subtraction scintigraphy: phantom analysis. *J Nucl Med Technol*. 2006;34(3):160–168.
58. Kester RR, Welch JP, Sziklas JP. The 99mTc-labeled RBC scan. A diagnostic method for lower gastrointestinal bleeding. *Dis Colon Rectum*. 1984;27(1):47–52.
59. Howarth DM. The role of nuclear medicine in the detection of acute gastrointestinal bleeding. *Semin Nucl Med*. 2006;36(2): 133–146.
60. Zuckerman DA, Bocchini TP, Birnbaum EH. Massive hemorrhage in the lower gastrointestinal tract in adults: diagnostic imaging and intervention. *AJR Am J Roentgenol*. 1993;161(4):703–711.
61. Leitman IM, Paull DE, Shires GT 3rd. Evaluation and management of massive lower gastrointestinal hemorrhage. *Ann Surg*. 1989;209(2):175–180.
62. Browder W, Cerise EJ, Litwin MS. Impact of emergency angiography in massive lower gastrointestinal bleeding. *Ann Surg*. 1986;204(5):530–536.
63. Widlus DM, Salis AI. Reteplase provocative visceral arteriography. *J Clin Gastroenterol*. 2007;41(9):830–833.
64. Arakawa D, Ohmiya N, Nakamura M, Honda W, Shirai O, Itoh A, et al. Outcome after enteroscopy for patients with obscure GI bleeding: diagnostic comparison between double-balloon endoscopy and videocapsule endoscopy. *Gastrointest Endosc*. 2009;69(4):866–874.
65. Ohmiya N, Yano T, Yamamoto H, Arakawa D, Nakamura M, Honda W, et al. Diagnosis and treatment of obscure GI bleeding at double balloon endoscopy. *Gastrointest Endosc*. 2007;66(3 Suppl):S72–S77.
66. Peck DJ, McLoughlin RF, Hughson MN, Rankin RN. Percutaneous embolotherapy of lower gastrointestinal hemorrhage. *J Vasc Intervent Radiol*. 1998;9(5):747–751.
67. Kuo WT, Lee DE, Saad WE, Patel N, Sahler LG, Waldman DL. Superselective microcoil embolization for the treatment of lower gastrointestinal hemorrhage. *J Vasc Intervent Radiol*. 2003;14(12):1503–1509.

ARTICLE IN PRESS

68. Ledermann HP, Schoch E, Jost R, Decurtins M, Zollikofer CL. Superselective coil embolization in acute gastrointestinal hemorrhage: personal experience in 10 patients and review of the literature. *J Vasc Intervent Radiol*. 1998;9(5):753–760.
69. Yap FY, Omene BO, Patel MN, Yohannan T, Minocha J, Knuttinen MG, et al. Transcatheter embolotherapy for gastrointestinal bleeding: a single center review of safety, efficacy, and clinical outcomes. *Dig Dis Sci*. 2013;58(7):1976–1984.
70. Uflacker R. Transcatheter embolization for treatment of acute lower gastrointestinal bleeding. *Acta Radiol*. 1987;28(4):425–430.
71. Rosenkrantz H, Bookstein JJ, Rosen RJ, Goff WB 2nd, Healy JF. Postembolic colonic infarction. *Radiology*. 1982;142(1):47–51.