THE EXPERT’S CORNER

Endoscopic Management of Acute Lower Gastrointestinal Bleeding

Louis M. Wong Kee Song, M.D., and Todd H. Baron, M.D.

GI Bleeding Team, Division of Gastroenterology and Hepatology, Mayo Clinic, Rochester, Minnesota

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INTRODUCTION

Hemorrhage from the lower gastrointestinal (LGI) tract accounts for approximately 20% of all episodes of acute GI bleeding (1–3). Although prior descriptions of LGI hemorrhage have encompassed bleeding sources beyond the ligament of Treitz, small intestinal bleeding represents a distinct entity whose management algorithm has been redefined by the advent of capsule and double-balloon endoscopy. For the purpose of this article, we define acute LGI bleeding as the recent and sudden onset of overt hemorrhage (hematochezia or melena) originating from a source within the distal terminal ileum, colon, and/or rectum, and within potential reach of a colonoscope.

Though general management principles will be outlined, this article will focus on the endoscopic approach and therapy for acute LGI bleeding. The approach described herein is derived primarily from our experience accumulated through the Mayo GI Bleeding Team practice.

INITIAL EVALUATION AND TRIAGE

Initial assessment of the patient presenting with presumed acute LGI bleeding consists of obtaining a focused history and physical examination, ordering the appropriate blood tests, assessing the severity of bleeding, providing the necessary resuscitative measures and blood transfusions, withholding particular drugs (i.e., anticoagulants, antiplatelets, nonsteroidal antiinflammatory drugs), correcting coagulation defects, and triaging the patient to the appropriate level of care (outpatient vs ward vs intensive care unit).

Elements in the history can direct the assessment toward a cause of probable or high likelihood, such as postpolypectomy bleeding in a patient who recently underwent polypectomy, exacerbation of known inflammatory bowel disease, or ischemic colitis in a vasculopathic patient with sudden onset abdominal pain followed by bloody diarrhea. Although most overt LGI bleeding episodes will manifest as hematochezia (maroon-colored stools or fresh blood and clots per rectum), melena stools may occur in the setting of bleeding from the cecum. Importantly, hematochezia associated with hemodynamic instability should prompt consideration for brisk bleeding from an upper gastrointestinal (UGI) source, particularly when risk factors such as a prior history of bleeding peptic ulcer or nonsteroidal antiinflammatory drug use are present. In the aforementioned circumstance, nasogastric tube (NGT) lavage is performed, and a positive or nondiagnostic (nonbilious, nonbloody) aspirate for blood prompts emergent upper endoscopy prior to performing colonoscopy.

Although the return of a large amount of bile via NGT makes an UGI source unlikely, we would nevertheless perform an upper endoscopy in cases where a source is not identified at colonoscopy.

The decision to manage in an outpatient setting or to admit to the intensive care unit (ICU) depends on several factors and sound clinical judgment. For example, a young, nonanemic patient with scant bleeding from suspected anorectal pathology (e.g., internal hemorrhoids) who is otherwise healthy can be managed as an outpatient. Conversely, a patient presenting with severe active bleeding (repetitive passage of 200 cc or more of bright red blood or blood clots), hemodynamic compromise (tachycardia, orthostasis, shock), >2 g/dL drop from baseline hemoglobin (Hgb), need for multiple transfusions (>2 units), supratherapeutic international normalized ratio (INR), perfusion-related complications (e.g., cardiac ischemia, dysrhythmia), advanced age, and/or significant comorbid illnesses warrants admission to the ICU. A patient in whom bleeding seems to have stopped, as evidenced by cessation of or increasing interval between bowel movements, with stable vital signs and blood counts may be admitted to the hospital ward.

Transfusion requirements should be individualized, but Hgb should be maintained at ≥10 g/dL in those with advanced age and comorbid conditions (e.g., coronary artery disease). Clotting derangements exacerbating bleeding should be corrected with fresh frozen plasma to an INR ≤2.0 and/or platelet transfusions to a platelet count ≥50,000. These thresholds also enable safer endoscopic evaluation and potential therapy. Patients with mechanical cardiac valves and/or recent metallic coronary stenting who need anticoagulation and/or antiplatelet therapy should be managed with the assistance of a cardiology specialist.

Although there is no role for barium studies in the evaluation of acute LGI bleeding, plain abdominal X-rays and/or abdominal computed tomography scan may be obtained prior to colonoscopic evaluation depending on the clinical presentation and suspected etiology, as in ischemic or infectious/inflammatory colitis or suspected aortoenteric fistula, and support anticipated findings at subsequent colonoscopy.
Although colonoscopy is our initial test of choice for diagnosis and therapy, bleeding may rarely be so severe or massive, requiring emergent intervention and precluding its use. In this situation, prompt angiography with localization of the bleeding site and potential angiotherapy should be performed. Emergent surgery should be considered only as a last resort and is rarely needed to prevent death from exsanguination. However, the morbidity and mortality associated with “blind” subtotal colectomy is higher than segmental resection of a preoperatively identified bleeding site.

A suggested algorithm for the evaluation of suspected acute LGI bleeding is presented in Figure 1.

**TIMING AND PREPARATION FOR COLONOSCOPY**

In patients with acute LGI bleeding necessitating hospitalization, we proceed with colonoscopic evaluation within 12–24 hours of presentation. Although unprepped colonoscopy is performed by some, we believe the limited visibility, marginal benefit, and perceived increased risk for perforation from blind maneuvers through often sticky bloody material precludes this approach. In patients with active bleeding and need for urgent examination, the colon is rapidly purged with 4–6 L of a polyethylene glycol (PEG)-based solution given at a rate of 1 L every 30 minutes. The solution is given by NGT if the rate of administration is not tolerated by mouth. Unless contraindicated, the prokinetic drug metoclopramide (10 mg IV) is administered prior to the purge to hasten bowel transit and control nausea and vomiting. Colonoscopy can generally be performed within 1–2 hours upon completion of the preparation and the start of liquid discharge because dilution of blood and clots can be readily aspirated or washed away from view. Moreover, accumulation of fresh blood within the residual lavage solution enables localization of the colon segment within which active bleeding is occurring.

In patients in whom bleeding seems to have stopped at the time of admission, the colon preparation can be administered in a standard and less hurried fashion, followed by colonoscopic evaluation on an “elective” basis the following day.

**COLONOSCOPIC PROCEDURE**

**Examination**

We typically employ an adult colonoscope because the larger (3.7 mm) working channel facilitates clearing of blood, clots,
and residual stool material, and enables passage of larger diameter hemostatic devices. The goal of the procedure is to identify a bleeding site and not cecal intubation per se. Meticulous examination, therefore, is performed both during insertion and withdrawal of the colonoscope because an intermittently bleeding lesion (e.g., diverticular or Dieulafoy) may only be identified during the insertion phase. Areas containing or accumulating fresh blood or clots are vigorously washed and examined as they are a potential harbinger of an underlying or adjacent bleeding site. Both syringe water flushes and a pedal-activated water jet irrigation device coupled to the endoscope are used for cleansing. The irrigation device is especially useful for removing adherent material from the mucosa and/or precisely pinpointing a bleeding site for targeted therapy (videoclip 1, Videoclips available online.) Examination under water immersion is also a useful technique to identify the nature and site of a bleeding lesion that is otherwise difficult to visualize (videoclip 2). Unless a definite bleeding colonic lesion is found or a specific lesion is sought (e.g., postpolypectomy bleeding), every attempt should be made to intubate the terminal ileum (TI). The presence of bilious, nonbloody fluid in the TI contrasting with fresh blood and clots in the right colon supports a colonic source of bleeding.

**Overview of Modalities for Hemostasis**

Endoscopic treatment modalities include injection, contact thermal coagulation, noncontact argon plasma coagulation, clipping, and banding. The use of one or a combination of these techniques depends on the site and features of the bleeding lesion(s), operator familiarity with the devices, and type of access to the bleeding site. General remarks regarding each of these techniques will be made, followed by our preferred endotherapeutic approach for specific LGI bleeding lesions.

**INJECTION THERAPY.** Epinephrine injection works by volume tamponade of the bleeding vessel and transient vasoconstriction. We commonly inject a dilute epinephrine solution (1:10,000) to stop or slow active bleeding from a focal point prior to application of a more definitive therapy. We typically instill 1–3 mL of the solution per injection site, and one or more injections in and around the bleeding point are performed until hemostasis is achieved. For a nonbleeding visible vessel (NBVV), injections 1–3 mm away from (not directly into) the vessel should be performed. Similarly, epinephrine is injected around a dense adherent clot prior to clot removal performed via cold snare guillotine or with the tip of a thermal probe (videoclip 3). It is essential to avoid or minimize injection over the distal aspect of the lesion as creation of the submucosal bleb may lift the bleeding site away from view and compromise access to further therapy. Conversely, injection can be performed in a manner that improves lesion access to other therapies (videoclip 4). Although small-volume injections (≤0.5 mL per injection; ≤2 mL total) of a desiccating agent (absolute ethanol) or a sclerosant (e.g., 5% ethalonamine oleate) can be effective at securing hemostasis, we almost never use these agents in the colon given the unpredictable depth of injury, increased risk for perforation, and availability of safer and equally effective alternatives.

**CONTACT THERMAL MODALITIES.** The heater probe and bipolar electrocoagulation devices are most commonly used for coaptive (contact) coagulation of bleeding and nonbleeding visible vessels. Both modalities are similarly effective and the selection of a particular technique rests on operator familiarity and personal preference. The objective of coaptive coagulation in the thin-walled colon is to employ the least amount of power/energy and short duration of application so as to minimize the risk of perforation, while achieving the desired effect of tissue whitening and shallow cavitation. Although optimal treatment settings have not been firmly established in human trials, the following parameters for colonic applications have been found to be effective and relatively safe in our experience: 15 J for heater probe and 12–14 W for bipolar; 2–3 s pulse duration; mild probe-tissue contact pressure. Several applications may be required to reach the treatment end point. The selection of the 7F or 10F bipolar or heat probes is dictated by the size of the bleeding point to be treated. Each probe enables water jet irrigation via a foot pedal for precise targeting and contact of the bleeding site with the probe.

**ARGON PLASMA COAGULATION.** We typically reserve the use of argon plasma coagulation (APC) for the ablation of vascular ectasias, particularly when multiple or diffuse. In these situations, the noncontact approach and ease of use make APC more suitable than contact thermal techniques. The APC settings we use are 30–45 W and 1 L/min argon flow rate, with 1–2 s pulses and tissue whitening as the end point. Although challenging at times, the APC probe should be fired at a distance of 1–3 mm from the target lesion. Inadvertent probe-tissue contact during activation may result in localized pneumatosis, which is immediately recognizable and often inconsequential.

**CLIPPING.** Both reusable and single-use clipping devices are available. Although the use of the reusable device (Olympus America Inc., Center Valley, PA) is less costly and enables the selection of clips of different sizes, it requires a skillful assistant for clip loading and handling of the device. Moreover, the rotation capability of the device becomes erratic after several uses and time delay in clip reloading are disadvantageous if a short therapeutic window is available to control an actively bleeding lesion. In most situations, we employ the single-use clipping devices due to ease of use and expeditious application. We prefer the use of the Resolution clip (Boston Scientific, Natick, MA) with reopening capability or the rotatable QuickClip2Long (Olympus America Inc., Center Valley, PA) to that of the Triclip (Cook Medical Inc., Bloomington, IN). For the latter to work, the
three-pronged configuration of the TriClip necessitates the lesion to be en face, which is not always achievable, and the thin pointy prongs may “cheese wire” through tissue during clip closure and aggravate or cause bleeding. Based upon results from animal studies (4,5), the duration of tissue anchoring and clip retention are longer for the Resolution clip compared with the QuickClip2, though in our experience, the clinical efficacy of the two clips appears to be comparable. When feasible, a tangential rather than perpendicular approach to the lesion and the application of suction prior to clip closure are maneuvers that allow the capture of more tissue between the two prongs. Both clips require enough soft tissue on either side of the prongs for adequate grasping. In circumstances with limited scope maneuverability and difficulty in accessing a particular lesion, the use of the QuickClip2 may be preferable as rotation of the clip may be the only maneuver that allows orientation of the clip in a favorable position. Clips work relatively well for pliable lesions that can be captured within the opened prongs (11–12 mm width span) and for mucosal defects that are less than 1–1.5 cm in size. These defects can be closed by sequential application of clips in a zipper-like fashion. Clips are ineffective for treating a vessel within a large hard, fibrotic base and may actually precipitate torrential bleeding by causing trauma to the vessel.

BANDING. Band ligation can be used for control of bleeding internal hemorrhoids or rectal varices and, in select circumstances, for treatment of focal and nonfibrotic bleeding colonic lesions that are < 2 cm in diameter. For example, we have used it for ligation of stigmata of diverticular bleeding that were otherwise difficult to access by other endoscopic means (Fig. 2). Caution with regard to the amount of tissue suctioned into the cap is warranted because of the potential for full-thickness tissue entrapment, necrosis, and resultant perforation.

Management of Specific LGI Bleeding Lesions

DIVERTICULAR BLEEDING. Diverticular bleeding is a common cause of acute LGI hemorrhage. Bleeding is arterial, occurring either at the neck or dome of the diverticulum. It is usually associated with painless hematochezia, although abdominal cramping may occur due to the cathartic effect of blood in the colon. Bleeding has usually stopped at the time of endoscopy although endotherapy is typically effective if an actively bleeding diverticulum or one with stigmata of recent hemorrhage is found. Our preferred approach is to use combination therapy of epinephrine injection that may evert the diverticulum for better access, followed by clipping of the vessel or the entire diverticular orifice (Fig. 3). If clipping is not feasible, contact thermal coagulation can be used cautiously, avoiding undue pressure particularly in the dome of the diverticular sac (videoclip 5). We also typically tattoo the offending diverticulum for future localization should rebleeding occur. Rarely, dense diverticular disease and a narrowed left-sided colonic lumen may impair clipping or thermal coagulation of a particular bleeding sigmoid diverticulum. In this instance, banding may be an option. Tattooing or placement of a clip adjacent to the culprit diverticulum will help localization of the latter during reinsertion of a dedicated upper endoscope equipped with the banding device, and the clip can serve as a marker for angiography should endotherapy fail.

Figure 2. Banding of colonic diverticulum (A) with vessel exposure during suction through the cap (B). Postbanding appearance (C).
In most instances, a diagnosis of presumed diverticular bleeding is made by the presence of colonic diverticula and by ruling out other causes at colonoscopy. At the index colonoscopy, residual blood and clots are cleared as best possible to leave a clean colon. The patient is maintained on a clear liquid diet only and typically kept in hospital for 48 h because a sizeable proportion of patients, in our practice, tend to rebleed during this time interval. Should rebleeding occur during this time period, one should be committed to promptly repeating the endoscopic examination and without reprepping the colon. This strategy maximizes the potential to identify an actively bleeding site and perhaps identify a cutoff between bloody and nonbloody areas, hence localizing the bleeding source to within a segment of the colon.

In patients with recurrent bleeding after two negative colonoscopic attempts, prompt angiography as close to the timing of bleeding may identify the bleeding site and allow for super-selective transcatheter embolization using particles such as microcoils or gelfoam pledgets. Empiric embolization is not recommended in the absence of bleeding given the risk of ischemic injury. We generally do not favor radionuclide bleeding scans prior to angiography given the often inaccurate localization of the bleeding site and missed opportunity to localize and treat the lesion at angiography due to the time delay in performing the bleeding scan.

POSTPOLYPECTOMY BLEEDING. Postpolypectomy bleeding may be delayed up to 1 month after polypectomy. Risk factors include the removal of large sessile right-sided colon polyps as well as resumption of anticoagulation after polypectomy. Approximately 70% of patients stop bleeding spontaneously, as evidenced by decreasing frequency or cessation of stooling, and can be managed conservatively. Urgent colonoscopy should be performed in those passing frequent bloody bowel movements suggestive of active bleeding. Given that the location(s) of the polypectomy site(s) are known a priori, some endoscopists perform colonoscopy in an unprepped colon but we prefer to purge the colon to facilitate the examination and improve the field of view. When feasible, clipping of the bleeding point, with or without epinephrine injection, is our method of choice because it does not extend tissue injury as with thermal therapy (videoclip 3). Small polypectomy defects can be completely closed by placing clips in a zipper-like fashion. However, induration within an ulcer base may hamper placement of clips, though it does provide some measure of safety for contact thermal coagulation.

ANGIODYSPLASIAS. Sporadic vascular ectasias (angiodysplasias) are more commonly found in the right colon and in the elderly. Although blood loss is typically occult,
these lesions occasionally manifest as overt LGI bleeding with orthostasis, particularly in the commonly associated setting of anticoagulation or platelet dysfunction. Although endoscopic management of an actively bleeding angiodysplasia is straightforward, decision making may be more difficult in patients who have stopped bleeding and are found to have nonbleeding angiodysplasias as well as diverticula. This is not an uncommon occurrence because both conditions affect the same age group. In this situation, we typically ablate the angiodysplasias in the absence of another definite bleeding site. Contact and noncontact thermal techniques are effective at treating vascular ectasias; clipping is less than ideal for these lesions. Our preferred method of therapy is to ablate the vascular ectasias using APC due to its ease of use and more speedy application, especially when multiple lesions are present. Prior to APC, we partially collapse the lumen but keep the lesions in view. It is important to avoid thinning of the colonic wall with excessive air insufflation as this increases the risk for perforation during therapy. Although some recommend ablation of the arborizing blood vessels starting at the periphery, we typically target the center vessel of the angiodysplasia as ensuing coagulation and edema will efface the peripheral branches and limit the extent of coagulation needed. Also, for a very large angiodysplasia, a small amount of epinephrine injected next to the central vessel will often downsize the lesion and reduce the amount and area of coagulation needed for eradication.

COLITIS. Several colitides can present with acute LGI bleeding, including inflammatory bowel disease, infectious colitis, and ischemic colitis. Features in the history, stool studies, and endoscopic assessment with biopsy typically lead to the definite diagnosis. Characteristic features of ischemic colitis include the presence of longitudinal ulcers with surrounding purplish, cyanotic mucosa as well as involvement of the “watershed” areas. In some cases, focal significant bleeding from an ulcerated area can be targeted for endoscopic therapy.

NEOPLASMS. Primary and metastatic neoplasms can manifest with acute LGI bleeding. Occasionally, there is focal bleeding from the tumor that can be temporized with coagulation therapy but bleeding is generally slow and diffuse from tumor friability. Similar to colitis, the role of endoscopy is primarily diagnostic in this setting.

RECTAL OUTLET BLEEDING. Rectal outlet bleeding is characterized by intermittent, low-volume hematochezia or blood clots mixed in stool and is caused by pathology such as internal hemorrhoids, fissures, and radiation proctopathy. Diagnosis is established by inspection of the external anal canal, anoscopy, and/or flexible endoscopy. Refractory hemorrhoidal bleeding is generally managed by a colorectal surgeon at our institution. Bleeding from radiation proctopathy is best managed with APC using short bursts of coagulation for superficial ablation of the radiation-induced telangiectasias (videoclip 6). It is important not to be overly aggressive and to avoid treatment-induced deep ulcerations given poor healing of these ulcers in a previously irradiated rectum.

DIEULAFOY LESION. A bleeding Dieulafoy lesion is caused by an exposed artery arising within a minute mucosal defect and, unless actively bleeding, may be difficult to detect. When seen, we prefer combination therapy with epinephrine injection and either contact thermal coagulation or clipping for eradication.

CONCLUSION

Acute LGI bleeding presents a more complex diagnostic and therapeutic challenge than UGI bleeding. Colonoscopy remains the mainstay of diagnosis and therapy for acute LGI bleeding. For LGI bleeding lesions that are amenable to endoscopic therapy, the proper selection of hemostatic tools and methods of use usually result in a successful outcome.

Reprint requests and correspondence: Louis M. Wong Kee Song, M.D., Mayo GI Bleeding Team, Division of Gastroenterology and Hepatology, Mayo Clinic, 200 1st Street S.W., Rochester, MN 55905.

REFERENCES


CONFLICT OF INTEREST

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### Supporting Information

Additional Supporting Information may be found in the online version of this article:

- **Videoclip 1**: Precise identification of bleeding site with water jet device.
- **Videoclip 2**: Pinpointing bleeding site using water immersion technique.
- **Videoclip 3**: Postpolypectomy bleeding site with adherent clot treated with epinephrine injection and clipping.
- **Videoclip 4**: Improving lesion access for definitive therapy using sub-mucosal fluid injection.
- **Videoclip 5**: Active diverticular bleeding treated with epinephrine injection and bipolar electrocoagulation.
- **Videoclip 6**: Radiation proctopathy treated with argon plasma coagulation.

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