I'm not a robot



Acg guidelines lower gi bleed

ACP Gastroenterology Monthly | Highlights | March 24, 2023 The American College of Gastroenterology (ACG) now recommends risk stratification to identify patients who can be discharged early and advises on the use of anticoagulant reversal agents, among other changes. The American College of Gastroenterology recently updated its guideline on management of patients with acute lower GI bleeding. The new recommendations include several changes from the previous version, published in 2016. Among these is the suggestion that clinicians use risk stratification tools (e.g., Oakland score ≤8) to identify low-risk patients with lower GI bleeding who are appropriate candidates for early discharge and outpatient diagnostic evaluation. Clinicians should use risk scores to supplement but not replace clinical judgment, the guideline noted. The guideline also newly advised clinicians on use of reversal agents in patients with life-threatening lower GI bleeding who are on vitamin K antagonists or direct oral anticoagulants and suggested CT angiography as the initial diagnostic test in patients with ongoing hemodynamically significant hematochezia. Clinicians should promptly refer patients whose CT angiography demonstrates extravasation to interventional radiology for transcatheter arteriography and possible embolization, the guideline noted. The guideline recommended colonoscopy for most patients hospitalized with lower GI bleeding because of its value in detecting a source of bleeding but added that most should be nonurgent, since studies have not shown that performing an urgent colonoscopy within 24 hours of presentation improves important clinical outcomes such as rebleeding. The guideline also suggested a restrictive strategy of red blood cell transfusion (threshold of hemoglobin level of 7 g/dL) in hemodynamically stable patients with lower GI bleeding and recommended against administration of antifibrinolytic agents such as tranexamic acid. The guideline also addressed medication after hospitalization for diverticular hemorrhage, recommending discontinuation of nonaspirin NSAIDs and aspirin for primary cardiovascular prevention. In patients with an established history of cardiovascular disease, the guideline suggested continuing aspirin. Clinicians should re-evaluate the risks and benefits of continuing nonaspirin antiplatelets such as P2Y12 receptor antagonists in a multidisciplinary setting, according to the guideline, which was published in the February American Journal of Gastroenterology. Access guidelines on the go with ACG's mobile app. Download Now Competencies in Endoscopy New - In Progress Nayantara Coelho-Prabhu, MD, FACG New - In Progress Edward Barnes, MD, MPH, FACG May 2025 Craig J. McClain, MD, FACG New May 2025 April 2025 Girish Mishra, MD, MS, FACG March 2025 Douglas R. Morgan, MD, MPH, FACG Published March 12, 2025 March 2025 Brian Jacobson, MD, MPH, FACG February 2025 Nicholas J. Shaheen, MD, MPH, MACG January 2025 Evan S. Dellon, MD, MPH Update published January 2, 2025 September 2024 William D. Chey, MD, FACG September 2024 Nicholas J. Shaheen, MD, MPH, MACG September 2024 Douglas K. Rex, MD, MACG July 2024 Catherine Frenette, MD March 2024 Scott M. Tenner, MD, MPH, FACG January 2024 Vijay H. Shah, MD January 2024 Neil Stollman, MD, FACG June 2023 Alexandra Shingina, MD, MSc June 2023 William D. Chey, MD, FACG March 2023 B. Joseph Elmunzer, MD, MSc February 2023 Neil Sengupta, MD January 2023 Alberto Rubio-Tapia, MD January 2023 Brian C. Jacobson, MD, MPH, FACG November 2022 Jonathan A. Leighton, MD, FACG October 2022 Shivangi T. Kothari, MD, FACG August 2022 Michael Camilleri, MD, FACG March 2022 Nicholas J. Shaheen, MD, MPH, FACG March 2022 Neena S. Abraham, MD, MSc (Epi), FACG February 2022 Jasmohan Bajaj January 2022 Swati G. Patel, MD, MS This is a focused update to the 2017 colorectal cancer screening recommendations from the U.S. Multi-Society Task Force on Colorectal Cancer, which represents the American College of Gastroenterology, the American Gastroenterological Association, and the American Society for Gastrointestinal Endoscopy. Published in the January 2022 issue of The American Journal of Gastroenterology. November 2021 Philip O. Katz, MD, FACG October 2021 Arnold Wald, MD, MACG May 2021 Colleen R. Kelly, MD, FACG May 2021 Loren A. Laine, MD, FACG May 2021 Naga P. Chalasani, MD, FACG March 2021 Aasma Shaukat, MD, MPH, FACG January 2021 Brian E. Lacy, MD, PhD, FACG November 2020 Aasma Shaukat, MD, MPH, FACG September 2020 Michael F. Vaezi, MD, PhD, MSc, FACG September 2020 C. Prakash Gyawali, MD, FACG March 2020 Tonya R. Kaltenbach, MD, MS, FACG March 2020 Samir Gupta, MD March 2020 Timothy B. Gardner, MD, MS, FACG February 2020 Mark Pimentel, MD, FACG January 2020 Douglas A. Simonetto, MD August 2019 Kris V. Kowdley, MD, FACG March 2019 David T. Rubin, MD, FACG June 2018 Alexander C. Ford, MB ChB, MD, FRCP March 2018 Gary R. Lichtenstein, MD, FACG Update - In Progress February 2018 Linda Lee, MD, FACG Published 2018 Update in progress 2025 June 2017 Paul M. Moayyedi, MB, ChB, PhD, MPH, FACG February 2017 Francis A. Farraye, MD, MSc, FACG January 2017 Douglas J. Robertson, MD, MPH January 2017 Paul Y. Kwo, MD, FACG, FAASLD April 2016 Mark S. Riddle, MD, DrPH March 2016 Charles J. Kahi, MD, MSc, FACG March 2016 Stephen A. McClave, MD February 2016 Tram T. Tran, MD, FACG, FAASLD Update - In Progress August 2015 Lauren B. Gerson MD, MSc, FACG April 2015 Keith D. Lindor, MD, FACG February 2015 Sapna Syngal, MD, MPH, FACG January 2015 Lawrence J. Brandt, MD, MACG January 2015 Douglas K. Rex, MD, MACG January 2015 Walter G. Park, MD, MS January 2015 Walter G. Park, MD, MS January 2015 Douglas G. Adler, MD, FACG January 2015 Sachin B. Wani, MD January 2015 Maged K. Rizk, MD, FACG January 2015 Jonathan Cohen, MD October 2014 David A. Johnson, MD, MACG August 2014 Francis M. Giardiello, MD April 2011 Nicholas J. Talley , MD, PhD, FACG New - In Progress Florence Wong, MD, FACG New - In Progress Vanessa M. Shami, MD, FACG New - In Progress Reem Z. Sharaiha, MD, MSc New - In Progress James Buxbaum, MD New - In Progress Martin L. Freeman, MD, MACG New - In Progress Anne Peery, MD, MSCR New - In Progress Carol A. Burke, MD, FACG New - In Progress Michael Kappelman, MD, FACG New - In Progress Marla Dubinsky, MD New - In Progress Marina G. Silveira, MD New - In Progress Benjamin Cohen, MD New - In Progress Jasmohan Bajaj, MD, FACG New - In Progress Alina Allen, MD New - In Progress Nadim Mahmud, MD New - In Progress Nadim Mahmud, MD New - In Progress Elliot Tapper, MD New - In Progress Shilpa Grover, MD, MPH New - In Progress Juan Gonzalez Abraldes MD, PhD As a library, NLM provides access to scientific literature. Inclusion in an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice . Author manuscript; available in PMC: 2016 Nov 7. Published in final edited form as: Am J Gastroenterol. 2016 Mar 1;111(4):459–474. doi: 10.1038/ajg.2016.41 This guideline provides recommendations for the management of patients with acute overt lower gastrointestinal hemorrhage. Hemodynamic status should be initially assessed with intravascular volume resuscitation started as needed. Risk stratification based upon clinical parameters should be performed to help distinguish patients at high and low-risk of adverse outcomes. Hematochezia associated with hemodynamic instability may be indicative of an upper GI bleeding source and thus warrants an upper endoscopy. In the majority of patients, colonoscopy should be the initial diagnostic procedure and should be performed within 24 hours of patient presentation after adequate colon preparation. Endoscopic hemostasis therapy should be provided to patients with high risk endoscopic stigmata of bleeding including active bleeding, non-bleeding visible vessel, or adherent clot. The endoscopic hemostasis modality used (mechanical, thermal, injection or combination) is most often guided by the etiology of bleeding, access to the bleeding site, and endoscopist experience with the various hemostasis modalities. Repeat colonoscopy, with endoscopic hemostasis performed if indicated, should be considered for patients with evidence of recurrent bleeding. Radiographic interventions (tagged red blood cell scintigraphy, CT angiography, angiography) should be considered in high-risk patients with ongoing bleeding who do not respond adequately to resuscitation, and who are unlikely to tolerate bowel preparation and colonoscopy. Strategies to prevent recurrent bleeding should be considered. NSAID use should be avoided in patients with a history of acute lower GI bleeding particularly if secondary to diverticulosis or angioectasia. In patients with established cardiovascular disease who require aspirin (secondary prophylaxis), aspirin should not be discontinued. The exact timing depends on the severity of bleeding, perceived adequacy of hemostasis and the risk of a thromboembolic event. Surgery for the prevention of recurrent lower gastrointestinal bleeding should be individualized and the source of bleeding should be carefully localized prior to resection. Acute overt lower gastrointestinal bleeding (LGIB) accounts for approximately 20% of all cases of GI bleeding, usually leads to hospital admission with invasive diagnostic evaluations, and consumes significant medical resources.1–3 Although most patients with acute LGIB stop bleeding spontaneously and have favorable outcomes, morbidity and mortality are increased in older patients and those with co-morbid medical conditions.4 An individual with acute LGIB classically presents with the sudden onset of hematochezia (maroon or red blood passed per rectum). However, in rare cases, patients with bleeding from the cecum/right colon can present with melena (black, tarry stools).5 In addition, hematochezia can be seen in patients with brisk upper gastrointestinal bleeding (UGIB). Approximately 15% of patients with presumed LGIB are ultimately found to have an upper GI source for their bleeding.6 Historically, LGIB was defined as bleeding from a source distal to the Ligament of Treitz. However, bleeding from the small intestine (middle GI bleeding) is distinct from colonic bleeding in terms of presentation, management and outcomes.7 For the purposes of this guideline, we define LGIB as the onset of hematochezia originating from either the colon or rectum.8 In this practice guideline, we discuss the main goals of management of patients with LGIB. First, we discuss the initial evaluation and management of patients with acute LGIB including hemodynamic resuscitation, risk stratification, and management of anti-coagulant and anti-platelet agents (anti-thrombotic agents). We then discuss colonoscopy as a diagnostic and therapeutic tool including preparation, timing and endoscopic hemostasis. Next, we outline non-colonoscopic diagnostic and therapeutic strategies for LGIB. Last, we discuss prevention of recurrent LGIB and the role of repeat colonoscopy for recurrent bleeding events. Each section of this document presents key recommendations followed by a summary of supporting evidence. A summary of the key recommendations is presented in Table 1. Summary and strength of recommendations Initial assessment Evaluation and risk stratification 1A focused history, physical examination, and laboratory evaluation should be obtained at the time of patient presentation to assess the severity of bleeding and its possible location and etiology. Initial patient assessment and hemodynamic resuscitation should be performed simultaneously (strong recommendation, very low-quality evidence). 2Hematochezia associated with hemodynamic instability may be indicative of an UGIB source, and an upper endoscopy should be performed. A nasogastric aspirate/lavage may be used to assess a possible upper GI source if suspicion of UGIB is moderate (strong recommendation, low-quality evidence). 3Risk assessment and stratification should be performed to help distinguish patients at high and low-risk of adverse outcomes and assist in patient triage including timing of colonoscopy and level of care (conditional recommendation, low-quality evidence). Hemodynamic resuscitation 4Patients with hemodynamic instability and/or suspected ongoing bleeding should receive intravenous fluid resuscitation with the goal of normalization of blood pressure and heart rate prior to endoscopic evaluation/intervention (strong recommendation, very low- quality evidence). 5Packed red blood cells should be transfused to maintain the hemoglobin above 7g/deciliter. A threshold of 9g/deciliter should be considered in patients with massive bleeding, significant comorbid illness (especially cardiovascular ischemia) or possible delay in receiving therapeutic interventions (conditional recommendations, low-quality evidence). Management of anticoagulant medications 6Endoscopic hemostasis may be considered in patients with an international normalized ratio of 1.5 – 2.5 prior to or concomitant with the administration of reversal agents. Reversal agents should be considered prior to endoscopy in patients with an INR > 2.5 (conditional recommendation, very low-quality evidence). 7Platelet transfusion should be considered to maintain a platelet count of 50 × 10/L in patients with severe bleeding and those requiring endoscopic hemostasis (conditional recommendation, very low-quality evidence). 8Platelet and plasma transfusions should be considered in patients who receive massive red blood cell transfusions (conditional recommendation, very low-quality evidence). 9In patients on anticoagulant agents, a multidisciplinary approach (e.g. hematology, cardiology, neurology, gastroenterology) should be used when deciding whether to discontinue medications or use reversal agents to balance the risk of ongoing bleeding with the risk of thromboembolic events (strong recommendation, very low-quality evidence). Colonoscopy Colonoscopy as a diagnostic tool 10Colonoscopy should be the initial diagnostic procedure for nearly all patients presenting with acute LGIB (strong recommendation, low-quality evidence). 11The colonic mucosa should be carefully inspected during both colonoscope insertion and withdrawal, with aggressive attempts made to wash residual stool and blood in order to identify the bleeding site. The endoscopist should also intubate the terminal ileum to rule out proximal blood suggestive of a small bowel lesion (conditional recommendation, very low-quality evidence). Bowel preparation 12Once the patient is hemodynamically stable, colonoscopy should be performed after adequate colon cleansing. Four to six liters of a polyethylene glycol (PEG) based solution or the equivalent should be administered over 3–4 hours until the rectal effluent is clear of blood and stool. Unprepped colonoscopy / sigmoidoscopy are not recommended (strong recommendation, low-quality evidence). 13A nasogastric tube can be considered to facilitate colon preparation in high-risk patients with ongoing bleeding who are intolerant to oral intake and are at low risk of aspiration (conditional recommendation, low-quality evidence). Timing of colonoscopy 14In patients with high-risk clinical features and signs or symptoms of ongoing bleeding, a rapid bowel purge should be initiated following hemodynamic resuscitation and a colonoscopy performed within 24 hours of patient presentation after adequate colon preparation to potentially improve diagnostic and therapeutic yield (conditional recommendation, low-quality evidence). 15In patients without high-risk clinical features or serious comorbid disease or those with high-risk clinical features without signs or symptoms of ongoing bleeding, colonoscopy should be performed next available after a colon purge (conditional recommendation, low-quality evidence). Endoscopic hemostasis therapy 16Endoscopic therapy should be provided to patients with high-risk endoscopic stigmata of bleeding: active bleeding (spurting, oozing), non-bleeding visible vessel, or adherent clot (strong recommendation, low-quality evidence). 17Diverticular bleeding —Through the scope endoscopic clips are recommended since clips may be safer in the colon than contact thermal therapy, and are generally easier to perform than band ligation particularly for right-sided colon lesions (conditional recommendation, low-quality evidence). 18Angioectasia bleeding—Non-contact thermal therapy using argon plasma coagulation (APC) is recommended (conditional recommendation, low-quality evidence). 19Post-polypectomy bleeding—Mechanical (clip) or contact thermal endotherapy, with or without the combined use of dilute epinephrine injection, is recommended (strong recommendation, low-quality evidence). 20Epinephrine injection therapy (1:10,000 or 1:20,000 dilution with saline) can be used to gain initial control of an active bleeding lesion and improve visualization, but should be used in combination with a second hemostasis modality including mechanical or contact thermal therapy to achieve definitive hemostasis (strong recommendation, very low- quality evidence). Role of repeat colonoscopy in the setting of early recurrent bleeding 21Repeat colonoscopy, with endoscopic hemostasis if indicated, should be considered for patients with evidence of recurrent bleeding (strong recommendation, very low-quality evidence). Non-colonoscopy interventions 22A surgical consultation should be requested in patients with high-risk clinical features and ongoing bleeding. In general, surgery for acute LGIB should be considered after other therapeutic options have failed, and should take into consideration extent and success of prior bleeding control measures, severity and source of bleeding and level of comorbid disease. It is important to very carefully localize the source of bleeding whenever possible prior to surgical resection to avoid continued or recurrent bleeding from an unresected culprit lesion. (conditional recommendation, very low quality evidence). 23Radiographic interventions should be considered in patients with high-risk clinical features and ongoing bleeding who have a negative upper endoscopy and do not respond adequately to hemodynamic resuscitation efforts, and are therefore unlikely to tolerate bowel preparation and urgent colonoscopy (strong recommendation, very low- quality evidence). 24If a diagnostic test is desired for localization of the bleeding site prior to angiography, CT angiography should be considered (conditional recommendation, very low-quality evidence). Prevention of recurrent lower gastrointestinal bleeding 25Non-aspirin NSAID use should be avoided in patients with a history of acute LGIB particularly if secondary to diverticulosis or angioectasia (strong recommendation, low quality evidence). 2626. In patients with established high-risk cardiovascular disease and a history of LGIB, aspirin used for secondary prevention should not be discontinued. Aspirin for primary prevention of cardiovascular events should be avoided in most patients with LGIB (strong recommendation, low-quality evidence). 27In patients on dual antiplatelet therapy or mono-therapy with non-aspirin antiplatelet agents (thienopyridine), non-aspirin antiplatelet therapy should be resumed as soon as possible and at least within 7 days based on multidisciplinary assessment of cardiovascular and GI risk and the adequacy of endoscopic therapy (as above, aspirin use should not be discontinued). However, dual antiplatelet therapy should not be discontinued in patients with an acute coronary syndrome within the past 90 days or coronary stenting within the past 30 days. (strong recommendation, low-quality evidence). With the assistance of a health sciences librarian, a systematic search of the literature was conducted covering the years 1/1/1968 through 2/3/2015 in the PubMed and EMBASE databases and the Cochrane Library including the Cochrane Database of Systematic Reviews, the Database of Abstracts of Reviews of Effect and Cochrane Central Register of Controlled Trials (CENTRAL). The PubMed search used a combination of Medical Subject Headings (MeSH) as well as terms appearing in titles and abstracts. The strategy used to cover the lower gastrointestinal tract included ("Exp Intestine, Large"[Mesh] OR "Exp Lower Gastrointestinal Tract"[Mesh] OR lower gastrointestinal[tiab] OR lower intestinal[tiab]). These terms were combined with terms for gastrointestinal bleeding including "Gastrointestinal Hemorrhage"[Mesh:noexp] OR rectal bleeding[tiab] OR colonic hemorrhage[tiab] OR colonic hemorrhages[tiab] OR colonic bleeding[tiab] OR hematochezia[tiab] OR haematochezia[tiab] OR rectal bleed[tiab] OR diverticular bleeding[tiab] OR diverticular bleed[tiab] OR diverticular hemorrhage[tiab] OR severe bleeding[tiab] OR active bleeding[tiab] OR melena[tiab] OR acute bleed[tiab] OR acute bleeding[tiab] OR acute haemorrhage[tiab] OR acute haemorrhage[tiab]) OR (LGIB[tiab] OR LIB[tiab]). The final group was limited to English language and human studies. Citations dealing with children and prostatic neoplasms were excluded. The following website will pull up the PubMed search strategy: . Search strategies in EMBASE and the Cochrane Library databases replicated the terms, limits and features used in the PubMed search strategy. In addition to the literature search, we reviewed the references of identified articles for additional studies. We also performed targeted searches on topics for which there is relevant literature for UGIB, but not LGIB including hemodynamic resuscitation/blood product transfusions and management of anti-coagulant and anti-platelet medications. We used the GRADE system to grade the quality of evidence and rate the strength of each recommendation.9 The quality of evidence, which influences the strength of recommendation, ranges from "high" (further research is very unlikely to change our confidence in the estimate of effect) to "moderate" (further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate) to "low" (further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate), and "very low" (any estimate of effect is very uncertain). The strength of a recommendation is graded as strong when the desirable effects of an intervention clearly outweigh the undesirable effects and is graded as conditional when uncertainty exists about the trade-offs.9 Other factors affecting the strength of recommendation include variability in values and preferences of patients, and if an intervention represents a wise use of resources.9 In the GRADE system, randomized trials are considered high quality evidence, but can be down rated depending on the size, quality and consistency of studies. Observational studies are generally rated as low quality studies. 1A focused history, physical examination, and laboratory evaluation should be obtained at the time of patient presentation to assess the severity of bleeding and its possible location and etiology. Initial patient assessment and hemodynamic resuscitation should be performed simultaneously (strong recommendation, very low-quality evidence).8, 10 2Hematochezia associated with hemodynamic instability may be indicative of an UGIB source, and an upper endoscopy should be performed. A nasogastric aspirate/lavage may be used to assess a possible upper GI source if suspicion of UGIB is moderate (strong recommendation, low-quality evidence).6, 11, 12 3Risk assessment and stratification should be performed to help distinguish patients at high and low-risk of adverse outcomes and assist in patient triage including timing of colonoscopy and level of care (conditional recommendation, low-quality evidence).13–18 Initial assessment of the patient presenting with presumed acute LGIB should include a focused history, physical examination, and laboratory testing with the goal of determining the severity of bleeding, its possible location, and etiology.8, 10 The history obtained should include the nature and duration of current bleeding, and any associated symptoms which may suggest a specific source such as abdominal pain and diarrhea (colitis), and altered bowel habits and weight loss (malignancy). Likewise, past medical history elements should include any prior GI bleeding events, abdominal and/or vascular surgeries, peptic ulcer disease, inflammatory bowel disease, or abdomino-pelvic radiation therapy. It is also important to assess comorbidities including cardiopulmonary, renal or hepatic disease that may put the patient at high risk of poor outcome and alter the management approach. Current or recent medication use should be noted, particularly those medications that may influence bleeding risk (nonsteroidal anti-inflammatory drugs (NSAIDs), anti-platelet agents, and anticoagulants). The physical examination should include the measurement of vital signs, including postural changes, to assess for hypovolemia. A cardiopulmonary, abdominal and digital rectal examination should also be performed. The latter can detect potential anorectal bleeding sources and determine the color of the stool. Initial laboratory testing should include a complete blood count, serum electrolytes, coagulation studies, and a type and cross match. Hematochezia associated with hemodynamic instability should lead to consideration of a brisk UGIB source especially in at-risk patients such as those with a history of peptic ulcer disease or liver disease with portal hypertension and those using anti-platelet or anti-coagulant medications.6, 11, 12, 19 An elevated blood urea nitrogen-to-creatinine ratio also suggests an UGIB source (likelihood ratio of UGIB with ratio >30:1 is 7.5),10 whereas red blood and clots are unlikely to be from an upper gastrointestinal source (likelihood ratio 0.05).10 If the likelihood of UGIB is high, an upper endoscopy should be performed. If suspicion for an UGIB source is modest, nasogastric aspirate/lavage can be used to assess possible UGIB. 6, 11, 12 A positive nasogastric aspirate indicates a very high likelihood of an UGIB (likelihood ratio=11), whereas a negative aspirate makes an UGIB less likely but still possible (negative predictive value 64%, likelihood ratio =0.6).20 Therefore, a positive or non-diagnostic (non-bloody, non-bilious) aspirate necessitates upper endoscopy prior to considering colonoscopy.12, 21 The nasogastric tube can be left in place to facilitate subsequent colon preparation.22 Clinical data available at the time of initial patient evaluation can be used to identify patients at high risk for severe bleeding and other adverse outcomes. Several tools have been developed to assess risk in acute LGIB (Tables 2 and 3),13–18 although the number of available studies is modest in comparison to UGIB. Risk factors identified for poor outcome in LGIB include markers of hemodynamic instability at presentation (tachycardia, hypotension, syncope), ongoing bleeding (gross blood on initial digital rectal examination, recurrent hematochezia), comorbid illnesses, age > 60 years, a history of diverticulosis or angioectasia, an elevated creatinine, and anemia (initial hematocrit ≤35%). In general, the likelihood of an adverse outcome increases with the number of risk factors present.16 Monitoring in an intensive care setting should be considered in patients with high-risk features. These patients may also benefit from colonoscopy after a rapid bowel preparation or radiographic interventions. Risk prediction tools for patients presenting with presumed LGIB Study No. patients Outcomes Area under the ROC curve Validation study Kollefa14 227 RebleedingSurgeryMortality 0.72 Yes Strate15, 16 252 Severe bleedingRebleeding 0.76 Yes Velayos17 94 Severe bleedingAdverse event - No Dasb13 120 RebleedingNeed for treatmentMortality 0.92–0.95 Yes Newman 161 Severe bleedingAdverse outcome 0.79 No Risk factors for poor outcome in patients with LGIB Study Risk factor Odds ratio 95% CI Kollefa14 Continuing hemorrhage 3.1 2.4–4.1 Systolic blood pressure < 100 mm Hg 3.0 2.2–4.1 Prothrombin time > 1.2 control 2.0 1.5–2.6 Altered mental status 3.2 1.5–6.8 Unstable comorbid illnessb 2.9 1.9–4.4 Strate15, 16 Heart rate >100 bpm 3.7 1.8–7.6 Systolic Blood Pressure 2 Comorbid conditionsc 1.9 1.1–3.4 Velayos17 Initial hematocrit < 35% 6.3 2.2–16.7 Abnormal vital signs after 1h 4.3 1.4–12.5 Gross blood on initial rectal exam 3.9 1.2–13.2 Newmand18 Hematocri < 35% 4.7 1.7–13.0 Bright red rectal bleeding 3.5 1.7–7.1 Age > 60 years 2.3 1.05–4.9 Newmane18 Creatinine > 150 μM 10.3 2.4–43.5 Age > 60 years 4.2 1.8–10.0 Abnormal hemodynamic parameters 2.1 1.0–4.6 Rebleeding 1.9 1.0–3.8 Smoking 0.5 0.2–1.0 4Patients with hemodynamic instability and/or suspected ongoing bleeding should receive intravenous fluid resuscitation with the goal of normalization of blood pressure and heart rate prior to endoscopic evaluation/intervention (strong recommendation, very low quality evidence). 23, 24 5Packed red blood cells should be transfused to maintain the hemoglobin above 7g/deciliter. A threshold of 9g/deciliter should be considered in patients with massive bleeding, significant comorbid illness (especially cardiovascular ischemia) or possible delay in receiving therapeutic interventions (conditional recommendation, low quality evidence).25, 26 Patients with hemodynamic instability should receive intravenous fluid resuscitation. 19, 23, 24 In UGIB, an intensive fluid (crystalloid) resuscitation strategy vs. standard of care may decrease mortality, myocardial infarction and time in the intensive care unit. However, in the lone small study these differences were not statistically significant,23, 24 and a specific resuscitation protocol was not outlined. In the critical care literature in general, there is considerable controversy regarding the timing, amount and type of fluid resuscitation.27 However, there does not appear to be a benefit of colloid over crystalloid fluids.28 In addition, some patients will require blood transfusions. Transfusion strategies specific to LGIB have not been developed. Large observational studies and a meta-analysis of three small trials of UGIB suggest that blood transfusion compared to no transfusion is associated with an increased risk of rebleeding and possibly death.25, 29–32 These findings are supported by results of a large randomized trial of patients with UGIB that found that a restrictive transfusion strategy with a transfusion threshold of hemoglobin less than 7 grams per deciliter improved survival (95% vs. 91%) and decreased rebleeding (10% vs. 16%) when compared to a threshold of 9 grams per deciliter.26 Patients with massive bleeding, acute coronary syndrome, symptomatic peripheral vascular disease or a history of cerebrovascular disease were excluded, and all patients underwent upper endoscopy within 6 hours of presentation. Therefore, patients with LGIB who have significant comorbid disease, massive, ongoing bleeding or delayed therapeutic interventions may benefit from a more lenient blood transfusion threshold. 6Endoscopic hemostasis may be considered in patients with an international normalized ratio of 1.5- 2.5 prior to or concomitant with the administration of reversal agents. Reversal agents should be considered prior to endoscopy in patients with an INR > 2.5 (conditional recommendation, very low quality evidence).33–35 7Platelet transfusion should be considered to maintain a platelet count of 50 × 109/L in patients with severe bleeding and those requiring endoscopic hemostasis (conditional recommendation, very low quality evidence).36, 37 8Platelet and plasma transfusions should be considered in patients who receive massive red blood cell transfusions (conditional recommendation, very low quality evidence).37–39 9In patients on anticoagulant agents, a multidisciplinary approach (e.g. hematology, cardiology, neurology, gastroenterology) should be used when deciding whether to discontinue medications or use reversal agents to balance the risk of ongoing bleeding with the risk of thromboembolic events (strong recommendation, very low quality evidence). 40, 41 The management of anticoagulants and antiplatelet medications in the setting of LGIB requires consideration of the risk of ongoing bleeding and the risk of thromboembolic events, and therefore requires an individualized approach. Observational studies of UGIB indicate that there is no increased risk of rebleeding following endoscopic hemostasis in patients with modest elevations in international normalized ratio (INR 1.5–2.7).33–35, 42–44 A retrospective study of 98 patients with GI bleeding suggested that patients with an INR greater than 4 had outcomes comparable to those with an INR in the 3–3.9 range, but these patients were not compared to patients with normal coagulation parameters.45 In addition, in these studies the use and timing of reversal agents was difficult to discern. An INR greater than 1.5 has been a predictor of mortality, but not rebleeding in two large observational cohort studies presumably because INR is a strong indicator of underlying comorbid disease.33, 34 After adjustment for other potential confounders, the odds ratios for mortality in these studies were 1.96 (95% CI, 1.13–3.41) and 5.63 (95% CI, 3.09–10.27), respectively.30, 32 Careful attention should therefore be given to the management of comorbid illness in patients with coagulopathy. Published standards in the hematology literature recommend platelet transfusion to maintain a platelet count of ≥ 50 × 109/L in patients with massive bleeding from any source.46, 47 There are no data to guide a threshold specific for gastrointestinal bleeding. Platelet transfusions should also be considered in patients who have a normal platelet count, but receive massive red blood cell transfusions. Traditionally, massive transfusion has been defined as more than 10 units of packed red blood cells within a 24-hour period, but recent studies in the trauma literature define this threshold as 3 or more units of packed red blood cells within 1 hour.48 The trauma literature suggests a ratio of one unit of platelets and fresh frozen plasma per unit of red blood cells transfused.38, 39, 49 A recent randomized trial indicated that a 1:1:1 ratio of plasma, platelets and red blood cells was associated with better hemostasis and fewer deaths due to exsanguination than a 1:1:2 protocol without a difference in other adverse events or death.37 The 1:1:1 ratio-based transfusion protocol likely applies outside the trauma setting,50 but no study has addressed a ratio-based transfusion protocol in gastrointestinal bleeding. New target-specific oral anticoagulants including dabigatran, rivaroxaban and apixaban are associated with an increased risk of GI bleeding. In a meta-analysis of 43 randomized controlled trials, the OR for overall bleeding was 1.45 (95% CI, 1.07–1.97).51 However, there is no direct evidence to guide the management of these agents in the setting of active GI bleeding. For elective procedures, a washout period based on the drug half-life is recommended,40 but may not be possible in patients with ongoing, acute bleeding or at high risk of thromboembolic events. In patients on target-specific oral anticoagulants, standard clotting tests may not reflect the degree of anticoagulation, and thus cannot be used to guide the safety of endoscopic interventions. A reversal agent for idarucizumab was recently approved by the FDA, and reversal agents for other non-vitamin K anticoagulants are in development.52 However, these antidotes may increase the risk of thrombosis.40, 41 Therefore, a multidisciplinary approach involving hematology, cardiology/neurology, and gastroenterology is necessary when managing patients on anticoagulant medications particularly if newer target-specific oral agents are involved to optimally balance the risk of ongoing bleeding with the risk of thromboembolic events. Please see the section on recurrent bleeding for recommendations regarding aspirin and anti-platelet medications. 10Colonoscopy should be the initial diagnostic procedure for nearly all patients presenting with acute LGIB (strong recommendation, low-quality evidence).53 11The colonic mucosa should be carefully inspected during both colonoscope insertion and withdrawal, with aggressive attempts made to wash residual stool and blood in order to identify the bleeding site.54 The endoscopist should also intubate the terminal ileum to rule out proximal blood suggestive of a small bowel lesion (conditional recommendation, very low quality evidence). Colonoscopy has both diagnostic and therapeutic roles in acute LGIB. The goal of colonoscopy in LGIB is to identify the site of bleeding and perform hemostasis if indicated. The diagnostic yield of colonoscopy in this patient population ranges from 48% to 90%.53, 55 The most common causes of acute severe LGIB include diverticulosis, angioectasia, post-polypectomy bleeding, and ischemic colitis. Other less common causes include colorectal polyps/neoplasms, Dieulafoy's lesion, inflammatory bowel disease, and anorectal conditions including solitary rectal ulcer, radiation proctitis, and rectal varices.56, 57 It is imperative to carefully inspect the colonic mucosa both on insertion and withdrawal since culprit lesions often bleed intermittently and may be missed when not actively bleeding. The endoscopist should intubate the terminal ileum to rule out proximal blood suggestive of a small bowel lesion. An adult or pediatric colonoscope with a large working channel (at least 3.3 mm) should be used because the larger working channel facilitates suctioning of blood, clots, and residual stool, and allows for the passage of large diameter (e.g. 10 Fr) endoscopic hemostasis tools. In addition, the use of a water-jet irrigation device (foot pedal controlled by the endoscopist) is recommended to facilitate removal of adherent material and residue from the colonic mucosa. 12Once the patient is hemodynamically stable, colonoscopy should be performed after adequate colon cleansing. Four to six liters of a polyethylene glycol (PEG) based solution or the equivalent should be administered over 3–4 hours until the rectal effluent is clear of blood and stool. Unprepped colonoscopy/sigmoidoscopy are not recommended (strong recommendation, low quality evidence). 10, 11, 19 13A nasogastric tube can be considered to facilitate colon preparation in high-risk patients with ongoing bleeding who are intolerant to oral intake and are at low risk of aspiration (conditional recommendation, low quality evidence). 8, 58 Colonoscopy should be performed after adequate preparation.11, 12, 22, 59 Preparation of the colon facilitates endoscopic visualization and diagnosis, and may reduce the risk of bowel perforation. Although there have been no head to head comparisons, studies using large volume (4–6 L), rapid (3–4 hours) purge protocols using PEG-based solutions with colonoscopy performed within 1–2 hours of preparation completion report high rates of definitive diagnosis (22–42%) and hemostasis (34%).11, 12, 22 Lower volume or alternative colon preparation solutions have been evaluated in the setting of colorectal cancer screening and surveillance, but not in the setting of LGIB.60 Regardless of the solution used, it is important to clear the colon of stool, clots and old blood to facilitate visualization and localization of the bleeding source. Many patients with acute LGIB are unable to tolerate rapid colon preparation and thus a nasogastric tube can be placed to facilitate this process.11, 22 In studies of urgent colonoscopy, as many as one third of patients required a nasogastric tube to facilitate rapid bowel preparation.22 In addition, administration of a prokinetic/anti-emetic agent immediately prior to initiating the colon preparation may reduce nausea and facilitate gastric emptying.8, 58 Complications of colon preparation with polyethylene glycol are rare but include aspiration pneumonia as well as fluid and electrolyte abnormalities.12, 61 Aspiration precautions should be used particularly in older and debilitated patients. Unprepped sigmoidoscopy/colonoscopy in the setting of acute LGIB is not recommended. In studies of urgent colonoscopy without oral or rectal preparation, cecal intubation rates are low (55–70%).62–64 Recent prospective pilot data in severe LGIB subjects (n=12) reported the feasibility and safety of "unprepared hydroflush colonoscopy" that combined three 1-L tap water enemas, a water-jet pump irrigation system, and a mechanical suction device to cleanse the colon.65 However, localization of bleeding, in particular diverticular bleeding, can be difficult in the setting of residual blood and stool and poor visualization may also increase the risk of perforation. Therefore, this method is recommended only as an adjunct to appropriate oral preparation until further data are available. 14In patients with high-risk clinical features and signs or symptoms of ongoing bleeding, a rapid bowel purge should be initiated following hemodynamic resuscitation and a colonoscopy performed within 24 hours of patient presentation after adequate colon preparation to potentially improve diagnostic and therapeutic yield (conditional recommendation, low-quality evidence).11, 22 15In patients without high-risk clinical features or serious comorbid disease or those with high- risk clinical features without signs or symptoms of ongoing bleeding, colonoscopy should be performed next available after a colon purge (conditional recommendation, low-quality evidence). 53, 66 Studies of timing of colonoscopy in the setting of acute LGIB are limited. Table 4 summarizes the three existing prospective studies of urgent colonoscopy for acute LGIB. In a prospective study of 48 patients with severe diverticular bleeding who underwent colonoscopy within 12 hours with endoscopic hemostasis, and 73 historical controls who underwent colonoscopy within 12 hours without endoscopic therapy, outcomes were significantly better in the endoscopic hemostasis group: rebleeding (0% vs. 53%), emergency surgery (0% vs. 35%) and hospital length of stay (median 2 days vs. 5 days).22 In addition, untreated stigmata of hemorrhage were predictive of subsequent outcomes in this study and a subsequent larger series, although the overall number of cases in each category is small and therefore the estimates may be imprecise. Rebleeding was seen in 84% of patients with active bleeding at endoscopy (n=16/19), 60% of patients with a non-bleeding visible vessel (n=3/5), and 43% with adherent clot (n=6/14).22, 67 A trial of 100 patients with acute LGIB randomized to colonoscopy within 8 hours of presentation or standard of care (colonoscopy next available or if unstable nuclear scintigraphy and angiography) found that urgent interventions significantly improved definitive diagnoses (42% vs. 22%, OR 2.6; 95% CI, 1.1–6.2), but not rebleeding, surgery or length of stay.11 No stigmata were identified on elective colonoscopy and the therapeutic yield was higher but not statistically significantly different in the urgent vs. elective group (34% endoscopic therapy vs. 20% angiographic therapy). In another trial of 72 patients randomized to colonoscopy within 12 hours or delayed colonoscopy (30–60 hours), there were no differences in rebleeding, diagnoses or need for therapy between the groups.6 Overall, retrospective studies support that urgent colonoscopy (defined variably as colonoscopy within 12–24 hours) improves diagnostic and therapeutic yield.53 In addition, studies have found that earlier time to colonoscopy is associated with reduced hospital length of stay likely because of more efficient discharge after a negative exam.53, 66, 68 It is not clear whether urgent colonoscopy improves important clinical outcomes such as rebleeding and the need for surgery. However, because diagnostic yield is improved with earlier timing, the lack of a significant benefit in existing studies may reflect inadequate statistical power or insufficient endoscopic therapy. Prospective studies of urgent colonoscopy for acute LGIB Study Study design No. patients Intervention Control Study conclusion Jensen22 Case- control, diverticular bleeding only 121 Colonoscopy < 12 h after rapid polyethylene glycol preparation; endoscopic hemostasis for stigmata of hemorrhage Colonoscopy < 12 h after rapid Polyethylene glycol preparation; NO endoscopic hemostasis for stigmata of hemorrhage Urgent colonoscopy with endoscopic therapy reduced rebleeding and need for surgery Green11 RCT 100 Colonoscopy < 8 h after rapid polyethylene glycol preparation Elective colonoscopy within 96 h; if ongoing bleeding technetium scan followed by angiography if positive More definite diagnoses in urgent colonoscopy arm; no difference in other outcomes Laine6 RCT 72 Colonoscopy < 12 h after rapid polyethylene glycol preparation Elective colonoscopy 36–60 h after admission No difference in clinical outcomes or costs 16Endoscopic therapy should be provided to patients with high-risk endoscopic stigmata of bleeding: active bleeding (spurting, oozing), non-bleeding visible vessel, or adherent clot (strong recommendation, low-quality evidence).22 17Diverticular bleeding – Through the scope endoscopic clips are recommended since clips may be safer in the colon than contact thermal therapy, and are generally easier to perform than band ligation particularly for right-sided colon lesions (Conditional recommendation, low quality evidence).69, 70 18Angioectasia bleeding – Non-contact thermal therapy using argon plasma coagulation (APC) is recommended (conditional recommendation, low quality evidence). 76, 77 19Post-polypectomy bleeding - Mechanical (clip) or contact thermal therapy, with or without the combined use of dilute epinephrine injection, is recommended (strong recommendation, very low quality evidence).71, 72 20Epinephrine injection therapy (1:10,000 or 1:20,000 dilution with saline) can be used to gain initial control of an active bleeding lesion and improve visualization, but should be used in combination with a second hemostasis modality including mechanical or contact thermal therapy to achieve definitive hemostasis. (strong recommendation, very low quality evidence).11, 22, 53 Colonoscopy with endoscopic hemostasis for colonic bleeding is safe. Adverse events were reported in 0.3% to 1.3% of more than 2,400 colonoscopies performed for acute LGIB.70, 73 Moreover, endoscopic hemostasis in the colon appears to be effective, although the optimal technique has not yet been fully characterized. Endotherapy options for acute LGIB include injection (most commonly dilute epinephrine), contact thermal therapies (bipolar/multipolar electrocoagulation, heat probe), non-contact thermal therapy (argon plasma coagulation), through-the-scope clipping devices, and band ligation. Emerging endoscopic treatments include hemostatic topical sprays/powders and large-sized over-the-scope clipping devices.74, 75 Each of these therapeutic modalities, used as monotherapy or in combination, has been reported to be safe and effective in controlling bleeding. In contrast to the numerous randomized comparative studies and meta-analyses evaluating endoscopic hemostasis modalities in acute UGIB, there have been no such studies in acute LGIB. Endoscopic treatments have most commonly been reported as individual case reports, retrospective cohort studies or prospective, non-randomized case series with small numbers of patients. Thus, the endoscopic hemostasis modality selected by the endoscopist is generally guided by the source of bleeding, access to the bleeding site, and experience with the various hemostasis device options. The most common causes of LGIB amenable to endotherapy are diverticulosis, angioectasia, and post-polypectomy bleeding.57 Endoscopic therapy for each of these bleeding etiologies will be discussed below. Diverticular bleeding is arterial, typically presents as painless hematochezia, and usually occurs from either the neck or dome of the diverticulum.22 Patients with diverticular bleeding are candidates for endoscopic treatment if active bleeding (spurting or oozing), a non-bleeding visible vessel, or an adherent clot (that cannot be removed with vigorous washing and suctioning) is found at the time of colonoscopy.22 As noted above, these stigmata of hemorrhage predict a high-risk of rebleeding without treatment.67 Jensen et al reported a prospective case series of 10 patients presenting with severe hematochezia found to be from a definitive diverticular source at the time of urgent colonoscopy. Endoscopic treatments included injection of dilute epinephrine (1:20,000 admixture with saline, in 1 or 2 ml aliquots per injection in four quadrants) as monotherapy for patients with active bleeding (n=5), and bipolar thermal coagulation (using 10–15W with moderate appositional pressure applied in 1-second intervals until vessel flattening was achieved) for those with a non-bleeding visible vessel (n=2). For patients with an adherent clot (n=3), dilute epinephrine was injected circumferentially around the site of bleeding, the clot was removed using a colon polyp snare, and any underlying stigmata was treated with bipolar thermal coagulation as described above.22 None of the 10 patients treated endoscopically had recurrent bleeding or required surgery. In a pooled analysis of case series (including 847 patients) evaluating colonoscopy and endoscopic hemostasis for diverticular bleeding, Strate and colleagues reported that following endoscopic hemostasis (n=137), early rebleeding occurred in 8% and late rebleeding in 12% of patients. There was no apparent advantage to combined endoscopic hemostasis over monotherapy.70 Endoscopic clips are an attractive treatment modality for diverticular bleeding. Compared to contact thermal therapies, clips avoid the theoretical risk of transmural injury and perforation in the thin-walled colon. In addition, improved clip design including greater tensile strength and the ability to rotate and open/close the clip prior to deployment has made clips easier to use for bleeding control.76–80 Control of diverticular bleeding using clips can be accomplished either by targeted clip placement directly on the bleeding stigma, or by closure of the diverticular orifice in a "zipper-like" fashion resulting in bleeding tamponade.80 When active bleeding is present, dilute epinephrine (0.5ml to 2ml per injection) can be injected in or around the diverticulum to slow bleeding, improve visibility, and facilitate clip placement.69 In the setting of a small or deep bleeding diverticulum, a translucent cap can be placed onto the tip of the colonoscope enabling eversion of the diverticulum for more precise localization and treatment of the bleeding lesion.69 Moreover, injection can also be used to evert the dome of the diverticulum and improve access to the bleeding site followed by clip placement.8 In the aforementioned pooled analysis by Strate and Naumann, no early rebleeding was reported after endoscopic clipping of diverticular bleeding; however, late rebleeding occurred in 17%.8 More recently, in a retrospective case series from two VA hospitals, Kaltenbach reported on the short and long term outcomes of endoscopic clipping in 24 patients with definitive diverticular hemorrhage.69 Successful endoscopic hemostasis was achieved in 21 (88%) using clips as monotherapy or in combination with epinephrine injection in the setting of active bleeding. There was no early rebleeding or adverse events (e.g. perforations). Late rebleeding (≥30 days following initial endoscopic hemostasis) occurred in 24%. Of the three patients in whom primary hemostasis was not achieved, two required emergency hemicolectomy and one patient received angiographic embolization. Case series including a total of 36 patients report good safety and efficacy of endoscopic band ligation for the treatment of diverticular bleeding with stigmata of recent hemorrhage.81–83 The banding technique described includes identification of the culprit diverticulum, marking of the site with a clip or India ink, followed by withdrawal of the colonoscope. A band ligation device is then loaded onto a gastroscope (if the bleeding lesion is located in the left colon) or a pediatric colonoscope. Once the lesion is re-identified, it is suctioned into the banding device and the band is deployed as is done in the treatment of variceal hemorrhage. Recently, Shibata et al reported on 27 cases of definitive colonic diverticular hemorrhage effectively treated (hemostasis achieved in 96.3%) using band ligation in combination with a disposable, transparent soft hood attached to the tip of the colonoscope.84 The hood allows improved visualization of diverticula and exposure of high-risk stigmata. Caution however should be exercised when contemplating using band ligation for a right side colonic diverticular bleed. Ex vivo colon specimen data have demonstrated serosal entrapment and inclusion of the muscularis propia post band ligation in the right colon.85, 86 The left colon, likely due to its thicker mucosal wall, had limited submucosal involvement and only a single site of muscularis propria involvement.85 The use of Doppler ultrasound probe monitoring has been reported as an adjunct to endoscopic treatment. In a study of 46 patients with diverticular bleeding, 24 were found to have major stigmata of hemorrhage at the time of colonoscopy.67 Doppler ultrasound probe noted arterial flow in 92% (and no flow in those without major stigmata). After treatment, no patient had residual blood flow and no patient experienced rebleeding at 30 days. However, there was no comparison to patients undergoing endoscopic treatment without Doppler probe guidance. Therefore, Doppler ultrasound probe guidance holds promise for improving the effectiveness of endoscopic hemostasis in diverticular bleeding but further data are needed. After endoscopic treatment, an India ink tattoo or clip (if not already used for hemostasis) should be placed adjacent to the culprit lesion to assist in re-localization should rebleeding occur.8, 83 Angioectasia are common in the right colon and in the elderly.87, 88 Colonic angioectasia, including radiation proctopathy, usually present with occult bleeding but can present with overt hematochezia especially in patients using anticoagulant/antiplatelet therapy.8, 58 Endoscopic hemostasis therapy is indicated if there is evidence of acute or chronic blood loss.89 Contact and noncontact thermal endoscopic therapies are effective for treatment of angiodyplasia. Non-contact thermal therapy (argon plasma coagulation) is more commonly employed because it is easy to use, safe, efficient, and has been shown to improve hemoglobin levels and reduce the frequency of blood transfusions.90, 91 Typical argon plasma coagulation power settings for the treatment of colonic angioectasia are 20–60W (lower power used in the right colon) with an argon gas flow rate 1–2.5L/min.90, 91 Lesions are obliterated using focal pulses of 0.5 – 2 seconds duration. Larger angioectasia (>10mm) and those located in the right colon may be lifted using submucosal saline injection prior to coagulation.90, 92 Post-polypectomy bleeding can occur immediately or days to weeks following polyp removal.93 Risk factors for post-polypectomy bleeding include large polyp size (>2cm), thick stalk, right colon location, and resumption of antithrombotic therapy. Endoscopic hemostasis treatments for post-polypectomy bleeding include endoscopic clipping, thermal contact, with or without the combined use of dilute epinephrine injection, and band ligation. Use of through-the-scope clipping, with or without epinephrine injection, may be preferred in order to limit additional tissue injury that occurs with contact thermal coagulation therapy.93 Hemostatic topical powders/sprays have recently been reported as an endotherapy options for acute LGIB.94 These powders/sprays (Hemostatic Agent TC-325 (Hemospray), EndoClot polysaccharide hemostatic system, and Ankaferd Bloodstopper) are delivered through the working channel of the endoscope and are intended to control 'actively' bleeding lesions. There are a limited number of case reports and small case series reporting on these modalities as primary or salvage therapy in post-polypectomy bleeding, colonic ulcerations including solitary rectal ulcer, radiation proctitis, colorectal neoplasia, and portal hypertensive colopathy.95–99 In addition, an over-the-scope clip (OTSC, Ovesco Endoscopy, Tubingen, Germany), made from a nitinol alloy, has been applied as salvage therapy in post-polypectomy bleeding.100 This clipping device is loaded onto an endoscope and deployed in a similar fashion as a band ligating device. Acute LGIB etiologies such as ischemic colitis, colitis due to inflammatory bowel disease, and colorectal neoplasms are generally not amenable to durable endoscopic hemostasis and are treated with supportive medical and/or surgical care of the underlying etiology. 21Repeat colonoscopy, with endoscopic hemostasis if indicated, should be considered for patients with evidence of recurrent bleeding (strong recommendation, very-low-quality evidence). 69, 80 The rate of rebleeding in patients with acute LGIB is poorly characterized. In randomized controlled studies, early rebleeding (defined as rebleeding prior to hospital discharge) following urgent colonoscopy is reported to be 22% and late rebleeding (defined as rebleeding after hospital discharge) is 16%.6, 11 Factors that may contribute to early or late rebleeding include underlying comorbid conditions, concurrent medication use (e.g., NSAIDs, anti-platelet agents, anticoagulants), source of index bleeding, and initial hemostasis modality.101 There are no published studies that directly evaluate the role of repeat colonoscopy in patients with early or late recurrent LGIB. However, small case series indicate that the yield of repeat colonoscopy for early rebleeding from a diverticular source is fairly high (20%).80 In this setting, the patient often remains in the hospital with a recently prepped colon, and repeat colonoscopy can be performed promptly. 22A surgical consultation should be requested in patients with high-risk clinical features and ongoing bleeding. In general, surgery for acute LGIB should be considered after other therapeutic options have failed, and should take into consideration extent and success of prior bleeding control measures, severity and source of bleeding and level of comorbid disease. It is important to very carefully localize the source of bleeding whenever possible prior to surgical resection to avoid continued or recurrent bleeding from an unresected culprit lesion. (conditional recommendation, very low quality evidence). 23Radiographic interventions should be considered in patients with high-risk clinical features and ongoing bleeding who have a negative upper endoscopy and do not respond adequately to hemodynamic resuscitation efforts, and are therefore unlikely to tolerate bowel preparation and urgent colonoscopy (strong recommendation, very low quality evidence). 102, 103 24If a diagnostic test is desired for localization of the bleeding site prior to angiography, CT angiography should be considered (conditional recommendation, very low quality evidence). 70 A number of radiographic modalities can be utilized in the setting of presumed acute LGIB. Few studies have compared radiographic interventions to colonoscopy. In one randomized trial evaluating colonoscopy within 8 hours of admission compared to elective colonoscopy if hemodynamically stable or tagged red blood cell scan followed by angiography if ongoing bleeding, more diagnoses and therapeutic interventions were made in the urgent colonoscopy arm.11 Retrospective studies also suggest the superior diagnostic and therapeutic yield of colonoscopy over radiographic algorithms.102, 103 In contrast to radiographic modalities, colonoscopy can provide a definitive diagnosis and treatment in the absence of active bleeding at the time of the exam. Nonetheless, in some patients brisk, ongoing hematochezia precludes adequate hemodynamic resuscitation and bowel preparation prior to colonoscopy. In this small subset, angiography can provide both localization and treatment. Angiography localizes a LGIB source in 25–70% of exams.104, 105 A systematic review found that super-selective angiographic embolization achieves immediate hemostasis in 40–100% of cases of diverticular bleeding with a rebleeding rate ranging from 0–50%.106 Bowel ischemia is reported in as many as one-third of patients following super-selective embolization,106 although the rate of ischemia is lower (1–4%) in more recent series.104, 107 Because angiography relies on active bleeding and has the potential for serious complications, it should be reserved for patients with very brisk, ongoing bleeding. There is considerable debate regarding the utility of tagged red blood cell (rbc) scintigraphy to localize GI bleeding prior to angiography. Some retrospective case series suggest that a screening tagged rbc scintigraphy study increases the diagnostic yield of angiography and enables targeted contrast injection.108–110 Other series have found that the diagnostic yield of

angiography is similar with or without a preceding tagged rbc scintigraphy.111, 112 If tagged rbc scintigraphy is positive, angiography should be performed immediately following to maximize the chance of a positive test. The ability of tagged rbc scintigraphy to accurately localize a bleeding source is suboptimal (65–80%),70, 73, 113 and bleeding location should be confirmed prior to surgical resection particularly if the tagged rbc scintigraphy is positive only on delayed images.114, 115 One advantage of tagged rbc scintigraphy is the ability to perform repeated scans after the initial injection of tagged cells. This makes rbc scintigraphy most suitable for the evaluation of intermittent, obscure-overt GI bleeding.108, 116 CT angiography or multi-detector row CT scan is another diagnostic modality for GI bleeding that is widely available and highly accurate at localizing the bleeding site (nearly 100%).70 However, in the only back-to-back comparison, tagged rbc scintigraphy was positive in 46% of patients and CT angiography in 27% of patients.112 Nonetheless, only 2 of 11 patients with a positive rbc scintigraphy and negative CT angiography went on to have bleeding requiring treatment. Therefore, although tagged rbc scintigraphy may be more sensitive for bleeding, CT angiography is a reasonable first line screening test if needed prior to angiography or emergent surgery because it is more expedient and accurate than tagged rbc scintigraphy. Standard precautions should be taken to avoid contrast induced nephropathy particularly since patients may undergo subsequent angiography with administration of arterial contrast.117 A surgery consultation should be requested in patients with brisk, ongoing LGIB. The quality of the evidence regarding surgery for acute LGIB is poor and mostly derived from small, retrospective reviews. Some studies report high overall mortality (up to 27%) after emergent total abdominal colectomy for massive LGIB,118 while others note no difference in morbidity or mortality when comparing limited resection to total colectomy for bleeding.119 Not surprisingly, the rebleeding rate is higher in patients after limited resection than total colectomy (18% vs. 4% in one study of 77 patients).119 In general, surgery for acute LGIB should be considered only after other therapeutic options have failed, and should take into consideration extent and success of prior bleeding control measures, severity and source of bleeding, and level of comorbid disease. It is important to very carefully localize the source of bleeding whenever possible prior to surgical resection to avoid continued or rebleeding from an unresected culprit lesion. 25Non-aspirin NSAID use should be avoided in patients with a history of acute LGIB particularly if secondary to diverticulosis or angioectasia (strong recommendation, low quality evidence). 120–122 26In patients with established high-risk cardiovascular disease and a history of LGIB, aspirin used for secondary prevention should not be discontinued. Aspirin for primary prevention of cardiovascular events should be avoided in most patients with LGIB (strong recommendation, low-quality evidence).123–125 27In patients on dual antiplatelet therapy or mono-therapy with non-aspirin antiplatelet agents (thienopyridine), non-aspirin antiplatelet therapy should be resumed as soon as possible and at least within 7 days based on multidisciplinary assessment of cardiovascular and GI risk and the adequacy of endoscopic therapy (as above, aspirin use should not be discontinued). However, dual antiplatelet therapy should not be discontinued in patients with an acute coronary syndrome within the past 90 days or coronary stenting within the past 30 days. (strong recommendation, low-quality evidence).123, 126, 127 Patients with bleeding from colonic diverticula or angioectasia are prone to recurrent bleeding events. The rate of diverticular hemorrhage recurrence at 1 year in patients who do not undergo surgical treatment was reported at 9% in a population-based study, 3 but was considerably higher (47%) in a single center study of patients with definitive diverticular bleeding.128 It is not clear that endoscopic therapy of diverticular stigmata decreases the rate of recurrent bleeding particularly because bleeding may arise from any existing diverticulum. Rates of late rebleeding are reported in approximately 15% of patients after combination injection plus thermal or clip therapy with variable follow-up periods.70 Angioectasia are also prone to rebleeding and new lesions may form throughout the gastrointestinal tract. In a systematic review, the rate of rebleeding with conservative/placebo therapy ranged from 37–45% at 1 year and 58–64% at 2 years.129 The authors rated the evidence for treatment with thalidomide or estrogen plus progesterone as low and that for octreotide as insufficient. Medical therapies resulted in a higher rate of complications than placebo. Small, retrospective studies have examined the use of argon plasma coagulation, heater probe and monopolar thermal coagulation in the treatment of angioectasia. Rates of rebleeding were no different between any of the endoscopic modalities and conservative care.129 Risk factors for recurrent LGIB are not well-studied. In one study of 83 patients with incident diverticular bleeding events who were followed for an average of 34 months, no predictors were identified including age, gender, blood transfusion requirements, hospital length of stay, endoscopic stigmata, or a previous history of bleeding.3 However, risk factors for incident diverticular bleeding events include obesity, physical inactivity, hypertension, hyperlipidemia and chronic renal insufficiency.130–133 It is not known if modification of these risk factors reduces the risk of recurrent events. Several studies indicate that NSAIDs increase the risk of both incident and recurrent LGIB. A prospective study of 132 patients hospitalized with diverticular bleeding found that recurrence was 77% among patients who continued NSAID use vs. 9% in those who discontinued.122 In another study of 342 patients with LGIB (50% due to a diverticular source) with a mean follow-up of 19 months, the cumulative rebleeding rate was 17% in patients on no antiplatelet medications, 31% on monotherapy, and 47% on dual antiplatelet therapy.121 In a multivariate analysis, the relative risk (RR) for NSAID use was 2.0 (95% CI, 1.2–3.3), for non-aspirin antiplatelet drugs 1.8 (95% CI, 1.0–2.3) and for low-dose aspirin 1.3 (95% CI, 0.8–2.3). The risk was higher in users of dual therapy than monotherapy (RR 1.8; 95% CI, 1.0–3.2). Based on this evidence, non-aspirin NSAID use should be avoided in patients with a history of acute LGIB particularly if secondary to a diverticular source. Although COX-2 selective agents are associated with a lower risk of UGIB than non-selective agents, their safety in LGIB is less clear as the results of studies are mixed perhaps due to the relative anti-platelet effect of different formulations or concomitant low-dose aspirin use in some studies.134–136 The risk of antiplatelet-associated rebleeding events may be higher in LGIB than UGIB given the lack of prophylactic measures including proton pump inhibitor (PPI) therapy and H. pylori treatment. In a study of aspirin, clopidogrel and PPI therapy following percutaneous coronary intervention, LGIB was more common than UGIB (74% vs. 26% of bleeds).137 Similarly, a large retrospective study of US Veterans, found that the incidence of lower GI events in patients on complex antithrombotic therapy was higher than that of upper GI events (70 vs. 20/1000 patient-years).138 In addition, the likelihood of early and late rebleeding in the setting of aspirin is likely to vary according to bleeding etiology and adequacy and type of initial hemostasis (for early rebleeding). As noted above, long-term recurrence is common in patients with bleeding from angioectasia and diverticulosis. The risk of early rebleeding in the setting of anti-platelet or anticoagulant use may be higher with thermal contact hemostasis methods than with mechanical methods (clips).139 The available data on resumption of aspirin in the setting of GIB are from patients with bleeding peptic ulcers. In a randomized controlled trial of immediate resumption of low-dose aspirin plus PPI vs. placebo plus PPI after endoscopic control of ulcer bleeding, there was no significant difference in rebleeding (10% vs. 5%). However, 60-day all-cause mortality (1% vs. 13%) as well as mortality secondary to cardiovascular, cerebrovascular or gastrointestinal complications was significantly lower in patients treated with aspirin.124 In a hospital-based cohort study, the risk of death was six-fold higher in patients with peptic ulcer bleeding who stopped aspirin vs. those who did not.125 Data from patients undergoing polypectomy suggest that the risk of bleeding is similar in patients discontinuing vs. continuing aspirin.140 Therefore, aspirin for secondary prophylaxis in patients with established cardiovascular disease should not be discontinued in the setting of LGIB to avoid thromboembolic events. In contrast, in patients without established cardiovascular disease and who are not at high-risk for cardiovascular events, aspirin (as primary prophylaxis) has been shown to have little net benefit (0.07% absolute risk reduction per year),141 and should be avoided in the setting of LGIB. The decision to use other antiplatelet and anticoagulant medications after an episode of LGIB requires a multidisciplinary approach that takes into consideration the risk of bleeding as well as the risk of thromboembolic events.139 During the first 30 days following coronary stenting the risk of death and myocardial infarction is doubled in patients who discontinue clopidogrel.127 The risk associated with discontinuation is also high in the first 90 days following an acute coronary syndrome. However, discontinuation for up to 7 days in patients with more distant coronary stenting or coronary syndrome appears to be safe as long as aspirin therapy is continued. In this guideline, we sought to evaluate and summarize the literature on major issues in the management of patients with acute LGIB. In general, we found the quality of the existing evidence to be low. There are only a few small, randomized trials of patients with acute LGIB, and therefore, we relied heavily on case-control or cohort studies, case series, systematic reviews or indirect evidence from trials of UGIB. Despite these limitations, we strongly endorse some of the recommendations because the potential benefits appear to outweigh the risk of harm. An approach to patients presenting with acute LGIB is outlined in Figure 1. To summarize, patients presenting with acute severe hematochezia should undergo a focused evaluation simultaneous with hemodynamic resuscitation. An upper GI bleeding source needs to be excluded in patients with hematochezia and hemodynamic instability. Colonoscopy following a colon purge is the initial test of choice in most patients presenting with acute hematochezia. In patients with high-risk features and ongoing bleeding, colonoscopy should be performed within 24 hours of presentation following a colon purge. Urgent colonoscopy (< 12 hours from presentations) may improve diagnostic and therapeutic yield, but has not been shown to reduce rates of rebleeding or surgery. Radiographic interventions should be reserved for the small group of patients with brisk bleeding who cannot be adequately stabilized for colonoscopy. Stigmata of hemorrhage can be safely and effectively treated endoscopically. The management of antiplatelet and anticoagulant medications in patients with acute LGIB requires a multidisciplinary, individualized approach that balances the risk of bleeding with the risk of a thrombotic event. However, aspirin should not be discontinued when used as secondary cardiovascular prophylaxis, and dual antiplatelet therapy should not be stopped in patients within 90 days of an acute coronary syndrome or 30 days of coronary stenting. Algorithm for the management of patients presenting with acute LGIB stratified by bleeding severity UGIB, upper gastrointestinal bleeding; CTA, computed tomographic angiography; NGT, nasogastric tube; DAPT, dual antiplatelet therapy; PEG, polyethylene glycol Grant support: This research was supported in part by grants from the National Institutes of Health R01 DK095964, and DK084157. The authors thank Lauren B. Gerson, MD, MSc for assistance with the GRADE ratings and Sherry Dodson for assistance with the literature search. ASA Aspirin NSAIDs Nonsteroidal anti-inflammatory drugs LGIB lower gastrointestinal bleeding UGIB upper gastrointestinal bleeding GI gastrointestinal PPI proton pump inhibitor RR relative risk INR international normalized ratio OR odds ratio

1.Chait MM. Lower gastrointestinal bleeding in the elderly. World J Gastrointest Endosc. 2010;2:147–54. doi: 10.4253/wjge.v2.i5.147. [DOI] [PMC free article] [PubMed] [Google Scholar]
2.Longstreth GF. Epidemiology of hospitalization for acute upper gastrointestinal hemorrhage: a population-based study. Am J Gastroenterol. 1995;90:206–10. [PubMed] [Google Scholar]
3.Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. Am J Gastroenterol. 1997;92:419–24. [PubMed] [Google Scholar]
4.Strate LL, Ayanian JZ, Kotler G, et al. Risk factors for mortality in lower intestinal bleeding. Clin Gastroenterol Hepatol. 2008;6:1004–10. doi: 10.1016/j.cgh.2008.03.021. quiz 955. [DOI] [PMC free article] [PubMed] [Google Scholar]
5.Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 20–1985. A 39-year-old man with melena and a radiologic abnormality of the cecum. N Engl J Med. 1985;312:1311–8. doi: 10.1056/NEJM198505163122008. [DOI] [PubMed] [Google Scholar]
6.Laine L, Shah A. Randomized trial of urgent vs. elective colonoscopy in patients hospitalized with lower GI bleeding. Am J Gastroenterol. 2010;105:2636–41. doi: 10.1038/ajg.2010.277. quiz 2642. [DOI] [PubMed] [Google Scholar]
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;58:330–5. doi: 10.1016/s0016-5107(03)00003-8. [DOI] [PubMed] [Google Scholar]
8.Wong Kee Song LM, Baron TH. Endoscopic management of acute lower gastrointestinal bleeding. Am J Gastroenterol. 2008;103:1881–7. doi: 10.1111/j.1572-0241.2008.02075.x. [DOI] [PubMed] [Google Scholar]
9.Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. Bmj. 2008;336:924–6. doi: 10.1136/bmj.39489.470347.AD. [DOI] [PMC free article] [PubMed] [Google Scholar]
10.Srygley FD, Gerardo CJ, Tran T, et al. Does this patient have a severe upper gastrointestinal bleed? Jama. 2012;307:1072–9. doi: 10.1001/jama.2012.253. [DOI] [PubMed] [Google Scholar]
11.Green BT, Rockey DC, Portwood G, et al. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage: a randomized controlled trial. Am J Gastroenterol. 2005;100:2395–402. doi: 10.1111/j.1572-0241.2005.00306.x. [DOI] [PubMed] [Google Scholar]
12.Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology. 1988;95:1569–74. doi: 10.1016/s0016-5085(88)80079-9. [DOI] [PubMed] [Google Scholar]
13.Das A, Ben-Menachem T, Cooper GS, et al. Prediction of outcome in acute lower-gastrointestinal haemorrhage based on an artificial neural network: internal and external validation of a predictive model. Lancet. 2003;362:1261–6. doi: 10.1016/S0140-6736(03)14568-0. [DOI] [PubMed] [Google Scholar]
14.Kollef MH, O'Brien JD, Zuckerman GR, et al. BLEED: a classification tool to predict outcomes in patients with acute upper and lower gastrointestinal hemorrhage. Crit Care Med. 1997;25:1125–32. doi: 10.1097/00003246-199707000-00011. [DOI] [PubMed] [Google Scholar]
15.Strate LL, Orav EJ, Syngal S. Early predictors of severity in acute lower intestinal tract bleeding. Arch Intern Med. 2003;163:838–43. doi: 10.1001/archinte.163.7.838. [DOI] [PubMed] [Google Scholar]
16.Strate LL, Saltzman JR, Ookubo R, et al. Validation of a clinical prediction rule for severe acute lower intestinal bleeding. Am J Gastroenterol. 2005;100:1821–7. doi: 10.1111/j.1572-0241.2005.41755.x. [DOI] [PubMed] [Google Scholar]
17.Velayos FS, Williamson A, Sousa KH, et al. Early predictors of severe lower gastrointestinal bleeding and adverse outcomes: a prospective study. Clin Gastroenterol Hepatol. 2004;2:485–90. doi: 10.1016/s1542-3565(04)00167-3. [DOI] [PubMed] [Google Scholar]
18.Newman J, Fitzgerald JE, Gupta S, et al. Outcome predictors in acute surgical admissions for lower gastrointestinal bleeding. Colorectal Dis. 2012;14:1020–6. doi: 10.1111/j.1463-1318.2011.02824.x. [DOI] [PubMed] [Google Scholar]
19.Gralnek IM, Barkun AN, Bardou M. Management of acute bleeding from a peptic ulcer. N Engl J Med. 2008;359:928–37. doi: 10.1056/NEJMra0706113. [DOI] [PubMed] [Google Scholar]
20.Witting MD, Magder L, Heins AE, et al. Usefulness and validity of diagnostic nasogastric aspiration in patients without hematemesis. Ann Emerg Med. 2004;43:525–32. doi: 10.1016/j.annemergmed.2003.09.002. [DOI] [PubMed] [Google Scholar]
21.Wong JL, Dalton HR. Urgent endoscopy in lower gastrointestinal bleeding. Gut. 2001;48:155–6. doi: 10.1136/gut.48.2.155. [DOI] [PMC free article] [PubMed] [Google Scholar]
22.Jensen DM, Machicado GA, Jutabha R, et al. Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. N Engl J Med. 2000;342:78–82. doi: 10.1056/NEJM200001133420202. [DOI] [PubMed] [Google Scholar]
23.Baradarian R, Ramdhaney S, Chapalamadugu R, et al. Early intensive resuscitation of patients with upper gastrointestinal bleeding decreases mortality. Am J Gastroenterol. 2004;99:619–22. doi: 10.1111/j.1572-0241.2004.04073.x. [DOI] [PubMed] [Google Scholar]
24.Lim CH. Early intensive resuscitation of patients with upper gastrointestinal bleeding decreases mortality. Am J Gastroenterol. 2004;99:2502. doi: 10.1111/j.1572-0241.2004.41389_5.x. author reply 2502–3. [DOI] [PubMed] [Google Scholar]
25.Jairath V, Hearnshaw S, Brunskill SJ, et al. Red cell transfusion for the management of upper gastrointestinal haemorrhage. Cochrane Database Syst Rev. 2010:CD006613. doi: 10.1002/14651858.CD006613.pub3. [DOI] [PubMed] [Google Scholar]
26.Villanueva C, Colomo A, Bosch A. Transfusion for acute upper gastrointestinal bleeding. N Engl J Med. 2013;368:1362–3. doi: 10.1056/NEJMc1301256. [DOI] [PubMed] [Google Scholar]
27.Kwan I, Bunn F, Chinnock P, et al. Timing and volume of fluid administration for patients with bleeding. Cochrane Database Syst Rev. 2014;3:CD002245. doi: 10.1002/14651858.CD002245.pub2. [DOI] [PMC free article] [PubMed] [Google Scholar]
28.Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev. 2013;2:CD000567. doi: 10.1002/14651858.CD000567.pub6. [DOI] [PubMed] [Google Scholar]
29.Blair SD, Janvrin SB, McCollum CN, et al. Effect of early blood transfusion on gastrointestinal haemorrhage. Br J Surg. 1986;73:783–5. doi: 10.1002/bjs.1800731007. [DOI] [PubMed] [Google Scholar]
30.Elizalde JI, Moitinho E, Garcia-Pagan JC, et al. Effects of increasing blood hemoglobin levels on systemic hemodynamics of acutely anemic cirrhotic patients. J Hepatol. 1998;29:789–95. doi: 10.1016/s0168-8278(98)80260-2. [DOI] [PubMed] [Google Scholar]
31.Hearnshaw SA, Logan RF, Palmer KR, et al. Outcomes following early red blood cell transfusion in acute upper gastrointestinal bleeding. Aliment Pharmacol Ther. 2010;32:215–24. doi: 10.1111/j.1365-2036.2010.04348.x. [DOI] [PubMed] [Google Scholar]
32.Restellini S, Kherad O, Jairath V, et al. Red blood cell transfusion is associated with increased rebleeding in patients with nonvariceal upper gastrointestinal bleeding. Aliment Pharmacol Ther. 2013;37:316–22. doi: 10.1111/apt.12170. [DOI] [PubMed] [Google Scholar]
33.Jairath V, Kahan BC, Stanworth SJ, et al. Prevalence, management, and outcomes of patients with coagulopathy after acute nonvariceal upper gastrointestinal bleeding in the United Kingdom. Transfusion. 2013;53:1069–76. doi: 10.1111/j.1537-2995.2012.03849.x. [DOI] [PubMed] [Google Scholar]
34.Shingina A, Barkun AN, Razzaghi A, et al. Systematic review: the presenting international normalised ratio (INR) as a predictor of outcome in patients with upper nonvariceal gastrointestinal bleeding. Aliment Pharmacol Ther. 2011;33:1010–8. doi: 10.1111/j.1365-2036.2011.04618.x. [DOI] [PubMed] [Google Scholar]
35.Wolf AT, Wasan SK, Saltzman JR. Impact of anticoagulation on rebleeding following endoscopic therapy for nonvariceal upper gastrointestinal hemorrhage. Am J Gastroenterol. 2007;102:290–6. doi: 10.1111/j.1572-0241.2006.00969.x. [DOI] [PubMed] [Google Scholar]
36.Siegal DM. Managing target-specific oral anticoagulant associated bleeding including an update on pharmacological reversal agents. J Thromb Thrombolysis. 2015;39:395–402. doi: 10.1007/s11239-015-1167-9. [DOI] [PubMed] [Google Scholar]
37.Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. Jama. 2015;313:471–82. doi: 10.1001/jama.2015.12. [DOI] [PMC free article] [PubMed] [Google Scholar]
38.Holcomb JB, Wade CE, Michalek JE, et al. Increased plasma and platelet to red blood cell ratios improves outcome in 466 massively transfused civilian trauma patients. Ann Surg. 2008;248:447–58. doi: 10.1097/SLA.0b013e318185a9ad. [DOI] [PubMed] [Google Scholar]
39.Shaz BH, Dente CJ, Nicholas J, et al. Increased number of coagulation products in relationship to red blood cell products transfused improves mortality in trauma patients. Transfusion. 2010;50:493–500. doi: 10.1111/j.1537-2995.2009.02414.x. [DOI] [PubMed] [Google Scholar]
40.Baron TH, Kamath PS, McBane RD. New anticoagulant and antiplatelet agents: a primer for the gastroenterologist. Clin Gastroenterol Hepatol. 2014;12:187–95. doi: 10.1016/j.cgh.2013.05.020. [DOI] [PubMed] [Google Scholar]
41.Siegal DM. Managing target-specific oral anticoagulant associated bleeding including an update on pharmacological reversal agents. J Thromb Thrombolysis. 2015 doi: 10.1007/s11239-015-1167-9. [DOI] [PubMed] [Google Scholar]
42.Choudari CP, Rajgopal C, Palmer KR. Acute gastrointestinal haemorrhage in anticoagulated patients: diagnoses and response to endoscopic treatment. Gut. 1994;35:464–6. doi: 10.1136/gut.35.4.464. [DOI] [PMC free article] [PubMed] [Google Scholar]
43.Lu Y, Loffroy R, Lau JY, et al. Multidisciplinary management strategies for acute non-variceal upper gastrointestinal bleeding. Br J Surg. 2014;101:e34–50. doi: 10.1002/bjs.9351. [DOI] [PubMed] [Google Scholar]
44.Thomopoulos KC, Mimidis KP, Theocharis GJ, et al. Acute upper gastrointestinal bleeding in patients on long-term oral anticoagulation therapy: endoscopic findings, clinical management and outcome. World J Gastroenterol. 2005;11:1365–8. doi: 10.3748/wjg.v11.i9.1365. [DOI] [PMC free article] [PubMed] [Google Scholar]
45.Rubin TA, Murdoch M, Nelson DB. Acute GI bleeding in the setting of supratherapeutic international normalized ratio in patients taking warfarin: endoscopic diagnosis, clinical management, and outcomes. Gastrointest Endosc. 2003;58:369–73. [PubMed] [Google Scholar]
46.Contreras M. Final statement from the consensus conference on platelet transfusion. Transfusion. 1998;38:796–7. doi: 10.1046/j.1537-2995.1998.38898375520.x. [DOI] [PubMed] [Google Scholar]
47.Razzaghi A, Barkun AN. Platelet transfusion threshold in patients with upper gastrointestinal bleeding: a systematic review. J Clin Gastroenterol. 2012;46:482–6. doi: 10.1097/MCG.0b013e31823d33e3. [DOI] [PubMed] [Google Scholar]
48.Savage SA, Sumislawski JJ, Croce MA, et al. Using critical administration thresholds to predict abbreviated laparotomy. J Trauma Acute Care Surg. 2014;77:599–603. doi: 10.1097/TA.0000000000000301. [DOI] [PubMed] [Google Scholar]
49.Dzik WH, Blajchman MA, Fergusson D, et al. Clinical review: Canadian National Advisory Committee on Blood and Blood Products--Massive transfusion consensus conference 2011: report of the panel. Crit Care. 2011;15:242. doi: 10.1186/cc10498. [DOI] [PMC free article] [PubMed] [Google Scholar]
50.Murphy CH, Hess JR. Massive transfusion: red blood cell to plasma and platelet unit ratios for resuscitation of massive hemorrhage. Curr Opin Hematol. 2015;22:533–9. doi: 10.1097/MOH.0000000000000184. [DOI] [PubMed] [Google Scholar]
51.Holster IL, Valkhoff VE, Kuipers EJ, et al. New oral anticoagulants increase risk for gastrointestinal bleeding: a systematic review and meta-analysis. Gastroenterology. 2013;145:105–112. e15. doi: 10.1053/j.gastro.2013.02.041. [DOI] [PubMed] [Google Scholar]
52.Husted S, Verheugt FW, Comuth WJ. Reversal Strategies for NOACs: State of Development, Possible Clinical Applications and Future Perspectives. Drug Saf. 2015 doi: 10.1007/s40264-015-0357-x. [DOI] [PubMed] [Google Scholar]
53.Strate LL, Syngal S. Timing of colonoscopy: impact on length of hospital stay in patients with acute lower intestinal bleeding. Am J Gastroenterol. 2003;98:317–22. doi: 10.1111/j.1572-0241.2003.07232.x. [DOI] [PubMed] [Google Scholar]
54.Jensen DM. Management of patients with severe hematochezia--with all current evidence available. Am J Gastroenterol. 2005;100:2403–6. doi: 10.1111/j.1572-0241.2005.00298.x. [DOI] [PubMed] [Google Scholar]
55.Davila RE, Rajan E, Adler DG, et al. ASGE Guideline: the role of endoscopy in the patient with lower-GI bleeding. Gastrointest Endosc. 2005;62:656–60. doi: 10.1016/j.gie.2005.07.032. [DOI] [PubMed] [Google Scholar]
56.Gralnek IM, Ron-Tal Fisher O, Holub JL, et al. The role of colonoscopy in evaluating hematochezia: a population-based study in a large consortium of endoscopy practices. Gastrointest Endosc. 2013;77:410–8. doi: 10.1016/j.gie.2012.10.025. [DOI] [PMC free article] [PubMed] [Google Scholar]
57.Ron-Tal Fisher O, Gralnek IM, Eisen GM, et al. Endoscopic hemostasis is rarely used for hematochezia: a population-based study from the Clinical Outcomes Research Initiative National Endoscopic Database. Gastrointest Endosc. 2014;79:317–25. doi: 10.1016/j.gie.2013.09.004. [DOI] [PMC free article] [PubMed] [Google Scholar]
58.Jensen DM, Machicado GA. Colonoscopy for diagnosis and treatment of severe lower gastrointestinal bleeding. Routine outcomes and cost analysis. Gastrointest Endosc Clin N Am. 1997;7:477–98. [PubMed] [Google Scholar]
59.The role of endoscopy in the patient with lower gastrointestinal bleeding. American Society for Gastrointestinal Endoscopy. Gastrointest Endosc. 1998;48:685–8. [PubMed] [Google Scholar]
60.Johnson DA, Barkun AN, Cohen LB, et al. Optimizing adequacy of bowel cleansing for colonoscopy: recommendations from the US Multi-Society Task Force on Colorectal Cancer. Am J Gastroenterol. 2014;109:1528–45. doi: 10.1038/ajg.2014.272. [DOI] [PubMed] [Google Scholar]
61.Dominitz JA, Eisen GM, Baron TH, et al. Complications of colonoscopy. Gastrointest Endosc. 2003;57:441–5. doi: 10.1016/s0016-5107(03)80005-6. [DOI] [PubMed] [Google Scholar]
62.Chaudhry V, Hyser MJ, Gracias VH, et al. Colonoscopy: the initial test for acute lower gastrointestinal bleeding. Am Surg. 1998;64:723–8. [PubMed] [Google Scholar]
63.Ohyama T, Sakurai Y, Ito M, et al. Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding. Digestion. 2000;61:189–92. doi: 10.1159/000007756. [DOI] [PubMed] [Google Scholar]
64.Tada M, Shimizu S, Kawai K. Emergency colonoscopy for the diagnosis of lower intestinal bleeding. Gastroenterol Jpn. 1991;26(Suppl 3):121–4. doi: 10.1007/BF02779279. [DOI] [PubMed] [Google Scholar]
65.Repaka A, Atkinson MR, Faulx AL, et al. Immediate unprepared hydroflush colonoscopy for severe lower GI bleeding: a feasibility study. Gastrointest Endosc. 2012;76:367–73. doi: 10.1016/j.gie.2012.03.1391. [DOI] [PMC free article] [PubMed] [Google Scholar]
66.Schmulewitz N, Fisher DA, Rockey DC. Early colonoscopy for acute lower GI bleeding predicts shorter hospital stay: a retrospective study of experience in a single center. Gastrointest Endosc. 2003;58:841–6. doi: 10.1016/s0016-5107(03)02304-6. [DOI] [PubMed] [Google Scholar]
67.Jensen DM, Ohning GV, Kovacs TO, et al. Natural history of definitive diverticular hemorrhage based on stigmata of recent hemorrhage and colonoscopic Doppler blood flow monitoring for risk stratification and definitive hemostasis. Gastrointest Endosc. 2015 doi: 10.1016/j.gie.2015.07.033. [DOI] [PMC free article] [PubMed] [Google Scholar]
68.Navaneethan U, Njei B, Venkatesh PG, et al. Timing of colonoscopy and outcomes in patients with lower GI bleeding: a nationwide population-based study. Gastrointest Endosc. 2014;79:297–306. e12. doi: 10.1016/j.gie.2013.08.001. [DOI] [PubMed] [Google Scholar]
69.Kaltenbach T, Watson R, Shah J, et al. Colonoscopy with clipping is useful in the diagnosis and treatment of diverticular bleeding. Clin Gastroenterol Hepatol. 2012;10:131–7. doi: 10.1016/j.cgh.2011.10.029. [DOI] [PubMed] [Google Scholar]
70.Strate LL, Naumann CR. The role of colonoscopy and radiological procedures in the management of acute lower intestinal bleeding. Clin Gastroenterol Hepatol. 2010;8:333–43. doi: 10.1016/j.cgh.2009.12.017. quiz e44. [DOI] [PubMed] [Google Scholar]
71.Guo SB, Gong AX, Leng J, et al. Application of endoscopic hemoclips for nonvariceal bleeding in the upper gastrointestinal tract. World J Gastroenterol. 2009;15:4322–6. doi: 10.3748/wjg.15.4322. [DOI] [PMC free article] [PubMed] [Google Scholar]
72.Rex DK, Lewis BS, Waye JD. Colonoscopy and endoscopic therapy for delayed post-polypectomy hemorrhage. Gastrointest Endosc. 1992;38:127–9. doi: 10.1016/s0016-5107(92)70376-9. [DOI] [PubMed] [Google Scholar]
73.Zuckerman GR, Prakash C. Acute lower intestinal bleeding: part I: clinical presentation and diagnosis. Gastrointest Endosc. 1998;48:606–17. doi: 10.1016/s0016-5107(98)70043-4. [DOI] [PubMed] [Google Scholar]
74.Barkun AN, Moosavi S, Martel M. Topical hemostatic agents: a systematic review with particular emphasis on endoscopic application in GI bleeding. Gastrointest Endosc. 2013;77:692–700. doi: 10.1016/j.gie.2013.01.020. [DOI] [PubMed] [Google Scholar]
75.Leung Ki EL, Lau JY. New endoscopic hemostasis methods. Clin Endosc. 2012;45:224–9. doi: 10.5946/ce.2012.45.3.224. [DOI] [PMC free article] [PubMed] [Google Scholar]
76.Binmoeller KF, Thonke F, Soehendra N. Endoscopic hemoclip treatment for gastrointestinal bleeding. Endoscopy. 1993;25:167–70. doi: 10.1055/s-2007-1010277. [DOI] [PubMed] [Google Scholar]
77.Hokama A, Uehara T, Nakayoshi T, et al. Utility of endoscopic hemoclipping for colonic diverticular bleeding. Am J Gastroenterol. 1997;92:543–6. [PubMed] [Google Scholar]
78.Kumar A, Artifon E, Chu A, et al. Effectiveness of endoclips for the treatment of stigmata of recent hemorrhage in the colon of patients with acute lower gastrointestinal tract bleeding. Dig Dis Sci. 2011;56:2978–86. doi: 10.1007/s10620-011-1683-1. [DOI] [PubMed] [Google Scholar]
79.Simpson PW, Nguyen MH, Lim JK, et al. Use of endoclips in the treatment of massive colonic diverticular bleeding. Gastrointest Endosc. 2004;59:433–7. doi: 10.1016/s0016-5107(03)02711-1. [DOI] [PubMed] [Google Scholar]
80.Yen EF, Ladabaum U, Muthusamy VR, et al. Colonoscopic treatment of acute diverticular hemorrhage using endoclips. Dig Dis Sci. 2008;53:2480–5. doi: 10.1007/s10620-007-0151-4. [DOI] [PubMed] [Google Scholar]
81.Farrell JJ, Graeme-Cook F, Kelsey PB. Treatment of bleeding colonic diverticula by endoscopic band ligation: an in-vivo and ex-vivo pilot study. Endoscopy. 2003;35:823–9. doi: 10.1055/s-2003-42611. [DOI] [PubMed] [Google Scholar]
82.Ishii N, Setoyama T, Deshpande GA, et al. Endoscopic band ligation for colonic diverticular hemorrhage. Gastrointest Endosc. 2012;75:382–7. doi: 10.1016/j.gie.2011.07.030. [DOI] [PubMed] [Google Scholar]
83.Setoyama T, Ishii N, Fujita Y. Enodoscopic band ligation (EBL) is superior to endoscopic clipping for the treatment of colonic diverticular hemorrhage. Surg Endosc. 2011;25:3574–8. doi: 10.1007/s00464-011-1760-8. [DOI] [PubMed] [Google Scholar]
84.Shibata S, Shigeno T, Fujimori K, et al. Colonic diverticular hemorrhage: the hood method for detecting responsible diverticula and endoscopic band ligation for hemostasis. Endoscopy. 2014;46:66–9. doi: 10.1055/s-0033-1344890. [DOI] [PubMed] [Google Scholar]
85.Barker KB, Arnold HL, Fillman EP, et al. Safety of band ligator use in the small bowel and the colon. Gastrointest Endosc. 2005;62:224–7. doi: 10.1016/s0016-5107(05)00557-2. [DOI] [PubMed] [Google Scholar]
86.Campo R, Brullet E, Montane JM, et al. Elastic band ligation in the bowel: is it really safe? Gastrointest Endosc. 1998;47:105–6. doi: 10.1016/s0016-5107(98)70309-8. [DOI] [PubMed] [Google Scholar]
87.Diggs NG, Holub JL, Lieberman DA, et al. Factors that contribute to blood loss in patients with colonic angiodysplasia from a population-based study. Clin Gastroenterol Hepatol. 2011;9:415–20. doi: 10.1016/j.cgh.2011.02.003. quiz e49. [DOI] [PMC free article] [PubMed] [Google Scholar]
88.Richter JM, Christensen MR, Colditz GA, et al. Angiodysplasia. Natural history and efficacy of therapeutic interventions. Dig Dis Sci. 1989;34:1542–6. doi: 10.1007/BF01537107. [DOI] [PubMed] [Google Scholar]
89.Sharma R, Gorbien MJ. Angiodysplasia and lower gastrointestinal tract bleeding in elderly patients. Arch Intern Med. 1995;155:807–12. [PubMed] [Google Scholar]
90.Kwan V, Bourke MJ, Williams SJ, et al. Argon plasma coagulation in the management of symptomatic gastrointestinal vascular lesions: experience in 100 consecutive patients with long-term follow-up. Am J Gastroenterol. 2006;101:58–63. doi: 10.1111/j.1572-0241.2006.00370.x. [DOI] [PubMed] [Google Scholar]
91.Olmos JA, Marcolongo M, Pogorelsky V, et al. Long-term outcome of argon plasma ablation therapy for bleeding in 100 consecutive patients with colonic angiodysplasia. Dis Colon Rectum. 2006;49:1507–16. doi: 10.1007/s10350-006-0684-1. [DOI] [PubMed] [Google Scholar]
92.Suzuki N, Arebi N, Saunders BP. A novel method of treating colonic angiodysplasia. Gastrointest Endosc. 2006;64:424–7. doi: 10.1016/j.gie.2006.04.032. [DOI] [PubMed] [Google Scholar]
93.Parra-Blanco A, Kaminaga N, Kojima T, et al. Colonoscopic polypectomy with cutting current: is it safe? Gastrointest Endosc. 2000;51:676–81. doi: 10.1067/mge.2000.105203. [DOI] [PubMed] [Google Scholar]
94.Bustamante-Balen M, Plume G. Role of hemostatic powders in the endoscopic management of gastrointestinal bleeding. World J Gastrointest Pathophysiol. 2014;5:284–92. doi: 10.4291/wjgp.v5.i3.284. [DOI] [PMC free article] [PubMed] [Google Scholar]
95.Holster IL, Brullet E, Kuipers EJ, et al. Hemospray treatment is effective for lower gastrointestinal bleeding. Endoscopy. 2014;46:75–8. doi: 10.1055/s-0033-1344988. [DOI] [PubMed] [Google Scholar]
96.Huang R, Pan Y, Hui N, et al. Polysaccharide hemostatic system for hemostasis management in colorectal endoscopic mucosal resection. Dig Endosc. 2014;26:63–8. doi: 10.1111/den.12054. [DOI] [PubMed] [Google Scholar]
97.Ibis M, Kurt M, Onal IK, et al. Successful management of bleeding due to solitary rectal ulcer via topical application of Ankaferd blood stopper. J Altern Complement Med. 2008;14:1073–4. doi: 10.1089/acm.2008.0314. [DOI] [PubMed] [Google Scholar]
98.Karaman A, Torun E, Gursoy S, et al. Efficacy of Ankaferd Blood Stopper in postpolypectomy bleeding. J Altern Complement Med. 2010;16:1027–8. doi: 10.1089/acm.2010.0089. [DOI] [PubMed] [Google Scholar]
99.Kurt M, Onal I, Akdogan M, et al. Ankaferd Blood Stopper for controlling gastrointestinal bleeding due to distinct benign lesions refractory to conventional antihemorrhagic measures. Can J Gastroenterol. 2010;24:380–4. doi: 10.1155/2010/896819. [DOI] [PMC free article] [PubMed] [Google Scholar]
100.Jayaraman V, Hammerle C, Lo SK, et al. Clinical Application and Outcomes of Over the Scope Clip Device: Initial US Experience in Humans. Diagn Ther Endosc. 2013;2013:381873. doi: 10.1155/2013/381873. [DOI] [PMC free article] [PubMed] [Google Scholar]
101.Anthony T, Penta P, Todd RD, et al. Rebleeding and survival after acute lower gastrointestinal bleeding. Am J Surg. 2004;188:485–90. doi: 10.1016/j.amjsurg.2004.07.020. [DOI] [PubMed] [Google Scholar]
102.Richter JM, Christensen MR, Kaplan LM, et al. Effectiveness of current technology in the diagnosis and management of lower gastrointestinal hemorrhage. Gastrointest Endosc. 1995;41:93–8. doi: 10.1016/s0016-5107(05)80588-7. [DOI] [PubMed] [Google Scholar]
103.Strate LL, Syngal S. Predictors of utilization of early colonoscopy vs radiography for severe lower intestinal bleeding. Gastrointest Endosc. 2005;61:46–52. doi: 10.1016/s0016-5107(04)02227-8. [DOI] [PubMed] [Google Scholar]
104.Ali M, Ul Haq T, Salam B, et al. Treatment of nonvariceal gastrointestinal hemorrhage by transcatheter embolization. Radiol Res Pract. 2013;2013:604328. doi: 10.1155/2013/604328. [DOI] [PMC free article] [PubMed] [Google Scholar]
105.Yi WS, Garg G, Sava JA. Localization and definitive control of lower gastrointestinal bleeding with angiography and embolization. Am Surg. 2013;79:375–80. [PubMed] [Google Scholar]
106.Khanna A, Ognibene SJ, Koniaris LG. Embolization as first-line therapy for diverticulosis-related massive lower gastrointestinal bleeding: evidence from a meta-analysis. J Gastrointest Surg. 2005;9:343–52. doi: 10.1016/j.gassur.2004.09.039. [DOI] [PubMed] [Google Scholar]
107.Yata S, Ihaya T, Kaminou T, et al. Transcatheter arterial embolization of acute arterial bleeding in the upper and lower gastrointestinal tract with N-butyl-2-cyanoacrylate. J Vasc Interv Radiol. 2013;24:422–31. doi: 10.1016/j.jvir.2012.11.024. [DOI] [PubMed] [Google Scholar]
108.Gunderman R, Leef JA, Lipton MJ, et al. Diagnostic imaging and the outcome of acute lower gastrointestinal bleeding. Acad Radiol. 1998;5(Suppl 2):S303–5. doi: 10.1016/s1076-6332(98)80338-3. [DOI] [PubMed] [Google Scholar]
109.Koh DC, Luchtefeld MA, Kim DG, et al. Efficacy of transarterial embolization as definitive treatment in lower gastrointestinal bleeding. Colorectal Dis. 2009;11:53–9. doi: 10.1111/j.1463-1318.2008.01536.x. [DOI] [PubMed] [Google Scholar]
110.Ng DA, Opelka FG, Beck DE, et al. Predictive value of technetium Tc 99m-labeled red blood cell scintigraphy for positive angiogram in massive lower gastrointestinal hemorrhage. Dis Colon Rectum. 1997;40:471–7. doi: 10.1007/BF02258395. [DOI] [PubMed] [Google Scholar]
111.Pennoyer WP, Vignati PV, Cohen JL. Mesenteric angiography for lower gastrointestinal hemorrhage: are there predictors for a positive study? Dis Colon Rectum. 1997;40:1014–8. doi: 10.1007/BF02050921. [DOI] [PubMed] [Google Scholar]
112.Zink SI, Ohki SK, Stein B, et al. Noninvasive evaluation of active lower gastrointestinal bleeding: comparison between contrast-enhanced MDCT and 99mTc-labeled RBC scintigraphy. AJR Am J Roentgenol. 2008;191:1107–14. doi: 10.2214/AJR.07.3642. [DOI] [PubMed] [Google Scholar]
113.Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. Gastrointest Endosc. 1999;49:228–38. doi: 10.1016/s0016-5107(99)70491-8. [DOI] [PubMed] [Google Scholar]
114.Dusold R, Burke K, Carpentier W, et al. The accuracy of technetium-99m-labeled red cell scintigraphy in localizing gastrointestinal bleeding. Am J Gastroenterol. 1994;89:345–8. [PubMed] [Google Scholar]
115.Hunter JM, Pezim ME. Limited value of technetium 99m-labeled red cell scintigraphy in localization of lower gastrointestinal bleeding. Am J Surg. 1990;159:504–6. doi: 10.1016/s0002-9610(05)81256-5. [DOI] [PubMed] [Google Scholar]
116.Funaki B, Kostelic JK, Lorenz J, et al. Superselective microcoil embolization of colonic hemorrhage. AJR Am J Roentgenol. 2001;177:829–36. doi: 10.2214/ajr.177.4.1770829. [DOI] [PubMed] [Google Scholar]
117.Yoon W, Jeong YY, Shin SS, et al. Acute massive gastrointestinal bleeding: detection and localization with arterial phase multi-detector row helical CT. Radiology. 2006;239:160–7. doi: 10.1148/radiol.2383050175. [DOI] [PubMed] [Google Scholar]
118.Bender JS, Wiencek RG, Bouwman DL. Morbidity and mortality following total abdominal colectomy for massive lower gastrointestinal bleeding. Am Surg. 1991;57:536–40. discussion 540–1. [PubMed] [Google Scholar]
119.Farner R, Lichliter W, Kuhn J, et al. Total colectomy versus limited colonic resection for acute lower gastrointestinal bleeding. Am J Surg. 1999;178:587–91. doi: 10.1016/s0002-9610(99)00235-4. [DOI] [PubMed] [Google Scholar]
120.Yuhara H, Corley DA, Nakahara F, et al. Aspirin and non-aspirin NSAIDs increase risk of colonic diverticular bleeding: a systematic review and meta-analysis. J Gastroenterol. 2014;49:992–1000. doi: 10.1007/s00535-013-0905-z. [DOI] [PubMed] [Google Scholar]
121.Aoki T, Nagata N, Niikura R, et al. Recurrence and mortality among patients hospitalized for acute lower gastrointestinal bleeding. Clin Gastroenterol Hepatol. 2015;13:488–494. e1. doi: 10.1016/j.cgh.2014.06.023. [DOI] [PubMed] [Google Scholar]
122.Nagata N, Niikura R, Aoki T, et al. Impact of discontinuing non-steroidal antiinflammatory drugs on long-term recurrence in colonic diverticular bleeding. World J Gastroenterol. 2015;21:1292–8. doi: 10.3748/wjg.v21.i4.1292. [DOI] [PMC free article] [PubMed] [Google Scholar]
123.Becker RC, Scheiman J, Dauerman HL, et al. Management of platelet-directed pharmacotherapy in patients with atherosclerotic coronary artery disease undergoing elective endoscopic gastrointestinal procedures. Am J Gastroenterol. 2009;104:2903–17. doi: 10.1038/ajg.2009.667. [DOI] [PubMed] [Google Scholar]
124.Sung JJ, Lau JY, Ching JY, et al. Continuation of low-dose aspirin therapy in peptic ulcer bleeding: a randomized trial. Ann Intern Med. 2010;152:1–9. doi: 10.7326/0003-4819-152-1-201001050-00179. [DOI] [PubMed] [Google Scholar]
125.Derogar M, Sandblom G, Lundell L, et al. Discontinuation of low-dose aspirin therapy after peptic ulcer bleeding increases risk of death and acute cardiovascular events. Clin Gastroenterol Hepatol. 2013;11:38–42. doi: 10.1016/j.cgh.2012.08.034. [DOI] [PubMed] [Google Scholar]
126.Eisenberg MJ, Richard PR, Libersan D, et al. Safety of short-term discontinuation of antiplatelet therapy in patients with drug-eluting stents. Circulation. 2009;119:1634–42. doi: 10.1161/CIRCULATIONAHA.108.813667. [DOI] [PubMed] [Google Scholar]
127.Ho PM, Peterson ED, Wang L, et al. Incidence of death and acute myocardial infarction associated with stopping clopidogrel after acute coronary syndrome. Jama. 2008;299:532–9. doi: 10.1001/jama.299.5.532. [DOI] [PubMed] [Google Scholar]
128.Aytac E, Stocchi L, Gorgun E, et al. Risk of recurrence and long-term outcomes after colonic diverticular bleeding. Int J Colorectal Dis. 2014;29:373–8. doi: 10.1007/s00384-013-1804-8. [DOI] [PubMed] [Google Scholar]
129.Swanson E, Mahgoub A, MacDonald R, et al. Medical and endoscopic therapies for angiodysplasia and gastric antral vascular ectasia: a systematic review. Clin Gastroenterol Hepatol. 2014;12:571–82. doi: 10.1016/j.cgh.2013.08.038. [DOI] [PubMed] [Google Scholar]
130.Okamoto T, Watabe H, Yamada A, et al. The association between arteriosclerosis related diseases and diverticular bleeding. Int J Colorectal Dis. 2012;27:1161–6. doi: 10.1007/s00384-012-1491-x. [DOI] [PubMed] [Google Scholar]
131.Strate LL, Liu YL, Aldoori WH, et al. Obesity increases the risks of diverticulitis and diverticular bleeding. Gastroenterology. 2009;136:115–122. e1. doi: 10.1053/j.gastro.2008.09.025. [DOI] [PMC free article] [PubMed] [Google Scholar]
132.Suzuki K, Uchiyama S, Imajyo K, et al. Risk factors for colonic diverticular hemorrhage: Japanese multicenter study. Digestion. 2012;85:261–5. doi: 10.1159/000336351. [DOI] [PubMed] [Google Scholar]
133.Yamada A, Sugimoto T, Kondo S, et al. Assessment of the risk factors for colonic diverticular hemorrhage. Dis Colon Rectum. 2008;51:116–20. doi: 10.1007/s10350-007-9137-8. [DOI] [PubMed] [Google Scholar]
134.Laine L, Connors LG, Reicin A, et al. Serious lower gastrointestinal clinical events with nonselective NSAID or coxib use. Gastroenterology. 2003;124:288–92. doi: 10.1053/gast.2003.50054. [DOI] [PubMed] [Google Scholar]
135.Laine L, Curtis SP, Langman M, et al. Lower gastrointestinal events in a double-blind trial of the cyclo-oxygenase-2 selective inhibitor etoricoxib and the traditional nonsteroidal anti-inflammatory drug diclofenac. Gastroenterology. 2008;135:1517–25. doi: 10.1053/j.gastro.2008.07.067. [DOI] [PubMed] [Google Scholar]
136.Nagata N, Niikura R, Aoki T, et al. Lower GI bleeding risk of nonsteroidal anti-inflammatory drugs and antiplatelet drug use alone and the effect of combined therapy. Gastrointest Endosc. 2014 doi: 10.1016/j.gie.2014.06.039. [DOI] [PubMed] [Google Scholar]
137.Casado Arroyo R, Polo-Tomas M, Roncales MP, et al. Lower GI bleeding is more common than upper among patients on dual antiplatelet therapy: long-term follow-up of a cohort of patients commonly using PPI co-therapy. Heart. 2012;98:718–23. doi: 10.1136/heartjnl-2012-301632. [DOI] [PubMed] [Google Scholar]
138.Abraham NS, Hartman C, Richardson P, et al. Risk of lower and upper gastrointestinal bleeding, transfusions, and hospitalizations with complex antithrombotic therapy in elderly patients. Circulation. 2013;128:1869–77. doi: 10.1161/CIRCULATIONAHA.113.004747. [DOI] [PubMed] [Google Scholar]
139.Bhatt DL, Scheiman J, Abraham NS, et al. ACCF/ACG/AHA 2008 expert consensus document on reducing the gastrointestinal risks of antiplatelet therapy and NSAID use. Am J Gastroenterol. 2008;103:2890–907. doi: 10.1111/j.1572-0241.2008.02216.x. [DOI] [PubMed] [Google Scholar]
140.Manocha D, Singh M, Mehta N, et al. Bleeding risk after invasive procedures in aspirin/NSAID users: polypectomy study in veterans. Am J Med. 2012;125:1222–7. doi: 10.1016/j.amjmed.2012.05.030. [DOI] [PubMed] [Google Scholar]
141.Antithrombotic Trialists C. Baigent C, Blackwell L, et al. Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials. Lancet. 2009;373:1849–60. doi: 10.1016/S0140-6736(09)60503-1. [DOI] [PMC free article] [PubMed] [Google Scholar]