Learning Objectives
For patients with severe hematochezia:

- To recognize and describe the most common colonic causes in adult patients hospitalized because of severe hematochezia and contrast the causes for those patients who develop inpatient hematochezia.
- To recommend new management algorithms for severe hematochezia.
- To distinguish and describe definitive, presumptive, and incidental diverticulosis in patients with severe hematochezia.
- To describe the vascular anatomy of diverticular hemorrhage, stigmata of recent hemorrhage (SRH), and rebleeding risks of patients with different SRH managed medically and endoscopically.
- To describe colonoscopic diagnosis and hemostasis for other bleeding colon lesions.

Introduction
“Lower (L)GI bleeding” in this presentation is limited to sources of bleeding from the colon or anus. The presentation is red blood or clots of any shade of red color passed per rectum. This contrasts with upper (U)GI or small intestinal bleeding, both of which can cause severe hematochezia and can masquerade as colon bleeding. Neither UGI nor small intestinal sources or causes of bleeding will be discussed in detail in this presentation.

The annual incidence of severe colorectal bleeding is about 20 per 100,000 population but is increased in the elderly. Severe hematochezia that is witnessed by a physician or nurse and is severe enough to result in hospitalization or occurs during hospitalization is the focus of this presentation. Considering the spectrum of colonic GI bleeding from hemoccult positive stools (with or without anemia) to BRB, severe hematochezia is uncommon and accounts for less than 5-10% of this group. However, severe hematochezia is increasing in prevalence as the adult patient population ages, colonoscopic diagnoses and treatments expand, and non-steroidal anti-inflammatory drugs (NSAIDs), anti-platelet drugs (e.g., aspirin [ASA]), and anti-coagulants (e.g., warfarin and newer drugs) increase in utilization. Severe hematochezia is also a common presentation for severe GI bleeding in inpatients with severe co-morbidities who start bleeding after hospitalization for an unrelated medical or surgical reason but can bleed from a focal or diffuse lesion anywhere in the GI tract, most commonly from the UGI tract or colon.

A new approach and algorithm to manage patients with severe hematochezia is similar to the algorithm of patients with severe upper GI hemorrhage now used worldwide. After cleansing the colon well with an oral purge, this combines urgent diagnosis of patients with hematochezia with colonoscopic treatment and focal lesions at urgent colonoscopy.

The advantages and disadvantages of traditional approaches to diagnosis and treatment of severe hematochezia (e.g., RBC scanning or angiography) compared to the new approach of urgent colonoscopy after purge or urgent capsule endoscopy are summarized in Table 1. The advantages, safety, and results of urgent colonoscopy for diagnosis and treatment of patients with severe hematochezia are the reasons to choose this approach.

Methods
CURE Hemostasis Group General Management of the Patient
While they are being resuscitated by the managing physicians, patients hospitalized for severe hematochezia should have consultation by a gastroenterologist (who can skillfully perform urgent colonoscopy and hemostasis after purge). A general surgeon is consulted upon only in selected high risk patients who develop hematochezia as inpatients or who may require urgent surgery because of continued hematochezia or hypotension despite ongoing resuscitation. A careful medical history and physical examination are fundamental for clues to the appropriate urgency of resuscitation, differential diagnosis, and bleeding lesion location. Use of large bore intravenous catheters and administration of fluids and blood to reverse hypovolemia, correct coagulopathies, and treat severe co-morbid conditions are all required. These are usually accomplished in a monitored bed or intensive care unit setting (Figure 1).

A gastric tube (e.g., nasogastric or orogastric) and gastric lavage are recommended in any patient with ongoing hematochezia and no history of hematemesis (e.g., vomiting red blood or coffee grounds). A clear lavage (without bile) is non-diagnostic, whereas fresh bile indicates continuity with the second portion of the duodenum and is a negative examination, if done in patients with ongoing or severe bleeding. This is particularly important in patients with inpatient development of hematochezia and any patient with cirrhosis.
and/or patients with a history of UGI bleeding. For a patient with a negative gastric lavage, purging with a PEG-based preparation (such as Golytely® or Colyte®) is recommended prior to urgent colonoscopy. However, those patients with a history of cirrhosis, prior UGI hemorrhage, a positive NG aspirate, or a history of melena or hematemesis in the last month—including those with these signs unwitnessed—should have an urgent EGD or push enteroscopy first to exclude a foregut source and treat it. Refer to Figure 2 for a summary of our management algorithm for severe hematochezia.

CURE Hemostasis Group’s Results Using New Hematochezia Algorithm

Refer to Figure 3 for the localization of sites of hemorrhage (presumptive: nothing else seen; or definitive: stigmata of hemorrhage [SRH] found).

These are the recent results when the CURE Hemostasis Group utilized this management algorithm for 795 consecutive patients with severe hematochezia. Most patients presented with hematochezia that started as an outpatient or at home.

However, the findings were different when patients with start of hematochezia as an inpatient (“inpatient hematochezia”) were analyzed (Table 2).

For all hematochezia patients, the eight most common lesions are shown in Table 3. Those found with focal lesions and SRH are checked.

Diverticular Hemorrhage: Acute Diagnosis and Treatment

For working definitions about diverticulosis and hematochezia, “incidental diverticulosis” is when colonic diverticulosis is present but another type of lesion is documented to cause the bleeding (such as angiomasa); “presumptive diverticular hemorrhage” is defined when diverticulosis without stigmata of hemorrhage is seen at colonoscopy and no other bleeding sites are identified by colonoscopy, anoscopy, and enteroscopy; and “definitive diverticular hemorrhage” is diagnosed when a major stigmata of hemorrhage (active bleeding, non-bleeding visible vessel [NBVV], or adherent clot) is found on a diverticulum by colonoscopy and/or angiography. The most recent calculations for the prevalence of these subgroups in 405 consecutive patients with colon diverticulosis and hematochezia are: incidental diverticulosis in 47.2% (some other cause of bleeding), presumptive diverticular bleed in 31.9%, and definitive diverticular hemorrhage in 21.0%.

Therefore, for patients whom we diagnosed as true diverticular hemorrhage based upon an urgent colonoscopy/enteroscopic approach (with or without angiography), 40% had active bleeding or another major stigmata of recent hemorrhage (“definitive” diverticular hemorrhage). In contrast, about 60% were presumptive diverticular hemorrhage. As the experience of the CURE Hemostasis Group has increased and the colon preparation for urgent colonoscopies has improved with training of hospital staff, the prevalence of definitive diverticular bleeding has increased from about 30% to 40% over the last decade. Target irrigation, hemoclips, caps to evert diverticula when the SRH is in the base, and large channel, small caliber colonoscopes have facilitated urgent colonoscopy, including
finding and treating diverticular SRH. Incomplete preps (usually from inadequate volume of purge), delay of colonoscopy more than 24 hours after the bleeding stops, and inexperienced colonscopists are the main reasons for low yields with this approach for diagnosis of definitive diverticular hemorrhage and other focal colonic lesions.

For 100 consecutive cases of definitive diverticular hemorrhage diagnosed by the CURE Hemostasis Group during urgent colonoscopy after purge and prospectively followed, the prevalence of different SRH were active bleeding in 30%, adherent clot in 42%, and non-bleeding visible vessel (NBVV) in 28%. The CURE Hemostasis Group also prospectively studied the natural history of bleeding for patients with major SRH who were managed with medical therapy alone, without colonoscopic hemostasis. For 37 patients with definitive diverticular hemorrhage based upon these SRH, the rebleeding and subsequent intervention rates for bleeding (e.g., surgery or angiographic embolization except in one patient who had repeat urgent colonoscopy and colonoscopic hemostasis) have been reported. The rates of rebleeding and intervention for active bleeding diverticula within 30 days were 83% and 56%, respectively; for non-bleeding visible vessel were 60% and 40%, respectively; and for adherent clot were 43% and 29%, respectively. For all 37 patients with these three different major SRH of diverticular hemorrhage, the rate of rebleeding was 65% and intervention for rebleeding was 43% within 30 days from the same diverticulum. These rates are significantly higher than peptic ulcer hemorrhage for similar SRH, probably because no drugs such as PPIs are currently available to reduce rebleeding rates as with ulcer hemorrhage.

The locations of arteries and the arcade formed in a diverticulum are shown in Figure 4. Arteries that are submucosal and subserosal unite to form the artery in the base of the diverticulum. In recent CURE studies, a colonoscopic Dop-
pler ultrasound probe detected arterial blood flow in 90% of patients (18/20) with major SRH and also could map the direction of the artery. In contrast, none of the patients (0/20) with presumptive diverticular bleeding and arteries interrogated in the bases of non-bleeding diverticula had arterial flow detected. Based upon these vascular anatomic findings and Doppler flow studies, the colonoscopic treatment now recommended is either hemoclipping (for SRH at the base or neck of the diverticulum) or multipolar probe thermal coagulation (at the neck) and treatment on either side of the SRH to seal the underlying vessel. Rechecking for residual arterial blood flow after such visually directed hemostasis and further treatment if the underlying artery has not been obliterated are also recommended. These techniques have been used routinely in the last three years and now the rebleeding rates after colonoscopic combination therapies (often using 1:20,000 epinephrine injection first followed by MPEC or hemoclipping) have reduced the diverticular rebleeding rates to less than 5%.

Several authors have reported over the last decade that patients with severe hematochezia can be effectively and safely managed with a strategy of thorough colon cleansing with oral or NG tube purge before urgent colonoscopy by experienced colonoscopists with hemostasis of focal lesions, risk stratification, and triage of patients to appropriate level of hospital care or early discharge. This strategy has been reported to maximize outcomes and minimize risks compared to traditional management of watchful waiting, RBC scanning or angiography, or urgent surgery. This is particularly evident for high-risk patients with severe hematochezia where improvements in routine outcomes, reduction in direct costs of hospitalization, and decreased length of hospital stay have been reported for patients having early colonoscopy. Similar to non-variceal UGI hemorrhage, most patients with this approach will stop bleeding on their own but others will require urgent definitive hemostasis for high-risk SRH or chronically bleeding lesions. However, nearly all patients evaluated with this management algorithm will have a specific diagnosis made and the information from urgent colonoscopy and other endoscopic tests can be utilized to individualize acute and long-term management.

Conclusions

Severe hematochezia is a common reason for hospitalization but the causes have changed in the last decade. Changes are in part related to the aging of the population, the evaluation and treatment by experienced colonoscopists rather than by surgeons or radiologists, and the changes in colonoscopic practices such as utilization of urgent colon purge and newer accessories, including hemoclips, caps for the tip of the colonoscope, and more recently Doppler ultrasound probe and capsule endoscopy.

A careful history, physical examination, rectal examination, and NG or orogastric lavage may help localize the bleeding site and focus the differential diagnosis. This should be routine while resuscitating the patient with cirrhosis, a history of UGI hemorrhage, and those with a history of melena or hematemesis within the last month. However, purging the colon for urgent colonoscopy is currently the most definitive way to combine diagnosis and treatment of bleeding colonic lesions. Complete colonoscopy with intubation of the terminal ileum is recommended and also examination of the rectum with a slotted anoscope to exclude internal hemorrhoids and fissures. If those examinations are negative, a push enteroscopy is recommended for combined diagnosis and treatment. Finally, if no foregut or colorectal lesion is found as the definitive or presumptive diagnosis, capsule endoscopy is recommended to exclude a small bowel source of severe hematochezia. Also, urgent capsule endoscopy in patients presenting to the hospital with ongoing hematochezia should be studied further as a potential replacement of RBC scanning or angiography, especially for lesion localization in patients without UGI hemorrhage. Capsule endoscopy appears to be more sensitive because it can detect lesions, bleeding, and non-bleeding SRH.

With recent advances in colonoscopic hemostasis, patients will have their outcomes maximized and risk minimized from early diagnosis and treatment by experienced colonoscopists. Also, urgent colonoscopy after purge has been reported to be more cost effective than elective colonoscopy or other strategies for diagnosis of severe hematochezia.
SELECTED REFERENCES


