EDITORIAL

The ins and outs of diverticular bleeding CME

I have several goals in this editorial. The first is to compliment Dr. Ishii and his colleagues for their interest in diverticular hemorrhage and for reporting a large cumulative experience of patients with definitive diverticular hemorrhage from Japan.[1] The diagnosis of definitive diverticular hemorrhage made during urgent colonoscopy is based on finding major stigmata of recent hemorrhage (SRH) in a diverticulum (ie, active bleeding, a nonbleeding visible vessel, or an adherent clot), as my CURE Hemostasis Research Group previously reported.[2] The second goal is to review the vascular anatomy of colonic diverticula and provide some insight on blood flow related to SRH of diverticular hemorrhage with Doppler US probe, which is very useful in risk stratification, deciding where to treat, and documenting complete endoscopic hemostasis.[3] The third is to clarify several misconceptions about diverticular hemorrhage that are common in the medical-surgical literature. The last is to offer colonoscopists some practical recommendations about colonoscopic diagnosis and hemostasis of patients with severe hematochezia and a presumed diagnosis of diverticular hemorrhage.[2,4,5] For a critical review of the topic and as an evidence-based approach, these are important for both the readers of the current report and reviewers of future articles about severe hematochezia and how the diagnosis of diverticular hemorrhage is made and treated.

The prognostic value for risk stratification and the natural history of colonic SRH in definitive diverticular hemorrhage have not been reported very often, except by my group.[2,4-6] The short-term natural history of documented definitive diverticular hemorrhage colonic has not changed since our original study in which 17 medically managed patients with major SRH and definitive diverticular hemorrhage had a 30-day rebleed rate of 53% with at least 2 more red blood cell transfusions (in addition to baseline), and 35% required emergency surgery.[2] Now with more than twice this total number of patients treated medically, the 30-day rebleed rate is still 54% and the need for surgery or interventional radiology (IR) embolization is 35%. Unlike peptic ulcer bleeding in which medical therapy (eg, high-dose proton pump inhibitors) can reduce the prevalence of major SRH as pretreatment before endoscopy and significantly decrease rebleeding rates after successful endoscopic hemostasis,[5] no medical therapy is specific or effective for the prevention of diverticular rebleeding. Definitive diverticular hemorrhage is a serious clinical problem, especially in elderly patients with significant comorbidities in which the rates of more bleeding are high with medical therapy.[2,4-6]

Whereas 2 decades ago, the management was primarily by general surgeons and interventional radiologists, now the GI colonoscopist can diagnose and effectively treat most patients successfully.

SRH during urgent colonoscopy is less frequently seen and more difficult to diagnose than SRH in peptic ulcers because vigorous preparation of the colon to clear it of clots, blood, and stool, early urgent colonoscopy, and careful, frequent inspection of many diverticula are required.[2,4-7] Identification of the bleeding site with the SRH and exclusion of other lesions facilitates classification of a patient with colon diverticulosis and severe hematochezia as having either presumptive diverticular hemorrhage (eg, no other source found besides diverticulosis after urgent colonoscopy, anoscopy, push enteroscopy, and capsule endoscopy), incidental diverticulosis (eg, some other nondiverticular colon, anorectal, upper GI or small-bowel source identified as the cause of the bleeding) or definitive diverticular hemorrhage.[2,4-6] I recommend that authors include these prevalences in their reports to give the reader perspective on comparative rates of identifying SRH and definitive diverticular hemorrhage. It is a major misconception to attribute diverticulosis as the bleeding source in all patients with colon diverticulosis who present for the first time with severe hematochezia. The CURE Hemostasis Group prospectively studied 340 patients with colon diverticulosis and severe hematochezia. Incidental diverticulosis was diagnosed in 46% (ie, some other nondiverticular GI source was found), presumptive diverticular hemorrhage in 34%, and definitive diverticular hemorrhage in 20%.[4,5] In other words, for true diverticular bleeding (as opposed to incidental diverticulosis), 37% patients had SRH on urgent colonoscopy (eg, definitive diverticular hemorrhage) and 63% do not (eg, presumptive). Ishii et al

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do not report the rates of incidental diverticulosis in their patients with severe hematochezia.[1] However, on urgent colonoscopy, our rates of diagnosis of definitive and presumptive diverticular hemorrhage are very similar to their Japanese series.[1,2,4,5] Another misconception is about the location of the SRH in the colon for heterogeneous populations compared with Asian patients with definitive diverticular hemorrhage. They also appear to be very similar. Specifically, for more than 85 patients with definitive diverticular hemorrhage whom we studied prospectively, the SRH was located in a colonic diverticulum at or proximal to the splenic flexure in 63%, although anatomically more than 70% of colonic diverticula were distal to the splenic flexure in our studies.[2,4-6,8] Based on these reports, there appear to be no significant differences in locations of SRH in the colon, prevalence of the definitive subgroup in true diverticular hemorrhage, or severity of definitive diverticular hemorrhage in Asians compared with heterogeneous patients with severe hematochezia.[1,2,4-6,8]

The pathophysiology of diverticular bleeding is not understood, although risk factors were advanced age (older than 70 years), black versus white ethnicity, and use of aspirin in a recent report.[9] At the SRH in the diverticulum is where the underlying arterial blood flow is detectable by Doppler probe, whereas it is not detectable more than 3 to 4 mm on either side of the SRH even though the artery may be visible.[3] Effective, targeted colonoscopic treatment obliterates the blood flow, prevents rebleeding, and improves clinical outcomes compared with medical therapy.[3]

The location of the SRH in a diverticulum is of paramount importance to the colonoscopist. In our prospective studies of definitive diverticular hemorrhage, the SRH was in the neck about 50% of the time and in the base about as frequently, although active bleeding was more common in the base and nonbleeding visible vessel in the neck.[4,8] Based on definitive endoscopic treatment (with multipolar electrocoagulation [MPEC]), laterally directed coagulation using moderate tamponade directly on the SRH at the neck of the diverticulum (which is thick) is currently the fastest, easiest, and most effective method we have found for hemostasis in this location.[4,5,8] Because of the theoretical risk of perforation with thermal coagulation in the base of the diverticulum, I now recommend hemoclipping (HC) of SRH there, with a new-generation HC that can grasp, release, and reopen before deployment.[8] If there is active bleeding or an adherent clot, preinjection of dilute epinephrine (1:20,000 in saline solution) is recommended to slow the bleeding or, for the clot, before cold guillotining to shave it down to a short pedicle and before HC in the base or MPEC at the neck of the diverticulum.[4,5] When SRH of definitive diverticular hemorrhage were studied with a Doppler probe before treatment, about 90% had arterial blood flow detected and the path of flow could be traced for 2 to 3 mm on either side of the SRH. Blood flow was obliterated by successful MPEC or HC, but not epinephrine alone.[3] After hemostasis, I recommend tattooing the mucosa in 3 to 4 areas adjacent to the diverticulum with the SRH in case of rebleeding or resection.[4] Hemoclips for definitive diverticular hemorrhage fall off within days and are not an effective marking technique.

Targeted treatment at the SRH during urgent colonoscopy or embolization by IR of the underlying artery usually results in definitive hemostasis of definitive diverticular hemorrhage, and surgery is not necessary.[1-6] Because there are arcades of arteries both submucosally and below the muscularis propria that unite to form the artery that can be seen at colonoscopy in the base of the diverticulum, SRH represents a rent in an underlying artery, and there is bidirectional blood flow, such targeted colonoscopic or IR treatment is necessary to obliterate the artery with the SRH and to achieve definitive nonsurgical hemostasis of definitive diverticular hemorrhage.[3-6,8] Treating distantly or away from the SRH during urgent colonoscopy or having the underlying artery remain patent, as in some of the cases in the Ishii et al study, where the bands came off often results in early and severe rebleeding.[1]

Rubber-band ligation (RBL) as endoscopic treatment for nonvariceal GI bleeding has been used for small ulcers, Dieulafoy's lesion, and diverticulosis.[5] The hypothesis is that the band will be deployed into the submucosa after suctioning a pedicle of compliant tissue and will cause focal ischemia and thrombosis of the artery, ulceration, and scarring. An example is shown in their figure showing the histology of a banded and resected diverticulum.[1] With a colonic diverticulum, which has a very thin submucosa and no muscle layer in the base, this might seem risky. However, as reviewed by Ishii et al, several groups have reported good safety profiles with RBL and no perforations in the treatment of definitive diverticular hemorrhage. However, suctioning of adequate tissue in a diverticulum is not always possible. In the Ishii et al series, the initial failure rate was 13% and the early rebleeding rate was 11%, for an overall failure rate of RBL for initial hemostasis of 24%.[1] In contrast, for the last 49 patients whom the CURE group treated for definitive diverticular hemorrhage with either MPEC or HC, there were no initial treatment failures and the 30-day rebleed rate was 12%, with all these after restart of anticoagulants for severe comorbid conditions. Only 6% required IR or surgery.[2-6] Before the availability of newer HCs, MPEC was used for all patients without perforations, and the only complication was 1 case of postcoagulation syndrome after SRH treatment in a diverticular base.[4,8]

The primary advantages of through-the-colonoscope treatments (such as MPEC and HC) are the rapidity, targeted nature of treatments, high primary and permanent hemostasis rates, convenience to the endoscopist, and low complication rate. The high success rate depends on the recognition of and finding SRH, selection of the targeted treatment according to location in the diverticulum (neck or base), and understanding the anatomy as well as the