Colonic Diverticulosis and Diverticular Hemorrhage

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ABSTRACT

Colonic diverticulosis predisposes individuals to lower gastrointestinal hemorrhage in up to 5% of cases. These sac-like protrusions are pseudodiverticula and arise due to a combination of anatomic, dietary, motility, and structural influences. In the setting of acute hemorrhage, patient stabilization takes priority, followed closely by maneuvers aimed at localizing and controlling blood loss. Through the use of an arsenal of tools including colonoscopy, angiography, and nuclear scintigraphy, most diverticular bleeds can be localized and subsequently controlled. When persistent and not controlled by colonoscopic or angiographic means, expeditious surgical resection serves as definitive therapy.

KEYWORDS: Diverticulosis, gastrointestinal hemorrhage, diverticulitis

Objectives: Upon completion of this article, the reader should have a clear understanding of the evaluation and management of patients with diverticular hemorrhage.

The presence of colonic diverticula, sac-like protrusions from the wall of the intestine, predisposes patients to the development of associated complications from the diverticula in 15 to 30% of cases. Diverticular bleeding represents a significant complication often associated with massive lower gastrointestinal hemorrhage. The acute hemorrhage presents diagnostic and management challenges in these patients. Successful therapy requires an understanding of the pathophysiology of the disease process coupled with a well-designed management protocol. This article focuses on the issues specifically related to diverticular hemorrhage.

EPIDEMIOLOGY

Colonic diverticular disease seems to be a phenomenon of industrialized, Western societies. Incidence is related to the age of the population studied, with an incidence of less than 2% in populations under age 30, 50% in pa-

tients over 50, and 70% in patients over age 80.^{1,2} Comparing populations, it has been noted that overall incidence is directly proportional to population-wide fiber intake.^{3,4} In Western nations, the incidence appears to be rising, while in populations where people consume high-fiber diets, such as in Japan or Africa, diverticular disease is almost nonexistent, affecting less than 0.2% of patients.³ Hemorrhage complicates 3 to 5% of patients with diverticulosis.⁵ Given the prevalence of colonic diverticulosis and the fact that most episodes of hemorrhage tend to cease spontaneously, many episodes of lower intestinal hemorrhage are attributed to colonic diverticulosis as a presumptive rather than a definitive diagnosis.

ANATOMY

The normal colon wall consists of mucosa, submucosa, and the muscularis propria. The muscular wall has an

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inner circular layer that forms a continuous sheath around the bowel. The outer longitudinal layer is concentrated in three narrow bands, the taenia coli. Generally, the colon wall distributes the three taeniae as two antimesenteric and one mesenteric linear muscular bands. Normally, both the longitudinal and circular muscular layers of the colon become thicker more distally throughout the large intestine. The taeniae eventually splay across the rectum comprising its entire circumference as a fusion of the three components at the recto-sigmoid junction.

Most anatomists divide diverticula into two general types—true and false. True diverticula involve all three layers of the bowel wall, the mucosa, submucosa, and muscularis externa, as seen in most congenital diverticula such as Meckel's diverticula. False diverticula, or "pseudodiverticula," are composed of mucosa and submucosa protruding through the muscularis externa, covered only by serosa. The majority of the colon diverticula are false diverticula and arise from weak points in the colonic wall where mesenteric blood vessels penetrate the circular muscle layer, typically along the mesenteric side of the antimesenteric taeniae (Fig. 1). Some have theorized that the pseudodiverticula represent pulsion diverticula acquired in the sigmoid colon. The increased intraluminal pressure may correlate with slow transit and segmentation within areas such as the sigmoid colon.

Figure 1 Cross-section of colon. Cross-sectional drawing of the colon showing principal points of diverticular formation between mesenteric and antimesenteric teniae. From Beck DE, Opelka FG. Diverticular disease. In: Beck DE, ed. *Handbook of Colorectal Surgery*. 2nd ed. New York, NY: Marcel Dekker; 2003: 241–258. Reprinted with permission.

The majority (65%) of colonic diverticula are located in the sigmoid colon. While 35% of patients can have diverticula localized to other colonic segments, they may be scattered throughout the entire colon in 5 to 10% of patients.¹ In Far Eastern populations, there is higher incidence of right-sided colonic diverticula, likely due to either a genetic predisposition or dietary habits.

COLONIC BLOOD SUPPLY

Understanding the vascular supply to the colon and the collateral blood flow assists in defining a source of hemorrhage and in the ultimate control of the site of continued bleeding. The major arteries that deliver blood to the colon arise directly from the abdominal aorta. The superior mesenteric artery first supplies the pancreas and small bowel, subsequently giving rise to the major branches supplying the right colon, the ileocolic, right colic, and middle colic arteries. The inferior mesenteric artery (IMA) arises 3 to 4 cm above the aortic bifurcation, close to the inferior border of the duodenum. It branches into the left colic artery, multiple sigmoid branches, and the superior rectal artery. The IMA and its branches supply the large intestine from the distal transverse colon to the proximal rectum. The three major intestinal branches typically form a series of primary and secondary arcades. The terminal arcades give rise to numerous straight vessels, the vasa recti, which enter the intestinal wall along the taenia and create the submucosal vascular plexus. Two major collaterals secure connections between the superior mesenteric artery and IMA, via the marginal artery of Drummond (parallel to the colonic wall) and the arc of Riolan (at the base of the mesentery).

PATHOGENESIS OF DIVERTICULUM FORMATION

Diet

Diverticulosis has been described as a disease of Westernized, industrialized civilizations. The most consistent factor associated with this increased incidence is a decreased intake of dietary fiber.³ This is supported by the fact that colonic diverticulosis is extremely rare in populations that consume high-fiber diets, such as rural Africa and Asia, as well as by the fact that vegetarians tend to have a lower incidence of diverticular disease. In addition, populations who either move from rural to urban environments or simply adopt a Western lifestyle experience an increase in the prevalence of diverticular disease, although with a predominance of right-sided lesions.⁶

The diet determines the intestinal contents and mechanically affects colonic structure and motility. Patients in countries where high-fiber diets are consumed

have large-bore colons, while the colons of Western-fed patient tend to be of a reduced diameter.⁷ The development of diverticula can be explained by the Law of Laplace, as pressure P is proportional to wall tension T and inversely proportional to bowel radius R :

$$P = kT/R$$

Accordingly, it takes a greater intraluminal pressure to propel feces through a narrow colon, favoring the development of diverticula.

Colonic Motility

The pathogenesis of colonic diverticulosis appears to be a function of the complex nature of colonic motility. Segmentation is a motility process in which segmental muscular contractions separate the lumen in specific chambers.⁸ These segmental contractions of the colon serve to slow the fecal stream, allowing time for water absorption and electrolyte exchange while the infrequent peristaltic contractions transport the fecal mass in a caudal direction. These high-amplitude propagated contractions that occur on average six times a day result in pressure alterations which exert a substantial lateral force, as high as 90 mm Hg, on the colonic wall.⁹ These repeated high-pressure contractions predispose to mucosal herniation at areas of weakness in the colonic wall (Fig. 2).

Structural Aspects

In addition, there appears to be a pathologic thickening of the muscular layer of the colon in cases of diverticulosis. Elastin within the taenia appears to be laid down in a contracted state, retaining their shortened position while leading to the haustral folds found throughout the colon. With a combination of elastosis

and thickening, the colon forms the characteristic concertina-like folds of the inner muscular layer, leading to close apposition of the serosa. As the vasa recti penetrate the muscular layers of the colonic wall at these sites, a source for potential breach in the colonic wall integrity is created, facilitating diverticula formation.

Senescent deteriorations contribute to the incidence of colonic diverticulosis with increasing age. Currently, disruptions in colonic wall collagen and metalloproteinases may contribute further to development of these pulsion diverticula. This may be related to a reduction in the tensile strength of the colonic wall. Collagen fibrils become smaller, more numerous, and more tightly packed, particularly in the left colon. Although there is a constant collagen content in the colonic wall, qualitative changes lead to changes in compliance and distensibility over time.¹⁰

PATHOGENESIS OF DIVERTICULAR BLEEDING

The various colonic arterioles penetrate the muscular wall en route to the colonic mucosa. Sometime these arterioles can divide, with one branch penetrating the wall at the site of a diverticulum and the other branch passing external to the muscular layer and being displaced over the dome of the diverticulum. It appears likely that luminal traumatic factors, including chronic injury and impacted fecaliths which cause abrasion of the vessels, lead to the formation of ulcerations and erosions that ultimately result in hemorrhage.¹¹ Structural changes occur in the wall of the affected vessel, with thickening of the intima and focal attenuation of the media. The anatomic basis for bleeding is thought to be asymmetric rupture of these intramural branches (the vasa recta) of the marginal artery at either the dome

Figure 2 Colonic segmentation. Painter's conception of the formation of "little bladders" in the sigmoid colon with myochosis. Manometric traces from 3 sites show that intraluminal pressure rises higher when contractions occlude the lumen and form isolated segments. From Painter NS. The etiology of diverticulosis of the colon with special reference to the action of certain drugs on behavior of the colon. *Ann R Coll Surg* 1964; 34: 98. Reprinted with permission.

of the diverticulum or at its antimesenteric margin. Vessel disruption occurs on the mucosal side of the artery, as bleeding occurs into the lumen instead of into the peritoneal cavity. Inflammation is no longer the presumed underlying cause of diverticular bleeding as little or no inflammation is found in resected specimens and hemorrhage is rarely seen in the setting of acute diverticulitis.¹¹

Diverticular hemorrhage is thought to occur in 3 to 5% of all patients with colonic diverticulosis, yet appears to cease spontaneously in up to 90% of patients.¹² Transfusion of greater than four units of packed red blood cells is rare, with some data suggesting that when hemorrhage is limited to less than four units/day, bleeding stops in up to 99% of cases.¹³ After an initial episode of hemorrhage, rebleeding is likely to occur in 10% of patients in the first year; thereafter, the risk for rebleeding increases to 25% at 4 years.¹³

Although left colon diverticula are more common, some authors have hypothesized that bleeding may be more common from diverticula arising on the right side of the colon. The authors feel that right-sided preponderance is poorly documented and questioned by many given the difficulty in establishing the actual cause for massive hematochezia. This greater incidence has been postulated as a result of these diverticula tending to have wider necks and domes, exposing a greater surface area of their vasa recta to injury. Accordingly, hemorrhage from right colon lesions may be more serious and with bleeding rates greater than 0.5 mL/min, they tend to be more frequently visualized on angiography. In fact, without confirmation by direct visualization, these episodes of right-sided bleeding may be attributable to alternative sources such as arteriovenous malformations.

DIAGNOSIS

Emergency surgical intervention for ongoing massive hemorrhage is rarely necessary before attempts are made to localize the precise source of bleeding. This allows an orderly approach to identification of the bleeding site, which is essential for appropriate therapy. After the initial patient resuscitation and concurrent to the ongoing stabilization, diagnostic testing should begin. The choice of initial investigation remains controversial and depends on local availability of resources and expertise.

MANAGEMENT

Resuscitation

The initial priority in the setting of acute blood loss is the stabilization of the patient. Large-bore intravenous access should be obtained, allowing low resistance infu-

sion with the assistance of pressure bags and rapid transfusers. Foley catheter placement will help guide resuscitation. Blood should be sent to the laboratory for hematocrit, platelets, coagulation profile, and electrolytes while a specimen should be sent to the blood bank for cross-matching. After initial administration of crystalloid, subsequent bleeding or hemodynamic instability should be treated with packed red blood cells. Other products should be administered according to the clinical situation; patients taking warfarin with an elevated international normalized ratio (INR) may require intravenous Vitamin K and/or fresh frozen plasma while patients on hemodialysis with uremia may require desmopressin. Early consideration should be made regarding patient disposition, as these patients often require the level of nursing care and monitoring found in an intensive care unit.

The causes of *massive* lower gastrointestinal hemorrhage, defined as bleeding distal to the ligament of Treitz that requires the transfusion of three or more units of blood over 24 hours, are numerous. Most common are diverticular disease (thought to be the source of bleeding in 35 to 55% of cases) and angiodysplasia (20 to 30%), followed by inflammatory bowel disease, tumors, infectious colitis, and ischemic colitis.¹⁴ Less common etiologies include vasculitis, aortocolonic fistula, solitary rectal ulcer, colonic varices, endometriosis, and Dieulafoy's lesion of the colon.

Of course, an upper gastrointestinal source of bleeding should be ruled out by the passage of a nasogastric tube and aspiration of bilious fluid. Proctoscopy should also be performed to rule out easily treated anorectal pathology, such as active bleeding from hemorrhoids.

Localization and Treatment

There is great debate and controversy surrounding the optimal means of diagnosing and treating patients who present with lower gastrointestinal hemorrhage. The three options for primary diagnostic testing are colonoscopy, visceral angiography, and radionuclide imaging. The approach varies, often based on the institutional experience, the available resources, and the expertise of the caring physician. Regardless of the modality used, prompt localization of the source of bleeding avoids increased episodes of bleeding, greater transfusion requirements, and poorer prognosis.

Colonoscopy

"Urgent colonoscopy," completed within 6 to 12 hours of admission, is indicated in patients who have ceased to have ongoing significant hemorrhage and in whom resuscitation and hemodynamic stability have been achieved. The identification of the bleeding site is

facilitated by cleansing the colon of clots, stool, and blood with a large-volume purge, most commonly administering 5 to 6 L of polyethylene glycol solution 3 to 4 hours before the colonoscopy. Patients presenting with massive ongoing lower gastrointestinal hemorrhage, which often compromises patient hemodynamics, are poor candidates for "emergent colonoscopy" as the procedure is technically difficult due to the inability to clear the mucosal surfaces of old or new hemorrhage. Finding a discrete, actively bleeding vessel in the unprepared bowel is difficult even for the most experienced endoscopist.

A definite diagnosis of diverticulosis as the source of bleeding is confirmed by the finding of either (1) active bleeding, (2) a nonbleeding visible vessel, or (3) the presence of an adherent clot.¹⁵ Case reports have confirmed the use of several endoscopic modalities, separately or in combination, to successfully control diverticular hemorrhage, including: (1) heater probe administration; (2) epinephrine injection; (3) bipolar coagulation; (4) endoscopic hemoclip; and (5) fibrin glue administration.

In a recent paper by Jensen and colleagues,¹⁵ the investigators prospectively followed 121 patients who presented with severe hematochezia and diverticulosis. For all patients, colonoscopy was performed within 6 to 12 hours of the initial bleeding episode. For the first 73 patients (Group A, 1986 to 1992), colonoscopy was strictly diagnostic and all recurrent bleeding was treated with surgical resection. The subsequent 48 patients (Group B, 1994 to 1998) underwent colonoscopy in which therapeutic interventions such as epinephrine injection and bipolar coagulation were utilized. The choice of intervention was determined by the findings on colonoscopy. *Active bleeding* was controlled by four-

quadrant injection of 1:20,000 epinephrine (1 to 2 mL aliquots) followed by one-second pulses of Gold Probe with 10 to 15 watts of power. A *visible vessel* was treated with Gold Probe bipolar coagulation. An *overlying clot* was peripherally injected with epinephrine, the clot was shaved with a snare, and any visible vessel was coagulated with the bipolar Gold Probe.

Of the 73 Group A patients, 17 (23%) had definite signs of diverticular hemorrhage. Nine of these 17 patients experienced further bleeding after colonoscopy, and six required definitive surgical resection. Of the 48 Group B patients, 10 (21%) had definite signs of diverticular hemorrhage. All of these patients were treated endoscopically and none had recurrent bleeding requiring surgery (Table 1). Although their results were based on a limited number of patients, the authors were able to show that conventional endoscopic therapy prevents further bleeding from colonic diverticula.

The argument against colonoscopic control of diverticular hemorrhage dates back more than 30 years when it was noted that up to 50% of patients experienced recurrent bleeding. These high rates were used as evidence in support of early surgical intervention in older, higher-risk patients who can experience more aggressive recurrent bleeding. More recent data suggest recurrence rates in the 38% range,¹³ but this number arises from a retrospective study which included patients who did not undergo colonoscopic intervention. In the study by Jensen and associates, no patient with definite diverticular hemorrhage had late recurrent bleeding, or bleeding more than 30 days after discharge, with a mean follow-up of 30 months. All of these patients were discharged from the hospital on high-fiber diets and advised to avoid anti-inflammatory drugs and anticoagulants. Following this algorithm, it appears

Table 1 Outcome of Treatment for Diverticular Hemorrhage.

Variable	Medical and Surgical Treatment (N = 17)	Medical and Colonoscopic Treatment (N = 10)	P Value
Endoscopic hemostasis — no. (%)	0	10 (100)	0.001
Additional bleeding — no. (%) ^a	9 (53)	0	0.005
Severe bleeding — no. (%) ^b	6 (35)	0	0.03
Emergency hemicolectomy — no. (%)	6 (35)	0	0.03
Median time to discharge after colonoscopy — days	5	2	<0.001
Complications — no. (%)	2 (12) ^c	0	0.26
Late bleeding — no. (%)	0	0	1.0
Follow-up — mo			
Median	36	30	
Range	24–54	18–49	

^aAdditional bleeding was defined as self-limited or recurrent hematochezia that occurred after purging of the colon and colonoscopy and that required no more than an additional 2 units of packed red cells.

^bSevere bleeding was defined as continued or recurrent hematochezia that required at least 3 units of packed red cells.

^cOne patient had pneumonia, and one had a wound infection.

Reprinted with permission from 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.

reasonable that surgical intervention can be reserved for patients who fail both medical and colonoscopic treatment.

Nuclear Red Blood Cell Scintigraphy

Nuclear scintigraphy has met with mixed success in the diagnosis of lower gastrointestinal hemorrhage since its introduction in the late 1970s. This nuclear medicine imaging procedure carries the advantages of being a safe, noninvasive, and very sensitive test to show active bleeding.

There are two common types of nuclear scans to assess for gastrointestinal bleeding: ^{99m}Tc -labeled red blood cell scintigraphy and ^{99m}Tc -labeled sulfur colloid scintigraphy. ^{99m}Tc -labeled red blood cell scintigraphy is performed by incubating the patient's red blood cells for 10 minutes with a technetium isotope. The labeling process is best performed in vitro (ex vivo) and the labeled blood is reintroduced into the circulation. In other words, an aliquot of the patient's blood is withdrawn, labeled, and reinjected for scanning. For 90 minutes, serial images are recorded with whole abdominal scintigraphy. Images are obtained at distinct intervals after injection, within the first 2 hours, and thereafter at 4- to 6-hour intervals, or at the time of clinical evidence of rebleeding, up to 24 hours after initial injection. Rates of bleeding as low as 0.12 to 0.5 mL/min can be detected.

It is important to note that ^{99m}Tc -labeled red blood cell scintigraphy features several advantages when compared with ^{99m}Tc -labeled sulfur colloid scintigraphy. Specifically, ^{99m}Tc -labeled sulfur colloid is rapidly cleared (half-life of 7 to 10 minutes) so does not allow for repeat examinations over time. It is also taken up by the liver and spleen, obscuring the identification of hemorrhage at the hepatic and splenic flexures. When using ^{99m}Tc -labeled red blood cell scintigraphy, patients with intermittent bleeding can be reimaged several times for 24 hours, providing the clinician with a readily available means to evaluate patients with intermittent bleeding.

If bleeding is present at the time of injection and initial imaging, nuclear scintigraphy blood cell scans can accurately identify a source of bleeding in up to 85% of cases.¹⁶ Ng and associates were the first to demonstrate that the timing of the appearance of radionuclide blush provides valuable information. If the appearance is delayed (>2 minutes), patients are as unlikely to benefit from an angiogram as those with negative bleeding scans, with a greater than 90% negative angiographic yield.¹⁷

^{99m}Tc -labeled red blood cell scintigraphy has limited ability to accurately identify the actual site of hemorrhage to direct surgical intervention. A wide review of the literature reveals that the study is accurate in

only 40 to 60% of patients—little better than a 50:50 ratio to isolate bleeding to the left or right colon, although variable rates between 24% and 91% have been published.¹⁸ Small intestine hemorrhage may mimic large intestine hemorrhage and, if bleeding is not active at the time of the initial study, or if delayed bleeding occurs, localization can be inaccurate because of the movement of the tracer in the intestinal lumen. Hence, patients in whom a surgical resection is anticipated to control recurrent or persistent hemorrhage should have the bleeding confirmed with either a positive angiogram or a positive colonoscopy. The red blood cell scans serve primarily to target the subsequent confirmatory study.

Selective Visceral Angiography

Mesenteric arteriography has been widely used in the evaluation and treatment of patients with lower gastrointestinal hemorrhage. Selective injection of radiographic contrast into the superior mesenteric or inferior mesenteric arteries identifies hemorrhage in patients bleeding at a rate of 0.5 mL/min or greater. When positive, angiography is 100% specific.

About 10% of patients develop a complication of angiography.¹⁹ Major complications include stroke, renal failure, femoral artery thrombosis, lower extremity immobilization, and hematoma formation. Given that most patients with lower gastrointestinal hemorrhage are older than 60 years of age, medical comorbidities, including vascular disease and renal insufficiency, may place these patients at high risk for the procedure. Hence, angiography is reserved for patients with evidence of significant ongoing hemorrhage.

In patients whose bleeding source is identified by angiography, a trial of angiographic therapy may be appropriate as a definitive measure, especially for high-risk surgical candidates. The therapeutic options include vasopressin infusion and transcatheter embolization, and there is ongoing controversy regarding the preferential use of embolotherapy versus vasopressor infusion.²⁰ With selective catheterization of a bleeding mesenteric vessel, intra-arterial vasoconstrictor therapy with vasopressin can temporarily achieve control of bleeding in up to 80% of patients. Continuous arterial infusion of vasopressin should begin at a rate of 0.2 U/min and may be continued for a period of 12 to 24 hours. The rate may occasionally be increased for short periods to as much as 0.4 U/min. If there is no evidence of further bleeding, the vasopressin may be tapered over 12 to 24 hours. Unfortunately, rebleeding is common after discontinuing the therapy, with short-term rebleeding rates of 16 to 50%.²⁰ Complications are frequent and range from minor, including fluid retention, transient hypertension, sinus bradycardia, and hyponatremia, to major, including myocardial ischemia, pulmonary edema, arrhythmias,

and mesenteric thrombosis. Therefore this therapy is relatively contraindicated in patients with coronary artery disease. The primary role of this therapy is to achieve temporary control of bleeding before emergency definitive surgical resection.

Utilizing newer technologies, interventional radiologists are now able to intervene with more specific and definitive therapies. Transcatheter embolization of massive bleeding, utilizing gelatin sponges or microcoils, can achieve control of bleeding from diverticula. As a result of the previously outlined lack of collateral blood supply to the colonic wall, several cases of postembolic infarction were reported in the 1980s. Since that time, newer catheter-based techniques which allow more precise, super-selective targeting of bleeding vessels have become available. These techniques preserve collateral blood flow to bowel mucosa and minimize the risk of bowel infarction, with no cases of infarction reported after 1992 (Table 2).

The use of superselective microcoil embolization in the setting of lower gastrointestinal hemorrhage has never been compared prospectively to surgery and is limited to only 122 reported cases in the literature. In 2003, Kuo and associates reviewed their institution's 10-year experience of lower gastrointestinal bleeding superselective microcoil embolization while concurrently reviewing all complications associated with the previously reported cases in the literature.²¹ Between the years 1992 and 2002, 22 patients had evidence for lower gastrointestinal bleeding discovered by nuclear scintigraphy (11), active bleeding (5), colonoscopy (3), abdominal CTA (1), ultrasound (1), and physical exam (1). The average pre-embolization blood transfusion requirement was 6.8 units. The primary embolic agent in each

case was microcoils, with the use of Gelfoam or polyvinyl alcohol as secondary agents. All patients were followed for evidence of postembolization ischemia and for re-bleeding. Immediate hemostasis was achieved in all 22 patients, with complete clinical success in 19 of 22 patients (86%). Three of 22 patients experienced re-bleeding within 1 day of embolization, all of which were in the rectum due to ulcers (2) or hemorrhoids (1) and were subsequently controlled endoscopically. A minor ischemic complication, defined as a condition that required nominal or no further therapy, was reported in 1 of 22 patients (4.5%) while there were no major ischemic complications, defined as a bowel infarction that required surgery. Compared with the author's review of the prior 122 published cases of lower gastrointestinal superselective microcoil embolization, their findings were in line with prior studies—a 9% minor complication rate and 0% major complication rate.

Provocative Tests

A certain subset of patients experience ongoing yet intermittent lower gastrointestinal hemorrhage while the site remains elusive despite both colonoscopic and angiographic evaluation. Interventional radiologists have described a provocative study in several reports as a way to induce bleeding at the time of angiography by stimulating clot lysis. Investigators have utilized heparin or thrombolytic agents (tissue plasminogen activator, streptokinase, or urokinase) in a controlled setting to localize the site to further direct either endovascular or surgical intervention.

In 2001, Ryan and coworkers reported their experience with intra-arterial provocative mesenteric

Table 2 Ischemic Complications Following Superselective LGI Embolization Involving Microcoils

Year/Author	Minor Ischemia	Infarction	Objective Follow-up	Cases
1996 Curzon ¹³	2	0	?	9
1997 Gordon ⁵	0	?	5	13
1998 Peck ¹⁴	0	0	3	6
1998 Lederman ⁸	0	0	2	10
1998 Nicholson ¹⁵	3	0	?	14
1999 Dobson and Nicholson ¹⁶	1	0	3	4
2000 Evangelista and Hallisey ⁶	2	0	12	17
2001 Funaki ¹⁷	3	0	7	24
2001 Bandi ¹¹	?	0	?	8
2001 Defreyne ¹⁸	0	0	0	1
2002 Debarros ¹⁹	1	0	16	16
2003 Current study	1	0	14	22
Total	13	0	62	144

?, data unspecified or indeterminate. The objective incidence of minor ischemia is 21% (13/62) with overall incidence of 9% (13/144). The estimated incidence of infarction is 0% (0/144).

Reprinted with permission from Kuo WT, Lee DE, Saad WE, et al. Superselective microcoil embolization for the treatment of lower gastrointestinal hemorrhage. *J Vasc Interv Radiol* 2003; 14: 1503–1509.

angiography, retrospectively reviewing 17 cases.²² All patients had negative colonoscopies (mean, 3.8 studies) and negative angiograms (mean, 1.5 studies). Fourteen patients underwent tagged red blood cell studies resulting in 10 positive studies, but 6 were weakly positive or did not adequately localize the site of bleeding. All patients underwent preprocedure blood group cross-matching and surgical consultation. After accessing the common femoral artery and full celiac and mesenteric angiogram, patients were infused with a combination of medications: heparin, tolazoline, and tPA. After 15 minutes, angiography of the provoked vessel was performed. Provoked bleeding was identified in six patients (37.5%) due to three episodes of diverticular hemorrhage, one case of angiodysplasia, and two episodes of small bowel bleeding. Of note, six of these patients had positive tagged red cell studies, with three of the six localizing to the same site of bleeding. Three of the patients were immediately treated with superselective embolization, two were treated medically, and one required surgical exploration and resection for recurrent bleeding 2 months after failed embolization.

Surgery

Historically, surgical intervention for colonic diverticulosis has been limited. In 1964, Reilly recommended sigmoid myotomy in the treatment of diverticular disease.²³ This procedure involves division of the antimesenteric taeniae and underlying circular muscle from the rectosigmoid junction to "whatever distance is necessary." In 1973, transverse taeniamyotomy was proposed by Hodgson.²⁴ In this procedure the two antimesenteric taeniae were transversely incised at 2-cm intervals from the rectosigmoid junction to normal colon proximally. Neither procedure gained acceptance and current surgical treatment for diverticulosis is reserved for complications of hemorrhage and inflammation.

Surgery is indicated for patients with either ongoing or recurrent hemorrhage. Transfusion of more than 6 units of packed red blood cells, ongoing transfusion requirement, or persistent hemodynamic instability are indications for colectomy in acute hemorrhage. Recurrence of bleeding after one episode has been estimated at 20 to 30%, with an incidence of > 50% after a second episode. A controversy persists in deciding the optimal time for surgical intervention: whether one should proceed after the second episode or delay until a third bleed occurs. The ultimate decision depends largely on the specific clinical scenario and the individual patient's medical condition.

In patients who require an operative solution, every effort should be made to localize the source of bleeding. Localized sites allow for a segmental intestinal resection rather than a blind subtotal abdominal colectomy. Certainty of the site of bleeding is very important

and operation based on a positive ^{99m}Tc red blood cell scan alone can result in recurrent hemorrhage in up to 35% of patients. There will seldom be signs that will reliably identify the cause of bleeding intraoperatively, although non-blood stained succus within the small bowel is a reliable indication that the source of bleeding is distal to the ileocecal valve. Intraoperative colonoscopy is rarely beneficial for further localization as it is difficult to visualize a bleeding source when it is occurring at a rate fast enough to require emergent laparotomy. "Blind" total abdominal colectomy carries significantly higher rates of perioperative morbidity and mortality. Diarrhea and rapid transit after total abdominal colectomy can also be debilitating conditions for elderly patients.

Due to the high morbidity and mortality associated with emergent operations in the setting of acute lower gastrointestinal hemorrhage, most surgeons are reluctant to proceed until all other options have been explored. Of course, in the setting of massive hemorrhage with resultant hemodynamic instability, there may not be options other than an expeditious trip to the operating room. There are also conditions that require surgical resection for definitive therapy, such as a bleeding carcinoma.

In the setting of diverticular hemorrhage, depending on the clinical scenario, one can consider four potential surgical interventions, each carrying its own distinct morbidity/mortality rate and risk of postoperative rebleeding:

1. Elective segmental resection for a known bleeding source, either for rebleeding or definitive therapy. When conducted in a controlled setting, this option is the safest with the lowest rebleeding rate (0 to 14%).²⁵
2. Emergency segmental resection for a known bleeding source that continues to bleed. This second-choice procedure carries a morbidity rate of 8.6%.
3. Emergency total abdominal colectomy with ileorectal anastomosis. This is the operation of choice when a patient has massive colonic hemorrhage without preoperative localization. Unfortunately, this procedure carries a high morbidity rate of 40%, a mortality rate of 30 to 33%, and a rebleed rate of 0 to 10%.^{25,26}
4. Emergency segmental resection for an unknown bleeding source, or "blind segmental resection." In general, this operation is not indicated, with the highest rates of morbidity (83%), mortality (12 to 57%), and rebleeding (42 to 63%).^{13,25,27}

THE POTENTIAL ROLE OF COMPUTED TOMOGRAPHIC ANGIOGRAPHY

In 2003, two sets of investigators evaluated the use of helical CT scanning as yet another tool in the early management of lower gastrointestinal hemorrhage.

Kuhle and Sheiman evaluated the feasibility of helical computed tomography in depicting active colonic hemorrhage using the swine model.²⁸ In an animal model, controlled extravasation of contrast-enhanced blood via a catheter placed at the splenic flexure was evaluated at varying rates, from 0.3 to 1.0 mL/min. Sixteen exams produced 16 dilution curves, and targeted CT could depict extravasation at rates as low as 0.3 mL/min. The authors concluded that helical CT has the potential to depict active colonic hemorrhage at conservative rates of 0.5 mL/min or less, a rate equivalent to the accepted rate of bleeding detected by visceral angiography. The advantage of CT is that it is a significantly faster and less morbid option than angiography. In addition, with CT reconstruction capability, the precise location of the site of hemorrhage may be identified. Also, computerized tomography is readily available in most medical centers. Major limitations for CT angiography may relate to the need for intravenous contrast. Intravenous contrast has proven a significant problem for adequate renal function. Positive CT angiograms may then need further selective contrast angiography. The contrast load could double and increase the risk of nephrotoxic events, especially in patients with decreased perfusion pressures related to the volume losses during hemorrhage. Contrast-enhanced magnetic resonance angiography does not have an increased risk of nephrotoxicity and may prove a safer diagnostic test.

Yamaguchi and Yoshikawa published their clinical experience in 2003, supporting this fast and less invasive means of evaluating patients with lower gastrointestinal tract bleeding.²⁹ Five patients who presented with lower gastrointestinal bleeding were immediately evaluated with contrast-enhanced CT scan. Pooling of contrast was seen in four of the five patients, three from diverticular disease, one from a rectal ulcer, and one from a small intestinal ulcer. The authors concluded that further studies would be needed before this technology could be recommended for routine patient evaluation.

CONCLUSION

Colonic diverticulosis predisposes individuals to lower gastrointestinal hemorrhage in up to 5% of cases. These sac-like protrusions are pseudodiverticula and arise due to a combination of anatomic, dietary, motility, and structural influences. In the setting of acute hemorrhage, patient stabilization takes priority, followed closely by maneuvers aimed at localizing and controlling blood loss. Utilizing an arsenal of tools including colonoscopy, angiography, and nuclear scintigraphy, most diverticular bleeds can be localized and subsequently controlled. When persistent and not controlled by colonoscopic or angiographic means, expeditious surgical resection serves as definitive therapy.

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