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Acute Gastrointestinal Hemorrhage: Radiologic Diagnosis and Management

Jeffrey D. Jaskolka, MD, FRCPC[a,*], Shagran Binkhamis, MBBS[b], Vikram Prabhudesai, MBBS, MS, FRCR[c], Tanya P. Chawla, MBBS, MRCP, FRCPC[d]

[a]*Department of Medical Imaging, Mount Sinai Hospital, Toronto, Ontario, Canada*
[b]*Department of Radiology, College of Medicine, King Saud University, Riyadh, Saudi Arabia*
[c]*Department of Medical Imaging, St Michael's Hospital, Toronto, Ontario, Canada*
[d]*Department of Medical Imaging, Mount Sinai Hospital, Toronto, Ontario, Canada*

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Acute gastrointestinal (GI) hemorrhage is common both in the emergency department and in the primary care setting [1]. In the United States, approximately 300,000 patients are admitted to the hospital annually [2]. Upper GI hemorrhage has an annual incidence that ranges from 40-150 episodes per 100,000 persons [3] compared with lower GI hemorrhage, which is less common, with an annual incidence that ranges from 20-27 episodes per 100,000 persons. The mortality rate from GI bleeding in the presence of hemodynamic instability may reach 40%, which makes accurate and prompt diagnosis of the source of bleeding crucial [4]. In more stable patients, the mortality is lower, ranging from 3.6%-19% [3,5,6].

In up to 75% of cases, bleeding will cease spontaneously, but recurrence may occur in 25%, which emphasizes the need for accurate diagnosis and effective therapy, preferably in the acute setting [2,6]. Endoscopy is considered the primary diagnostic and therapeutic modality in the setting of GI hemorrhage. Nevertheless, due to the large number of potential etiologies, a high prevalence of obscure bleeding sources, and the long length of the GI tract, with many segments inaccessible to endoscopy, imaging continues to play an important role in the diagnosis of GI hemorrhage [7]. Therefore, it is important that all diagnostic and interventional radiologists fully understand the role of imaging and endovascular treatment of GI hemorrhage. In this article, we briefly discuss the clinical presentation, major causes of GI tract hemorrhage, and the diagnosis and management from a clinical perspective. We will primarily focus on the radiologic diagnosis and management with a special focus on the emerging role of computed tomographic angiography (CTA) as well the most recent advances in endovascular treatment.

Clinical Presentation and Major Causes of Upper and Lower GI Tract Hemorrhage

By definition, upper GI bleeding originates proximal to the ligament of Treitz, and lower GI imaging originates beyond the ligament of Treitz. Upper GI bleeding may originate in the esophagus, stomach, and duodenum, and carries a mortality rate of approximately 10% [5]. Common causes of upper GI bleeding are peptic ulcer disease, variceal bleeding, Mallory-Weiss tear, vascular lesions, and neoplasms (Table 1) [2]. Lower GI bleeding may originate in the small bowel, colon, or rectum [8] and carries a mortality rate of 3.6% [9]. Lower GI bleeding is more common in elderly patients and is 200 times more likely to affect an 80-year-old patient than a 20-year-old patient [2]. Common causes of lower GI bleeding are diverticular disease, angiodysplasia, neoplasms, colitis, and benign anorectal lesions such as hemorrhoids, anal fissures, and rectal ulcers (Table 2) [2].

Upper GI bleeding usually presents with hematemesis (vomiting of blood or "coffee ground"-like material) and/or melena (black, tarry stools). Lower GI bleeding classically presents with hematochezia (passage of maroon or bright red

* Address for correspondence: Jeffrey D. Jaskolka, MD, Department of Medical Imaging, Mount Sinai Hospital, 600 University Avenue, Room 564, Toronto, Ontario M5G 1X5, Canada.
E-mail address: jjaskolka@mtsinai.on.ca (J. D. Jaskolka).

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Table 1
Common causes of upper gastrointestinal hemorrhage[a]

Peptic ulcer disease (55%)
Infections (*Helicobacter pylori*, viral)
Drug induced
Stress induced
Zollinger Ellison syndrome
Other causes of esophagitis and/or gastritis and/or duodenitis
Portal hypertension
Varices (14%)
Portal gastropathy
Neoplasms (4%)
Traumatic or iatrogenic
Mallory-Weiss tear (5%)
Foreign body ingestions
After surgery
After biopsy and/or polypectomy
Vascular
Arteriovenous malformations (6%)
Dieulafoy lesion (1%)
Aortoenteric fistula
Pancreaticobiliary

[a] The numbers in parentheses indicate the frequencies of the most common etiologies for upper gastrointestinal hemorrhage.

blood or blood clots per rectum). However, this distinction is not free from exceptions, because bleeding from the right colon (or the small intestine) can present with melena, and massive upper GI bleeding can present with hematochezia [10–12]. The use of nasogastric-tube insertion and gastric lavage in all patients with suspected upper GI bleeding is controversial. Although a lavage that yields blood or ''coffee ground''-like material confirms the diagnosis, lavage may not be positive if bleeding is no longer active or if the source of bleeding is beyond a closed pylorus. Studies of the use of nasogastric-tube insertion have failed to demonstrate a benefit with regard to clinical outcomes [13,14]. In all patients who present with clinically significant GI hemorrhage, initial management is aimed at resuscitation of the patient, followed by rapid diagnosis, and, if possible, treatment.

Diagnosis

Options for the diagnosis of GI hemorrhage include endoscopy and/or colonoscopy, nuclear scintigraphy, CTA, and catheter angiography. The relative importance of each of these modalities is different for upper- and lower GI bleeding, and will be reviewed in turn.

Table 2
Common causes of lower gastrointestinal hemorrhage[a]

Diverticulosis (15%-55%)
Angiodysplasia (3%-37%)
Neoplasm (3%-11%)
Polyp
Carcinoma
Iatrogenic (0%-13%)
After biopsy and/or polypectomy
Ischemia (6%-18%)
Infection
Inflammatory bowel disease
Benign anorectal lesions

[a] The numbers in parentheses indicate the frequencies of the most common etiologies for upper gastrointestinal hemorrhage.

Endoscopy and/or Colonoscopy

In patients with upper GI bleeding, endoscopy is considered the criterion standard for diagnosis. It has the advantage of being both a diagnostic and therapeutic modality. Although somewhat invasive, it is generally considered safe to perform in patients with upper GI bleeding, including those with medical comorbidities, severe hemorrhage, and altered coagulation. The reported sensitivity and specificity of endoscopy for upper gastroduodenal bleeding are 92%-98% and 30%-100%, respectively, and a repeated endoscopy may further increase the diagnostic yield when performed by an experienced operator. Endoscopy has the ability to identify active bleeding but may also identify etiology and the site of hemorrhage in those patients who have ceased bleeding. These patients can potentially be treated despite a lack of ongoing hemorrhage, although the value of this approach is controversial. In cases in which bleeding can be identified but not treated endoscopically, a metallic clip can be placed to guide angiography for either selective or empiric embolization (Figure 1). In the setting of upper GI bleeding, imaging is reserved for nondiagnostic endoscopy in which a bleeding site cannot be identified and/or treated, which may occur when there is very rapid bleeding and blood in the stomach obscures the endoscopic visualization of the underlying source.

In lower GI hemorrhage, the diagnostic approach is somewhat more variable. The American College of Gastroenterology has issued guidelines that suggest that colonoscopy should be the first-line diagnostic modality for evaluation and treatment of lower GI bleeding [15]. However, there are several limitations to colonoscopy in the setting of acute lower GI hemorrhage, including the potential for inadequate bowel preparation, the inability to evaluate most of the small bowel, risks of sedation in hemodynamically unstable patients, a low prevalence of stigmata of hemorrhage, and a potential lack of availability of an appropriate team for performance of the procedure [16]. Although colonoscopy can determine the etiology of hemorrhage in 91% of patients overall [17], this number decreases in patients without adequate bowel preparation, and successful treatment may only be possible in as few as 21% of patients in the acute setting [18]. The American College of Radiology recommends colonoscopy as the initial modality in hemodynamically stable patients (in whom there is time to undergo colonic preparation) and angiography in those who are hemodynamically unstable with massive bleeding [19].

Nuclear Scintigraphy

GI hemorrhage has long been diagnosed with scintigraphic techniques. In current practice, the most commonly used agent is technetium 99m–labeled red blood cells. Active bleeding can generally be detected at rates of 0.3 mL/min, and some investigators believe that rates as low as 0.1 mL/min can be detected [20]. Nuclear scintigraphy has the

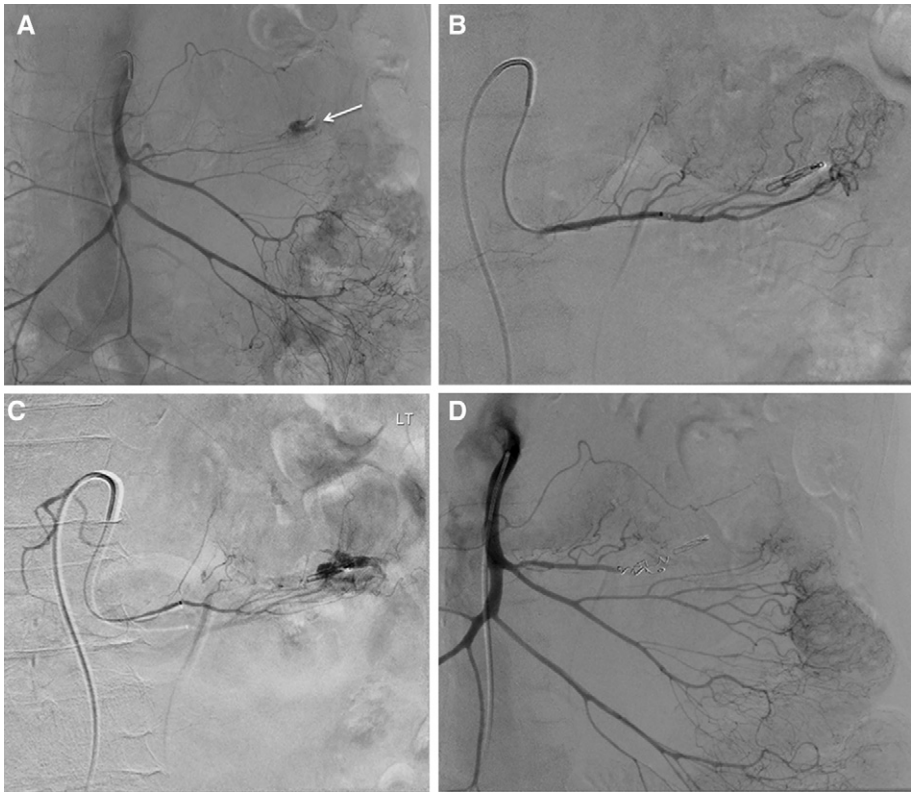

Figure 1. Postanastamotic hemorrhage after Bilroth 2 gastrojejunostomy treated with embolization. Endoscopic treatment failed twice prior to arteriography. (A) Superior mesenteric artery (SMA) arteriogram showing hemorrhage from first jejunal artery, immediately adjacent to endoscopically placed clip (arrow). (B) Initial subselective arteriogram of first jejunal artery showing absence of extravasation. (C) Subsequent subselective arteriogram of first jejunal artery, showing active extravasation. (D) SMA arteriogram after embolization with three 2 × 3.3-mm microcoils showing no further hemorrhage.

advantage of being noninvasive and having the ability to image over long periods of time and, therefore, potentially detect intermittent bleeding. However, the spatial resolution of scintigraphy is lower than that of other modalities, and the precise etiology of hemorrhage cannot be determined. In addition, intervention is not possible with this modality. Some investigators suggest that scintigraphy can be used as a screening test for angiography with positive red blood cell scans, which increases the likelihood of finding active extravasation on angiography and, therefore, predicting the likelihood of finding a treatable lesion [21,22]. When positive, scintigraphy is useful for localizing bleeding for endoscopic, surgical, or endovascular therapy (Figure 2). Nuclear scintigraphy is likely most useful for evaluation of subacute or chronic hemorrhage in centres where endoscopy, computed tomography (CT), and/or CTA are available.

CTA

Over the past decade, there has been a growing interest in the use of CTA for evaluation of patients with both upper- and lower GI hemorrhage [23–32]. These studies all support an emerging role for CT in the diagnosis of GI hemorrhage, which highlights the fact that CT is very accurate for determining the location and etiology of hemorrhage. These studies are generally limited by their small numbers, heterogeneity in imaging technique and patient populations, and also by selection bias. The most recent prospective study [32] studied 47 patients, with a reference standard of angiography, endoscopy, surgery, or a combination of these and showed sensitivity, specificity, positive predictive value, and negative predictive value of 100%, 96%, 95%, and 100%, respectively.

Three meta-analyses have been performed with this patient population. The most recent [33] used results from 6 prospective studies and 3 retrospective studies, which incorporated a total of 198 patients. The pooled sensitivity and specificity for diagnosis of GI hemorrhage were 89% and 85%, respectively. The area under the summary receiver operator characteristics curve was 0.93, which indicates very good diagnostic accuracy. The investigators conclude that CT should be used as the first-line radiologic test for evaluation of patients with acute GI hemorrhage.

There are many potential advantages to performing CTA for the diagnosis of GI hemorrhage. First, it is noninvasive and more widely available than catheter angiography. Second, it may be more sensitive than angiography for detection of bleeding. In an animal model, it has been shown that CTA can detect extravasation of blood at a rate as low as 0.3 mL/min, although more reliably at 0.5 mL/min [34], which is lower than that reported for catheter angiography [35,36] and similar to the rates of bleeding detectable by scintigraphy [20]. A CT can also demonstrate evidence of recent bleeding, for example, blood in the bowel lumen [24]. It can identify lesions such as neoplasms or vascular

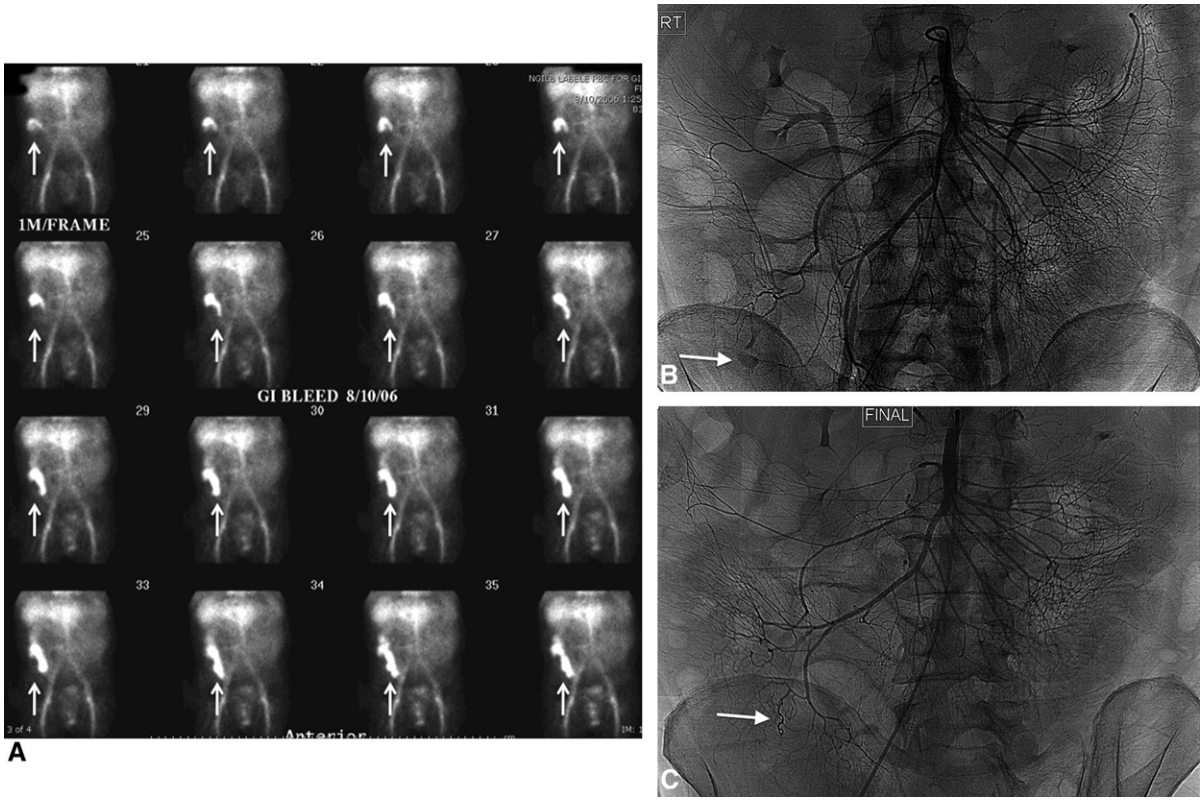

Figure 2. Right colonic diverticular hemorrage with positive ^{99m}Tc-red blood cell (RBC) scan, confirmed on arteriography and subsequently embolized. (A) Multiple frames from ^{99m}Tc-RBC scan showing abnormal activity in right lower quadrant (arrows), which progressively increases over time and has appearance of the cecum in the later frames. (B) Superior mesenteric artery (SMA) arteriogram showing active extravasation in the right lower quadrant (arrow) with a shape suggestive of a diverticulum. (C) SMA arteriogram after selective embolization with two 2 × 20-mm microcoils (arrow) shows no further extravasation.

malformations that can be the cause of hemorrhage, even when they are not actively bleeding. It has also been proposed that performing CTA before conventional angiography is useful for procedure planning and may decrease procedure time, radiation dose, and contrast volumes during embolization [37]. CT may also detect some less common arterial sources of hemorrhage such as hepatic or pancreatic branches or the internal iliac arteries.

A variety of CTA techniques have been used for evaluation of GI bleeding, including intra-arterial CTA [23], single-detector CT [23,24], and multidetector CT with up to 64 detectors [32,38]. Two studies have specifically addressed the number of phases needed for evaluation. Results of one of these studies suggested no added value of arterial and venous phase scanning after contrast injection compared with a single arterial or venous phase [39], which is in contrast to an animal model in which it was shown that both phases are necessary to obtain maximum sensitivity and specificity [40].

Most investigators recommend the use of triphasic technique [41,42], which includes a precontrast study, a CTA, and a venous phase scan (Table 3). The noncontrast phase is considered necessary to prevent a false-positive diagnosis from preexisting density in the bowel, such as from suture material, surgical clips, prior administration of positive enteric contrast, ingested pills, and so forth. Arterial-phase imaging with high-density contrast and thin-section acquisition can detect extravasation of contrast and also provide angiogram-like images for directing embolization. It can also increase conspicuity of vascular tufts, highly vascularized masses, and early draining veins. Density of intraluminal contrast >90 HU is considered diagnostic of active

Table 3
Suggested computed tomography protocol for evaluation of acute gastrointestinal hemorrhage

Large-bore intravenous catheter in antecubital fossa if possible
120 kVp and 240 mAs for all phases
No enteric contrast
May consider oral water if neoplasm or vascular malformation is suspected
Noncontrast
5-mm slice thickness through entire abdomen and pelvis
Consider a low-dose technique
Arterial phase
100-150 mL of high-density, nonionic, iodinated contrast
Power injection at a rate of 4-5 mL/s
A normal saline solution bolus chase of 20 mL at the same rate as the contrast injection
Initiate the scan when attenuation of aortic blood is 150 HU
≤1-mm slice thickness through the entire abdomen and pelvis
Coronal and sagittal reformations
Consider 5–10-mm-thick coronal maximum intensity projection reconstructions
Venous phase
70 s after initial scan
5-mm slice thickness through entire abdomen and pelvis
Consider coronal and sagittal reformations 3–5-mm thick

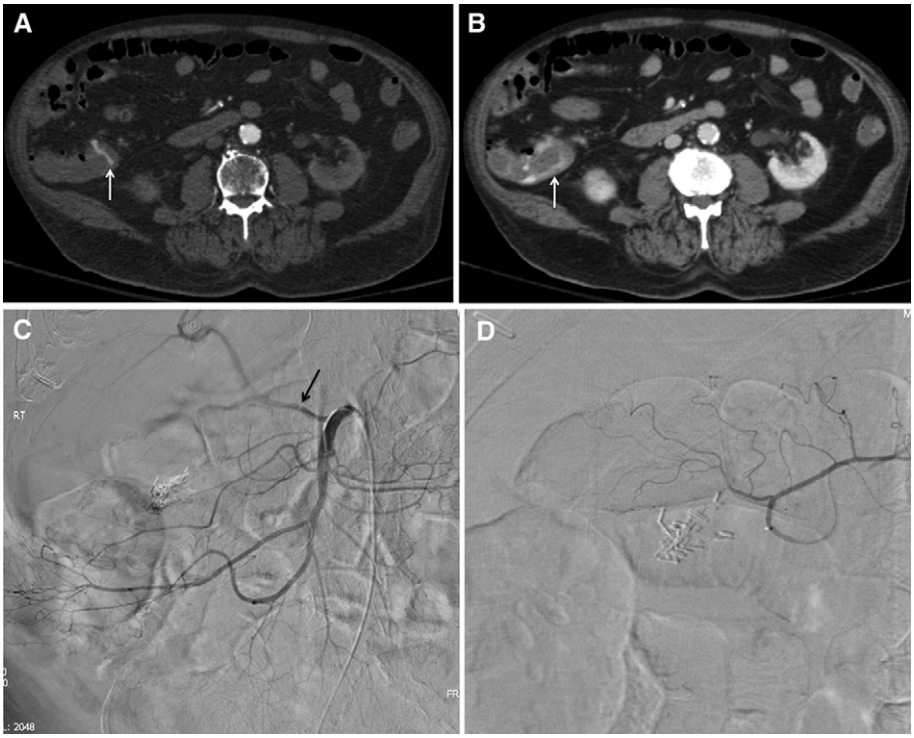

Figure 3. Spontaneous cessation of bleeding after computed tomography (CT) angiography positive for diverticular hemorrhage. The patient improved clinically and has had no evidence of recurrent hemorrhage at 6-month follow up. (A) Axial image from arterial phase CT showing active jet-like arterial enhancement (arrow) in ascending colon at site of diverticulum. (B) Axial image from venous phase of CT shows progressive increasing intraluminal enhancement (arrow). (C) Superior mesenteric artery arteriogram showing lack of active extravsation. (D) Subselective right colic arteriogram confirming lack of extravasation.

hemorrhage. The configuration of contrast can be linear, jet like, swirled, ellipsoid, or pooled [43]. The venous phase is considered useful for evaluation of progressive contrast extravasation as well as some of etiologies of hemorrhage, such as neoplasms or polyps. Some investigators considered bowel-wall thickening, perienteric stranding, and bowel-wall enhancement as indicators of a location of hemorrhage. Positive oral contrast should not be given because it may obscure the density from extravasated contrast. There is variability with regard to administration of negative oral contrast, with some investigators advocating the use of oral water [44] and others suggesting that oral contrast is unnecessary [34] because it may dilute extravasated contrast and decrease sensitivity for detection of hemorrhage [7]. At present, the triphasic technique is recommended for optimal sensitivity and specificity because the radiation dose is a lesser concern in the setting of severe GI bleeding, which may be life threatening. Interpretation of the CT study is aided by use of multiplanar reformats and maximum intensity projection images [42,45]. Active bleeding is required for a positive CT. Detection rates are better in patients with more severe bleeding, that is, in those with massive bleeding (>4 units in 24 hours), hemoglobin level decrease of more than 20 g/dL, or systolic blood pressure <90 mm Hg.

Although most investigators advocate the use of endoscopy as the initial diagnostic test [15,19], especially for upper GI bleeding, several investigators recommend CTA instead of catheter angiography as the next step in evaluation of patients with negative endoscopy, and some investigators suggest the use of CT before colonoscopy in the initial evaluation of patients with lower GI hemorrhage. It is clear from the literature that CTA is a potentially useful tool in the diagnosis of GI bleeding. However, its exact role in the evaluation of patients with GI hemorrhage requires further study. A positive study is highly specific and should lead to definitive therapy, usually catheter angiography, depending on local institutional practice. However, the value of a negative study in further triage of the patients needs to be elucidated. Results of some studies indicate that a negative CTA suggests spontaneous cessation of bleeding and carries with it a low rate of rebleeding [30], and, therefore, a negative CTA obviates the need for catheter angiography (Figure 3), which is supported by the high negative predicative value in the study of Marti et al [32]. However, there is a wide range of a negative predictive value, from 33%-100%, in the other published studies. In 1 study [44], the investigators assumed that a positive CT in the setting of a negative angiogram constituted a true-positive study, whereas in most other studies, these were considered as false-positive studies. Although, strictly speaking, this is an incorrect approach because other modalities are referred to as the criterion standard, when one considers the intermittent nature of bleeding and the possibly improved sensitivity to bleeding on CT, this may have been a reasonable approach. In almost all the published studies, there were some cases of positive CT and negative angiography. Some of these had endoscopic

correlation of hemorrhage, which raises the question of what should be the true criterion standard for a diagnosis of GI hemorrhage. Even at surgery, a presumed bleeding lesion is often removed, without conformation of active bleeding at the time of laparotomy.

Based on the above discussion, a reasonable role for the use of CT in evaluation of patients with GI bleeding is as follows:

1. In patients with mild GI bleeding, endoscopy or colonoscopy should be done first. If the endoscopy is negative, then a CT may be useful to detect bleeding and to determine the etiology. If a bleeding source is found, then it can be managed as determined by institutional practice. In these patients with low acuity, it is likely reasonable to assume that a negative study is truly negative and that, regardless, this represents a group of patients with good outcomes and that further urgent therapy is unlikely to be needed.
2. In patients with massive GI hemorrhage but who are stable, endoscopy should be done first in upper-GI bleeding and, if it is negative, then a CT could be considered before angiography. If the CT is negative for active bleeding or an etiology of bleeding, then it may be reasonable to manage expectantly without angiography or surgery, although this has not been proven. For lower-GI bleeding, if there is sufficient time for bowel preparation and experienced endoscopists available, then colonoscopy should be considered. Alternatively, CTA could be done and used to guide further management.
3. The group of patients with massive GI hemorrhage and hemodynamic instability has not specifically been studied and, in some of the published studies, were excluded. In these patients, there is a high likelihood of active extravasation, and they should likely proceed directly to angiography unless it is not locally available. CT should only be considered if it can be done in such a way as to not delay angiography.

Catheter Angiography

Catheter angiography is an attractive diagnostic modality for evaluation of GI hemorrhage because it is a potentially diagnostic and therapeutic tool. It is widely quoted that angiography can detect bleeding at rates between 0.5 and 1.0 mL/min [35,36]. Complications include access-site hematoma or

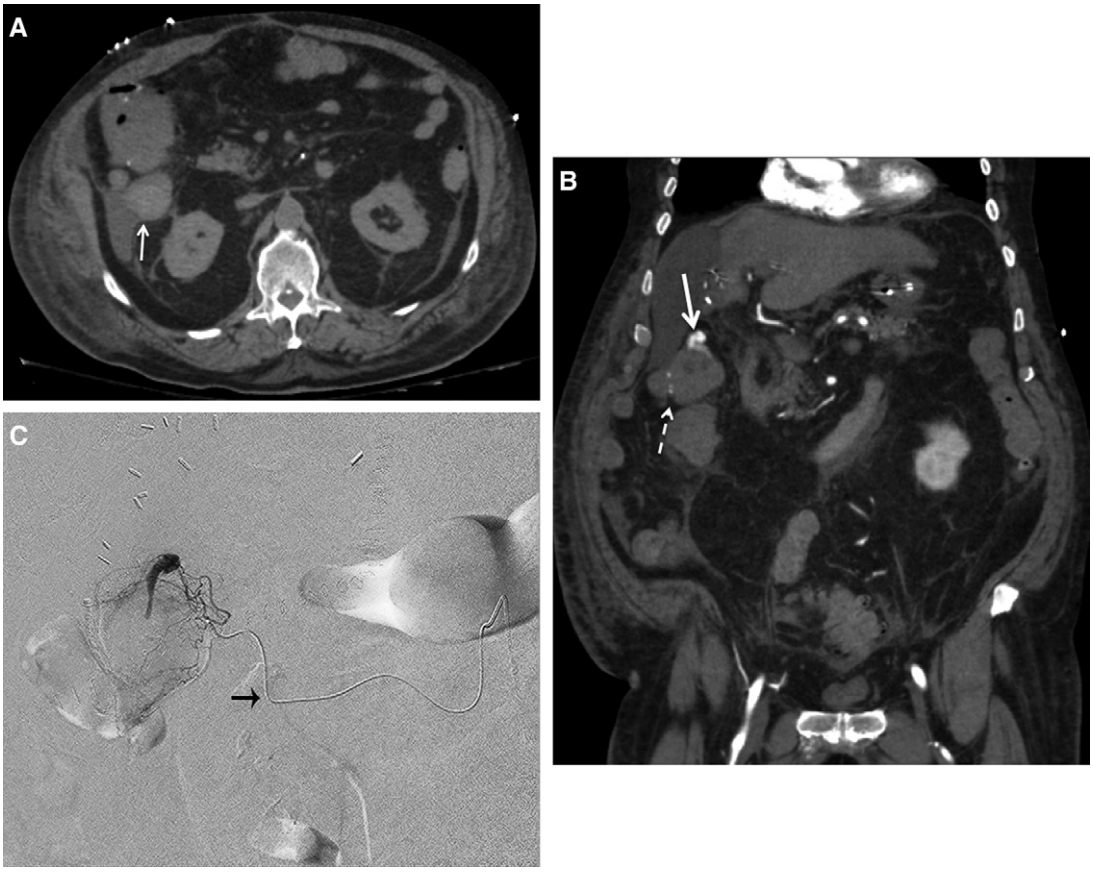

Figure 4. Diverticular hemorrage from transverse post-right hemicolectomy, diagnosed with computed tomography (CT) and treated by embolization. (A) Axial unenhanced CT image shows dense material in distal small bowel (arrow), proximal to ileocolic anastamosis. (B) Coronal arterial phase CT showing active extravasation into colonic diverticulum (solid arrow). Note the surgical staple line (dashed arrow). The CT findings suggest a middle colic branch as the source of bleeding. (C)Superselective distal middle colic arteriogram via a microcatheter (arrow), confirming active extravasation. Rapid selection of bleeding artery was facilitated by prior CT angiography. This vessel was then embolized (images not shown).

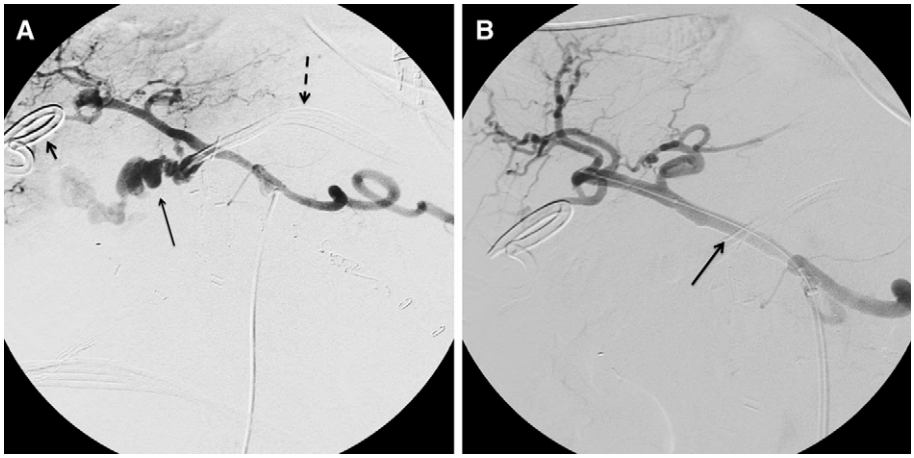

Figure 5. Acute intra-abdominal and upper gastrointestinal bleed from gastroduodenal artery (GDA) stump blowout after Whipple's procedure. The patient presented with hematemesis and bloody discharge from the Jackson-Pratt (JP) drain. (A) Celiac arteriogram showing active extravasation from GDA stump (long arrow). Note the right upper quadrant drainage catheter (short arrow) and the JP drain (dashed arrow). (B) Celiac arteriogram after placement of a 7-mm-diameter stent graft (arrow) shows cessation of hemorrhage.

pseudoaneurysm, arterial dissection or spasm, and contrast-induced nephropathy or allergic reaction. Serious complications are uncommon, occurring in <2% of patients [46]. Angiography is limited, however, by the fact that it requires the presence of active hemorrhage at the time of study. Angiography is further complicated by the fact that 75% of bleeding ceases spontaneously (Figure 3) and there can be minute-to-minute changes in bleeding (Figure 1) [47]. The diagnostic yield of angiography is variable due to these factors. The sensitivity for a diagnosis of GI bleeding is 42%-86% and the specificity approaches 100% [48]. One group showed that an increased shock index (defined as the ratio of heart rate to systolic blood pressure) increases the likelihood of a positive angiogram [49]. The yield of angiography may also be improved by the use of CO_2 as a contrast agent [50]. A negative study requires selective angiography of all 3 mesenteric vessels, the celiac axis, superior mesenteric artery, and inferior mesenteric artery.

Some investigators have proposed the use of provocative angiography to improve the ability to detect and then treat hemorrhage, especially in the setting of recurrent hemorrhage after previously negative angiography and endoscopy [51,52]. Generally, provocative angiography is accomplished by selective intra-arterial injection of various agents, including heparin, thrombolytics (streptokinase, urokinase, tissue plasminogen activator, or reteplase), and vasodilators (nitroglycerine, tolazoline, or verapamil). Only a few studies have examined this approach, and, although the technique is safe and can induce bleeding in some patients, the studies are small, and there is no established protocol. Therefore, the technique has limited applicability to wider practice.

Treatment

Endoscopy and/or Colonoscopy

For upper GI bleeding, endoscopic therapy is highly successful, with reported success rates of 80%-90% for both arterial bleeding [19] and variceal hemorrhage [53]. For lower GI bleeding, the rate of successful treatment by colonoscopy is 8%-37% [16]. Therefore, upper GI bleeding is primary managed endoscopically with endovascular techniques reserved for failed endoscopic management. In the lower GI tract, endovascular management is used after failed colonoscopy and may be the first line of therapy in patients with massive GI bleeding and hemodynamic instability.

Endovascular Treatment

Endovascular management of GI hemorrhage was first described in 1971 and was performed by selective mesenteric arterial injection of vasopressin [35]. This technique requires prolonged infusion of vasopressin and intensive monitoring in an intensive care unit setting. The technique has a high initial success rate of 60%-100% but is hampered by high rebleeding rates of 18%-50% [54–57]. In addition, the technique is contraindicated in patients with severe coronary artery disease, limb ischemia, severe hypertension, or arrhythmia [46]. The use of vasopressin has largely been supplanted by modern embolization techniques.

With advances in catheter technology and the introduction of microcatheters, selective and superselective embolization is now the endovascular treatment of choice for GI hemorrhage. A wide variety of embolic materials are available, including autologous blood clot, gelatin sponge, small particles of polyvinyl alcohol or resin, and coils (both macrocoils formed of 0.035-inch-diameter metal or microcoils formed of 0.018-inch-diameter metal) and liquid embolic agents, such as n-butyl cyanoacrylate glue [58] or liquid polyvinyl alcohol copolymer [59]. Coils are often the preferred agent due to the wide range of lengths and diameters available and to the ability to very precisely deploy them. Particulate agents carry a risk of distal embolization or nontarget embolization. Liquid embolic agents can be difficult to control and should only be used by experienced

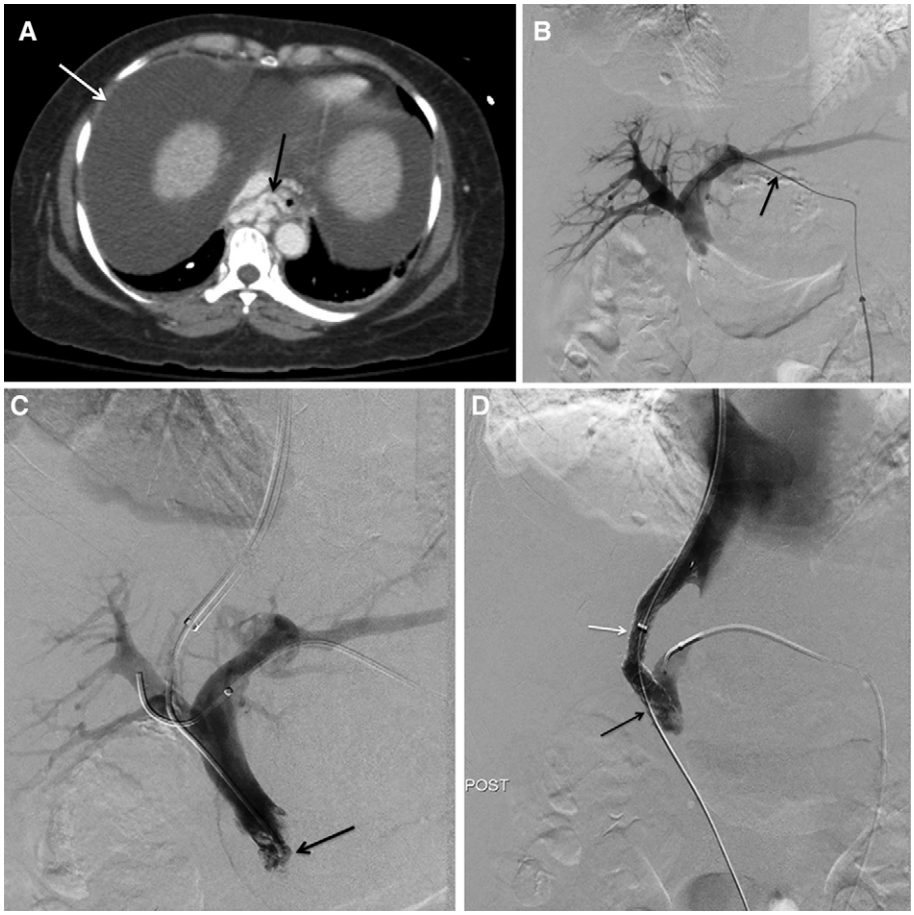

Figure 6. Transjugular portosystemic shunt (TIPSS) insertion for refractory variceal hemorrhage after 2 attempts at endoscopic hemostasis. (A) Axial venous phase computed tomography shows large volume ascites (white arrow) and large varices at the gastroesophageal junction (black arrow). (B) Direct portography via a percutaneous catheter (arrow) inserted via the left portal vein. This catheter allows for portography, direct portal pressure measurement and acts as a target for the transhepatic puncture needle. (C) A transhepatic catheter has been advanced from the right hepatic vein into the upper superior mesenteric vein (SMV). Note the filling defect (arrow) indicating thrombus in the proximal SMV. (D) After TIPSS insertion, venography shows preferential filling of the TIPSS and appropriate decreased filling of the peripheral portal veins. Note the different appearance of the uncovered portion of the stent in the portal vein (black arrow) and the covered portion of the stent in the hepatic parenchymal tract (white arrow).

operators. The success rate of embolization in modern series is from 70%-90% for lower GI bleeding [60] and from 66%-82% for upper GI bleeding, with recurrence rates of <15% [61]. Recurrent hemorrhage can be treated by repeated embolization if necessary.

The approach to embolization depends on the site and nature of the hemorrhage. Ideally, superselective embolization of an actively bleeding vessel is performed with a microcoil after cannulating the bleeding artery with a microcatheter (Figures 1 and 4), which is particularly important in the lower-GI tract where there are fewer collaterals and a higher risk of ischemia with less-selective embolization [37,62,63]. In the upper GI tract, nonselective embolization of the gastroduodenal artery may be performed in the setting of diffuse bleeding or a large pseudoaneurysm, or an inability to access an actively bleeding artery. Because of the extensive collateral arterial network in the upper GI tract, ischemic complications are rare. Another consequence of these collaterals is the need to embolize both proximal and distal to an aneurysm or bleeding vessel to prevent retrograde perfusion from a collateral vessel, which is colloquially known as embolizing both the ''front door'' and the ''back door,'' which is particularly important in embolization of the gastroduodenal artery or the inferior and superior pancreaticoduodenal arteries. It is critical to perform both celiac and superior mesenteric artery arteriography after completing embolization in the gastroduodenal artery or the pancreaticoduodenal branches to rule of persistent bleeding via collaterals.

Occasionally, in the upper GI tract, in the setting of a recent positive endoscopy in which bleeding could not be stopped by endoscopic techniques, active extravasation cannot be identified. In this setting, it is acceptable to perform empiric embolization of either the left gastric artery or the gastroduodenal artery. The appropriate artery can be chosen based on the placement of an endoscopic clip at the site of bleeding. Alternatively, empiric embolization can be based on the described location of bleeding, with antral and duodenal lesions treated by empiric embolization of the gastroduodenal artery and lesser curvature or fundal lesions being treated with left gastric artery embolization. This approach has been shown to stop bleeding and to reduce

recurrent hemorrhage [64]. It has also been shown to be as effective as embolization of an actively bleeding vessel [65,66], particularly in patients with gastric sources of bleeding [67].

GI hemorrhage may also be due to arterioenteric fistula or postsurgical pseudoaneurysm or arterial stump "blow-out." These etiologies of hemorrhage can also be treated with endovascular techniques [68]. In the setting of a noncritical parent artery, hemostasis can be obtained by embolization of the parent artery [69]. If the parent vessel is critical, then hemostasis can be obtained by placement of a stent graft [70] (Figure 5). There is a high infection rate in these stent grafts, and surgery is often needed at a later date to definitively manage this difficult problem.

For patients with variceal hemorrhage, who have failed endoscopic therapy, endovascular treatment is accomplished by insertion of a transjugular intrahepatic portosystemic shunt [71,72]. In this method, a tract is created between a hepatic vein (usually the right) and a portal vein (usually the right). This tract is then lined with a specially designed partially covered and partially uncovered stent (Figure 6) and is dilated with an angioplasty balloon with the goal of reducing the portosystemic gradient to <12 mm Hg. This technique has a high technical success rate and low rebleeding rate, particularly for esophageal varices [73]. This purpose-built stent graft has 89% primary patency at 12 months, improved over noncovered stents and nondedicated stent grafts [74]. Early use of a transjugular intrahepatic portosystemic shunt is associated with improved survival over best medical therapy [71].

Gastric varices can also be treated by balloon-occluded retrograde transvenous obliteration [75], which is a relatively new technique with a high technical and clinical success rate in the acute setting; it is also effective in reducing rebleeding rates [76,77]. Balloon-occluded retrograde transvenous obliteration is performed via a retrograde approach from the common femoral vein. An occlusion balloon is placed into the left adrenal vein, and the varices can then be embolized with a variety of agents, most commonly a sclerosant such as sodium tetradecyl sulfate or n-butyl cyanoacrylate glue.

Surgery

In the acute setting, most investigators consider less-invasive treatment with endoscopy or transcatheter techniques preferable to surgical treatment, which has high morbidity and mortality [78]. Surgery is reserved for intractable bleeding after failed attempts at endoscopic and endovascular therapy. When surgery is performed, a directed segmental resection is the preferred treatment. This approach has a lower morbidity and mortality rate with a low rate of rebleeding [48].

Conclusion

Radiologists have an extremely important role in the diagnosis and treatment of patients with acute GI hemorrhage. Although this role is somewhat secondary in patients with upper GI bleeding because of the diagnostic and therapeutic success of endoscopy, imaging and endovascular therapy has a more primary role in the management of lower GI bleeding. With the results of recent prospective studies and meta-analyses, CT can be recommended as the initial imaging modality in hemodynamically stable patients with GI hemorrhage. For those who are unstable, catheter angiography should be the modality of choice because it allows for simultaneous diagnosis and treatment. If endovascular management is appropriate, then the best practice, when possible, is superselective embolization of the bleeding vessel.

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