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Techniques in
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Acute Gastrointestinal Hemorrhage

T. Gregory Walker, MD

Although most cases of acute gastrointestinal (GI) hemorrhage either spontaneously resolve or respond to medical management and/or endoscopic treatment, there remain a significant number of patients who require emergency evaluation and treatment by the interventional radiologist. Any angiographic evaluation should begin with selective catheterization of the artery supplying the most likely site of bleeding, as determined by the available clinical, endoscopic, and imaging data. If a source of hemorrhage is identified, superselective catheterization followed by transcatheter embolization with microcoils is the most effective means of successfully controlling hemorrhage while minimizing potential complications. This is now well-recognized as a viable and safe alternative to emergency surgery. In selected situations transcatheter intra-arterial infusion of vasopressin may also be useful in controlling acute GI bleeding. One must be aware of the various side effects and potential complications associated with this treatment, however, and recognize the high rebleeding rate. In this article, we review the current role of angiography, transcatheter arterial embolization, and infusion therapy in the evaluation and management of GI hemorrhage.
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KEYWORDS gastrointestinal hemorrhage, angiodysplasia, transcatheter embolization, vasopressin infusion

Most cases of acute bleeding resolve spontaneously and, of those that do not, the majority respond to either medical management with fluid resuscitation, correction of any coagulopathy, and administration of blood products or endoscopic intervention.[1] Although the number of patients who present with acute gastrointestinal (GI) hemorrhage requiring angiography and/or transcatheter intervention has generally decreased, there are still patients who do not respond to either medical or endoscopic management and thus require emergency evaluation and treatment by the interventional radiologist.

GI hemorrhage is categorized according to the location of bleeding and is thus subdivided into the upper and lower GI tracts. The upper GI system extends from the esophagus to the ligament of Treitz, while the latter includes the small bowel, colon, and rectum. The distinction is important, as localization of the bleeding source determines the therapeutic approach.

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Clinical Evaluation and Management of the Patient

Because most cases of acute GI hemorrhage spontaneously resolve, the initial management should be directed at patient stabilization through administration of fluids and blood product, correction of coagulation abnormalities, placement of appropriate intravenous access lines, and insertion of a nasogastric tube if necessary. Direct clinical evidence of active hemorrhage may be present in the form of persistent melena, hematochezia, or hematemesis. The vital signs must be closely monitored for signs of active bleeding that could manifest as tachycardia, hypotension, and potential hypoxemia. Hemodynamic instability despite vigorous resuscitation is the best indicator of active bleeding.

Statistically, acute GI hemorrhage is far more common from an upper than a lower GI source, with the former most commonly caused by either peptic ulcer disease or gastritis (Fig. 1). The etiology of the latter, in young adults, is inflammatory bowel disease (Fig. 2) and, in patients older than 50 years, is diverticulitis (Fig. 3) and, to a lesser extent, angiodysplasia. Lower GI bleeding originates from the colon in 80% of individuals, involving the ascending colon in one-third, the transverse colon in another one-third, and the remainder in the descending colon and rectosigmoid.

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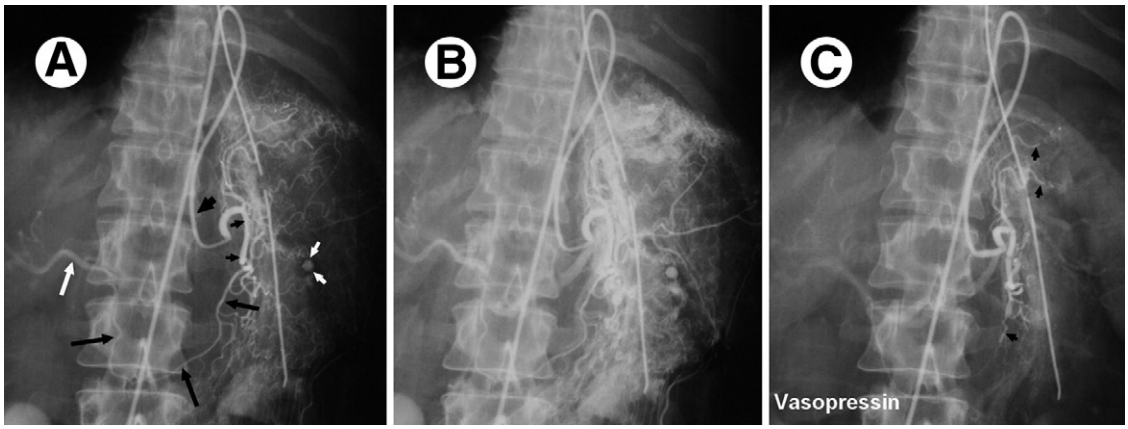

Figure 1 (A) A Cobra catheter (large black arrowhead) configured into a Waltman loop has been used to selectively catheterize the left gastric artery (small black arrowheads), demonstrating a small contrast collection (white arrowheads) that represents a bleeding gastric ulcer. There is also filling of the right gastric artery (black arrows) and a hepatic arterial branch (white arrow). (B) In the parenchymal phase of the arteriogram, the contrast collection remains prominent, with diffuse gastric mucosal hyperemia, representing gastritis. (C) Selective transcatheter vasopressin infusion was effective in controlling bleeding. The gastric arterial branches show marked vasoconstriction (small black arrowheads) and contrast extravasation is no longer present.

If an upper GI source of bleeding seems most likely, endoscopy should be the initial diagnostic study, as a source can usually be identified and often treated by the gastroenterologist. The detection and treatment success rates in patients with lower GI bleeding are generally less, particularly if there is rapid and significant bleeding.

Indications for Angiography

Imaging studies may be useful if a bleeding site cannot be identified endoscopically or if a catheter-based intervention is being considered as a treatment option. Because GI hemorrhage is typically intermittent, the successful angiographic demonstration of the source of hemorrhage is dependent on the presence of active bleeding at the time of the examination. Bleeding rates of 0.5-1.0 mL/min have been considered necessary if contrast extravasation is to be visible angiographically,[2] but digital subtraction angiography (DSA) may be far more sensitive in detecting active extravasation than previously thought.[3]

In an attempt to demonstrate active bleeding, a patient may initially be evaluated with a radionuclide technetium^{99m}-tagged red blood cell scan, as the nuclear medicine study is able to demonstrate active bleeding at rates as low as 0.1 mL/min.[4-6] An arteriogram can be subsequently obtained following a positive bleeding scan (Fig. 4), as a positive scintigram increases the likelihood of a positive angiogram from 22% to 53%.[7] One must consider, however, the potential disadvantage of first obtaining a nuclear medicine study if there is clinical evidence of active bleeding at the time of the radionuclide study: active bleeding may cease during the delay caused by the nuclear medicine scan. Therefore, a potentially useful algorithm to consider is that hemodynamically unstable patients should immediately undergo angiography, while hemodynamically stable patients should first undergo nuclear medicine imaging.

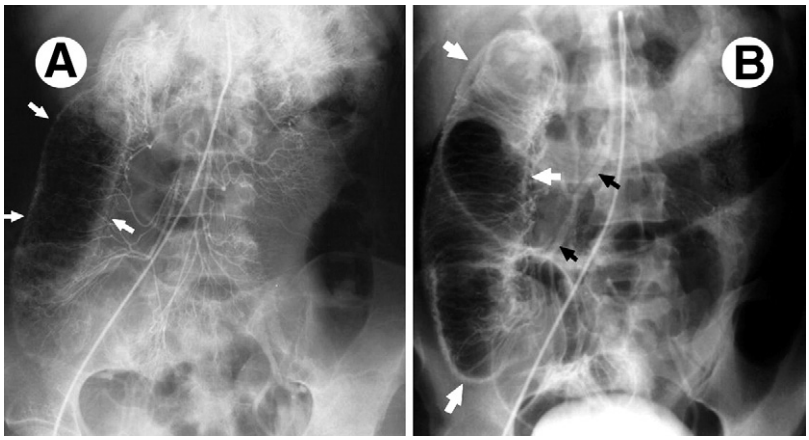

Figure 2 (A) Arterial phase of an superior mesenteric artery (SMA) angiogram shows a hyperemic blush (white arrows) to the ascending colon in a patient with lower GI bleeding, known Crohn's disease. (B) In the venous phase the hyperemia is even more evident (large white arrows) and there is a prominent ileocolic vein (black arrows).

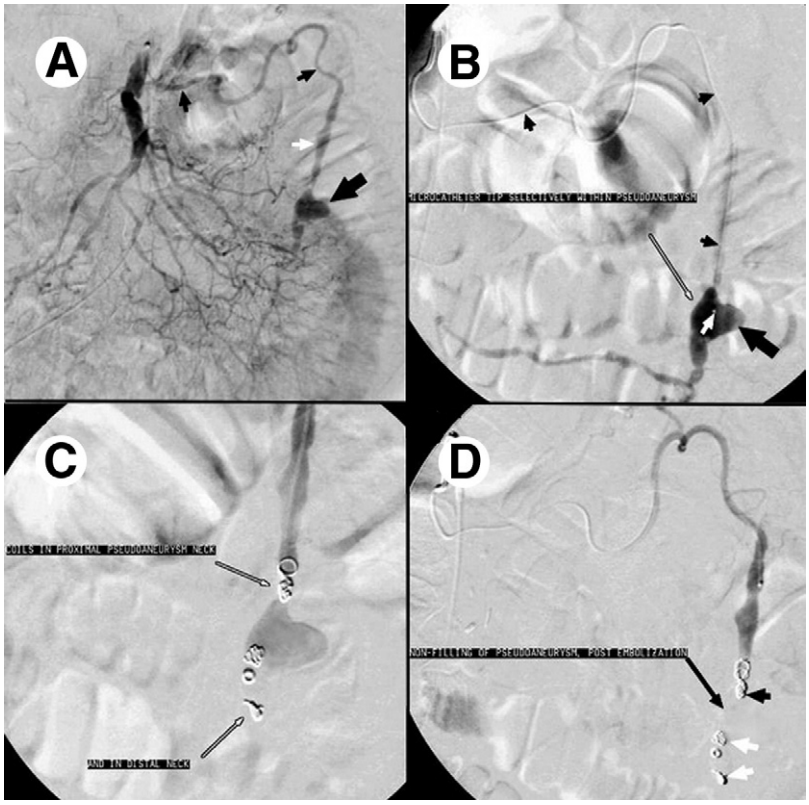

Figure 3 (A) Selective SMA arteriogram in a patient with a history of acute lower GI bleeding and prior diverticulitis shows a rounded contrast collection (large black arrow) arising from the marginal artery (white arrow) of Drummond, a continuation of the left branch of the middle colic artery (small black arrows). (B) A microcatheter (small black arrows) was successfully negotiated from the SMA into the marginal artery and the catheter tip (white arrow) was positioned in the pseudoaneurysm (large black arrow). (C) Microcoils (thin white arrows) were successfully deployed both proximally and distally relative to the pseudoaneurysm. (D) A completion angiogram shows successful pseudoaneurysm exclusion as a result of the coils placed proximal (black arrow) and distal (white arrows) to the pseudoaneurysm.

Angiographic Evaluation of Acute GI Hemorrhage

The angiographic evaluation should begin with selective catheterization of the artery supplying the most likely site of bleeding, as determined by the available clinical, endoscopic, and imaging data. For suspected upper GI hemorrhage, therefore, the celiac artery should be initially evaluated. The superior mesenteric artery (SMA) may also contribute to a site of upper GI hemorrhage through the pancreaticoduodenal arcade. The primary territories within the distribution of the SMA, however, are the small bowel and the ascending and transverse portion of the colon. The inferior mesenteric artery (IMA) supplies the splenic flexure, descending and sigmoid colonic segments, as well as the rectum and anus. There is an additional arterial supply to the rectosigmoid and anus that arises from the internal iliac arteries, particularly in the presence of IMA occlusion (Fig. 5). One must be aware of other variations that may occur in the mesenteric circulation in the presence of occlusive disease; for example, the SMA may provide the entire supply to the descending and sigmoid colon when there is IMA occlusion. Congenital variant vascular anatomy must also be considered during the angiographic evaluation of GI bleeding. The dorsal pancreatic artery may give origin to the entire middle colic artery in up to 2% of patients (Fig. 6). A celiac arteriogram may be necessary to fully evaluate the lower GI arterial supply if this situation exists, particularly in the presence of negative SMA and IMA arteriograms. If SMA, IMA, celiac, and internal iliac arteriography fail to localize active hemorrhage or fail to demonstrate all the mesenteric vascular segments, then variant anatomy must be considered, such as anomalous vessels arising directly from the aorta (eg, anomalous ileocolic artery or a middle mesenteric artery) (Fig. 7).

Catheter configurations that have an angled hook, such as the Cobra, Sos Omni selective, and Simmons, are useful for engaging the celiac and SMA origins, while selective catheterization of a cephalad coursing vessel, such as the left gastric artery, may require formation of a Waltman loop. For accessing the IMA, catheters, such as the Simmons, Sos Omni selective, and Mikaelsson, are useful. The injection and filming parameters should allow not only for visualization of the arterial phase but also of the parenchymal and venous phases. Proper magnification and the use of optimal projections that include the entire vascular territory of the artery that is being evaluated are necessary for detection of a bleeding source.

The classic angiographic finding that confirms active GI bleeding is extravasation of contrast material (Fig. 8). Extrav-

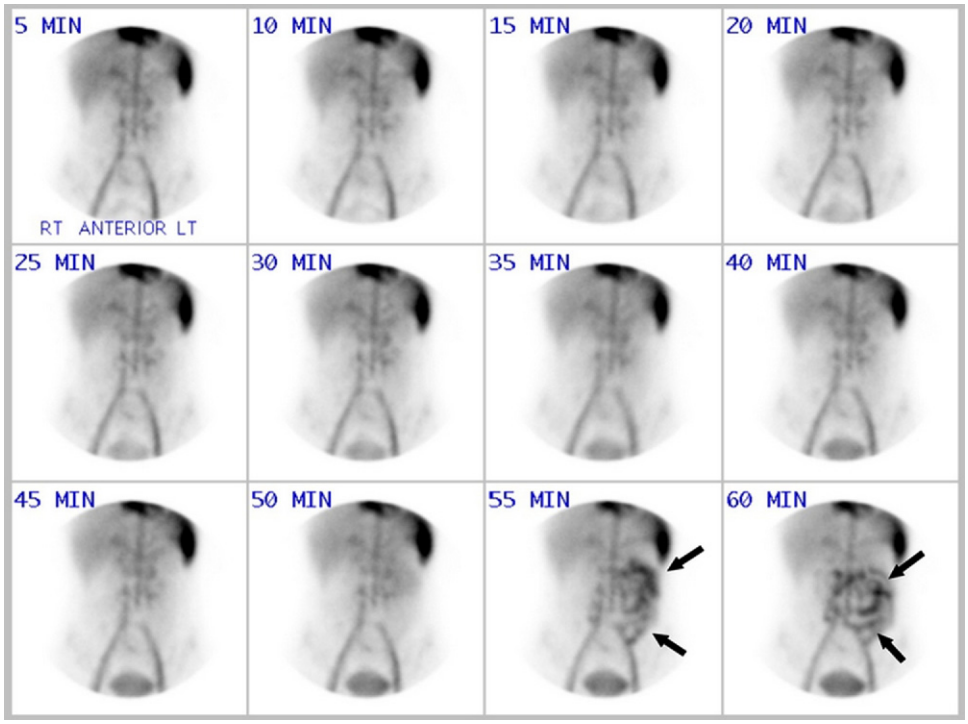

Figure 4 A radionuclide Tc^{99m}-labeled red blood cell scan in a patient who presented with acute lower GI hemorrhage shows marked accumulation of the radioisotope within small bowel loops (black arrows) in the left lower abdomen, indicative of active bleeding from a small intestinal source. (Color version of figure is available online.)

asation must occasionally be differentiated from entities that mimic its appearance, such as a hypervascular bowel mucosa, adrenal gland vascular blush, and DSA misregistration artifacts from bowel peristalsis or respiratory motion. There are also angiographic findings other than contrast extravasation that may be seen in certain pathologic conditions and are suggestive of the cause and/or source of the GI bleeding. In peptic ulcer disease, for example, small contrast collections may be seen within an ulcer crater or may outline the gastric or duodenal mucosa. Occasionally, the extravasated contrast medium will pool within the gastric rugae or within bowel folds or haustra so the contrast assumes the appearance of a vein, the "pseudo-vein sign" (Fig. 9). This may be differentiated from a true venous structure by the unusual location and appearance, as well as by the persistence beyond the venous phase of the contrast injection. Angiographic demonstration of a gastric ulcer may require subselective catheterization of celiac branches, such as the left gastric artery, although the bleeding source may also potentially arise from the right gastric, short gastric, or either the left or the right gastroepiploic arteries. Additionally, the gastroduodenal artery may supply a bleeding pyloric or duodenal ulcer.

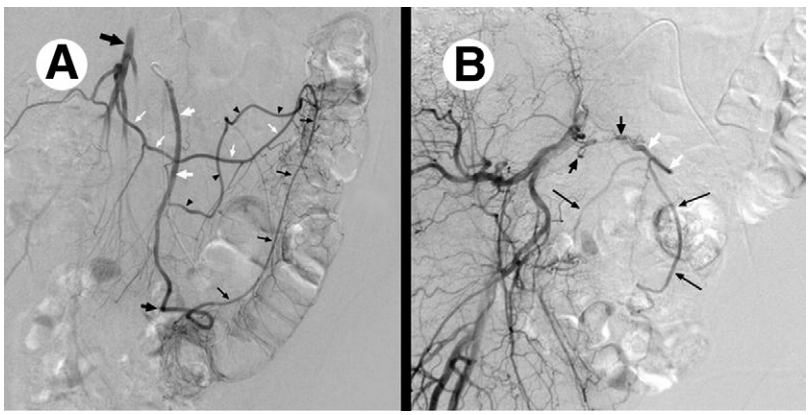

Figure 5 (A) Selective IMA (large white arrows) angiography opacifies the left colic (small black arrowheads), middle colic (small white arrows), and marginal (small black arrows) arteries, with retrograde filling of the SMA (large black arrow), but there is abrupt occlusion (medium black arrow) where the IMA should continue as the superior hemorrhoidal artery. (B) Selective internal iliac angiography shows collaterals (small black arrowheads) reconstituting the distal superior hemorrhoidal artery (white arrowheads) and terminal branches (thin black arrows).

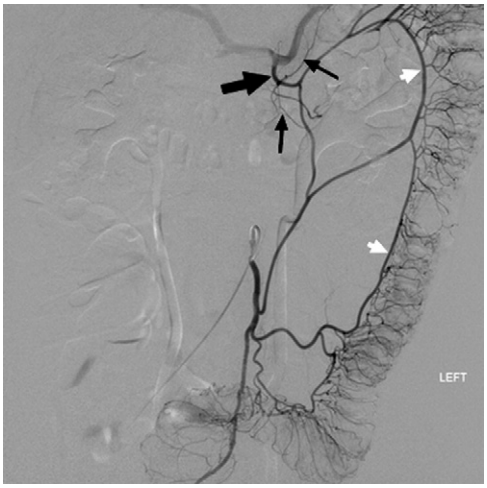

Figure 6 IMA arteriogram shows an intact marginal artery of Drummond (white arrowheads) and demonstrates retrograde filling of the left branch of the middle colic artery that arises from the dorsal pancreatic artery (large black arrow). Pancreatic branches are seen (small black arrows) and there is partial retrograde filling of the hepatic and splenic arteries.

Arterial pseudoaneurysm formation is another angiographic abnormality that may be a manifestation of GI hemorrhage and may serve to localize the site of bleeding. These occur most frequently in patients who have chronic pancreatitis. Hemosuccus pancreaticus refers to bleeding through the pancreatic duct and is caused by a pseudocyst or an arterial pseudoaneurysm that has resulted from chronic exposure of the arterial wall to the inflammatory effects of the pancreatic digestive enzymes.[8] The weakened arterial wall may allow for the intermittent bleeding that characterizes hemosuccus pancreaticus, or the pseudoaneurysm may catastrophically rupture and cause acute massive, life-threatening intra-abdominal hemorrhage. Contrast-enhanced computed tomographic angiography can be very effective in demonstrating the characteristic pseudoaneurysms (Fig. 10) and plays an increasingly important role in the diagnosis of acute GI hemorrhage that is secondary to pancreatitis, while angiographic transcatheter therapy provides the best treatment option in these patients (Fig. 11). Given both the diffuse nature of the inflammatory process seen in pancreatitis and the pancreatic arterial supply from both the celiac and the superior mesenteric arteries, pseudoaneurysms may be present involving either or both of these arteries or their branches. The angiographic evaluation must therefore include both the celiac and the SMA and transcatheter treatment may involve multiple sites (Fig. 12). Surgical intervention is particularly difficult in these patients given the extensive inflammatory process that characterizes this cause of bleeding.

Arterial pseudoaneurysms may also be seen in patients with chronic occlusive disease of the celiac artery and involve the pancreaticoduodenal arteries (Fig. 13). These are rupture-prone and may cause acute massive upper GI hemorrhage. Because of the celiac arterial occlusive disease, the angiographic evaluation involves selective catheterization of the SMA, to image the pancreaticoduodenal arcade. Any intervention, such as coil embolization, must typically also be performed from an SMA access, although there have been rare cases in which a percutaneous route has been used to treat such an abnormality.

Rather than contrast extravasation, angiodysplasia and arteriovenous malformations (AVMs) are characterized by early and prolonged opacification of a draining vein, accompanied by an abnormal tangle of vessels that may appear as a blush (Fig. 14). The simultaneous filling of the feeding artery and the draining vein may give a characteristic "tram-track" sign. Up to 80% of angiodysplastic lesions are in the right colon, although other parts of the gut, particularly the lower small intestine, may be affected. Angiodysplasia is a relatively common finding in older patient, aged 60-80 years, and may be an asymptomatic incidentally noted lesion identified at colonoscopy. Because the dilated vessels are superficial, however, they may bleed spontaneously and patients can either present acutely with overt hemorrhage or insidiously with iron deficiency anemia. Once bleeding has begun, recurrent episodic hemorrhage or persistent iron deficiency requiring

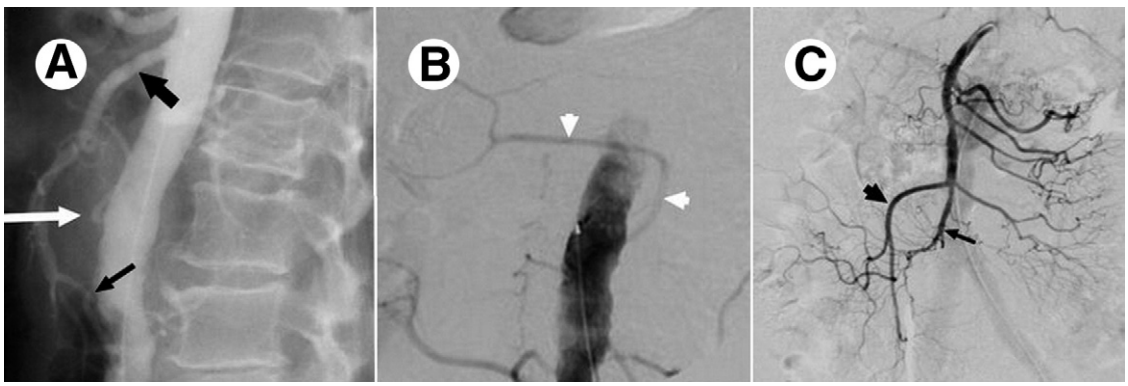

Figure 7 (A) Lateral aortogram shows an anomalous vessel (white arrow) arising from the infrarenal aorta between the SMA (large black arrow) and the IMA (small black arrow). (B) Anteroposterior aortogram shows this vessel (white arrowheads) coursing toward the right lateral abdomen. (C) An SMA arteriogram shows the ileocolic artery (black arrowhead) but absent right and middle colic arteries. A prominent ileal branch (small black arrow) courses inferiorly. The anomalous vessel from the aorta, termed the middle mesenteric artery, perfuses the right and middle colic arterial territories of the colon.

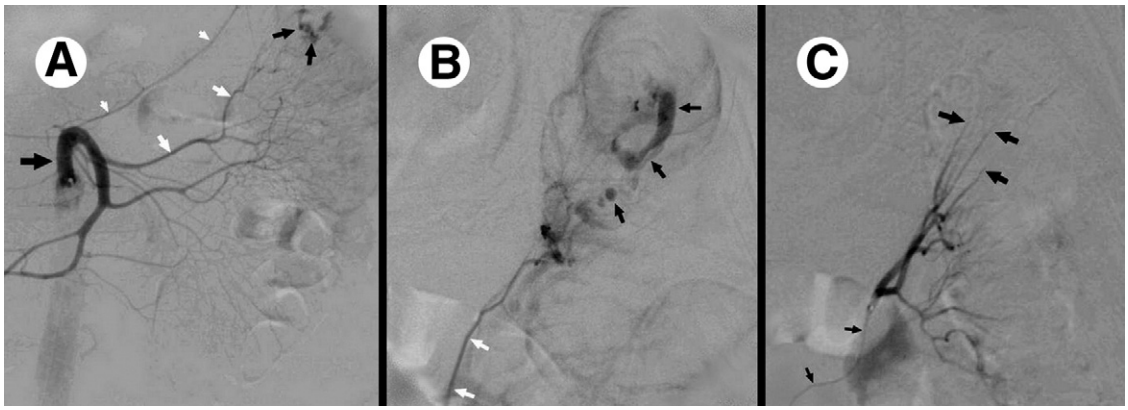

Figure 8 (A) SMA angiography shows the left branch of the middle colic artery (small white arrowheads) and demonstrates contrast extravasation (small black arrows) arising from the first jejunal division (white arrows) of the SMA (large black arrow). (B) Superselective catheterization (small white arrows) shows the extensive extravasation (black arrows) at the site of hemorrhage. (C) Following embolization through the microcatheter (small black arrows) with a small volume of 500- to 700-μm particles there is cessation of hemorrhage and distal nonfilling of terminal branches (large black arrows). Use of particles may result in a more distal embolization and may thus increase the risk of bowel ischemia.

repeated transfusion is not uncommon. Symptomatic lesions may be effectively treated endoscopically with laser or heat coagulation or with sclerotherapy. These procedures are not risk-free in the thin cecal wall and have caused serosal irritation and posttreatment bleeding. For patients with repeated bleeding from intestinal vascular malformations, pharmacologic treatment using estrogens (eg, 0.05 mg ethinyl estradiol and 1 mg norethisterone) can be effective in reducing transfusion requirements. Transcatheter arterial embolization of colonic angiodysplasia can be an effective treatment in emergencies[8-11] but it also carries a risk of inducing significant colonic ischemia.[12] Definitive and curative treatment usually requires surgical resection.

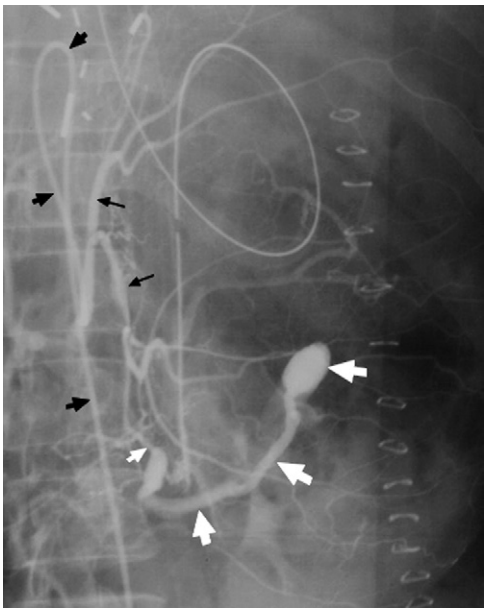

Figure 9 A catheter configured into a Waltman loop (black arrowheads) has been used to selectively catheterize the left gastric artery (small black arrows) in a case of massive upper GI bleeding. A prominent tubular structure (large white arrows) originating from a gastric branch (small white arrow) represents extravasated contrast medium that has pooled within the gastric rugae and has assumed the appearance of a vein, the "pseudo-vein sign."

Angiographic Management of Acute GI Hemorrhage

Transcatheter Arterial Embolization

Transcatheter arterial embolization is an effective means of interrupting blood flow to the source of hemorrhage, while maintaining bowel viability. Although there is a risk of bowel ischemia and/or infarction, the coaxial catheter systems and the variety of available embolic agents that are used for embolotherapy allow for very selective and precise treatment and thus minimize these complications. Additionally, the GI tract has a rich collateral blood supply, with extensive vascular arcades that allow for safe embolization if certain principles are observed. The objective of embolization therapy is to achieve a compromise between selective arterial inflow reduction and maintenance of collateral arterial blood flow. Thus, arterial inflow must be sufficiently decreased to allow for hemostasis, but not to the extent that causes complete devascularization. This goal underscores the importance of both an intact coagulation cascade and a superselective embolization.[11]

In most of the early literature, embolization was performed proximally in the superior or inferior mesenteric arteries because microcatheters facilitating superselective embolization were not yet available. The technique that is currently used for transcatheter arterial embolization involves the initial placement of a diagnostic catheter (4 or 5 F) into the main trunk of the feeding artery, followed by coaxial introduction of a microcatheter. The latter is the crucial step in modern embolization and requires superselective placement of a 3-F coaxial microcatheter over a 0.018-in or 0.014-in wire. The characteristics of the guidewire are important, as it should be flexible, hydrophilic, and steerable with a torque device. The wire tip must be carefully advanced under fluoroscopy in a

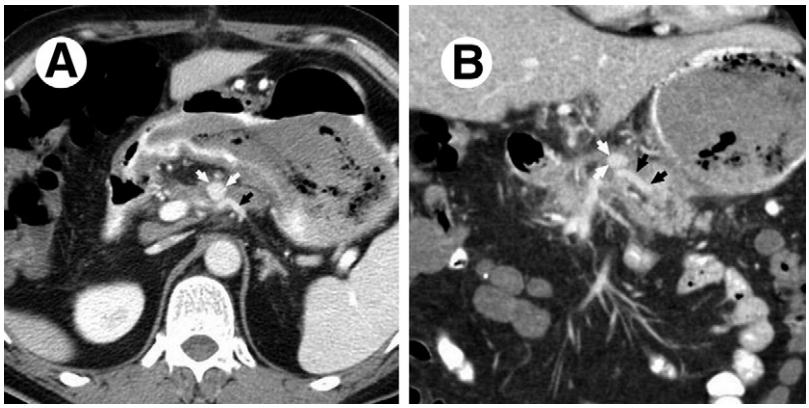

Figure 10 (A) Axial and (B) coronal images from a contrast-enhanced computed tomographic scan show a pseudoaneurysm (white arrows) arising from the splenic vein (black arrows) in a patient with GI bleeding and a history of pancreatitis.

smooth and controlled motion to avoid vasospasm, dissection, or vessel perforation. Vasodilators, such as verapamil 100-200 μg or nitroglycerin 100-300 μg, may be used to treat any vasospasm that may occur. Using the wire as a guide, the catheter should travel closely behind and eventually engage the target vessel.

The optimal level of embolization varies according to the site of hemorrhage. In general, we deploy embolic agents as selectively as possible and prefer not to routinely attempt embolization unless a microcatheter has been advanced close to the bleeding point. The risk of infarction is related to both the embolic agent and the proximity of embolization. In the colon, for example, ischemia and infarction may result from embolization of proximal branches supplying a large area of bowel or embolization of multiple distal arteries that do not have sufficient collateral flow. Submucosal collateral blood flow may be preserved only when arteries to a short segment of bowel are embolized, so one should attempt to occlude arteries to as short a segment of bowel as possible. One must be aware that embolization in the setting of prior GI surgery or radiation therapy may impose a greater risk of infarction because of the interruption of collaterals.

Various agents may be used for transcatheter embolization. The most commonly used agents include pledgets of absorbable gelatin sponge (Gelfoam Pfizer, Inc, New York, NY), microcoils and particulate agents, such as polyvinyl alcohol (eg, Bead Block; Biocompatibles International, Farnham, UK), and other spherical agents (eg, Embospheres; BioSphere Medical, Inc, Rockland, MA; Embozene microspheres; CeloNova BioSciences, Inc, Newnan, GA). We often use microcoils, which have the advantage of good radiopacity that allows a precise deployment that permits one to reduce the arterial perfusion pressure to the bleeding site while preserving sufficient collateral flow. The wide range of coil sizes allows one to appropriately match the coil to the target vessel diameter. Each microcoil is delivered sequentially, until hemostasis has been achieved. Intra-arterial microcoil placement is analogous to placement of a surgical ligature. The coil physically occludes the vascular lumen and causes a decreased perfusion pressure, while the attached

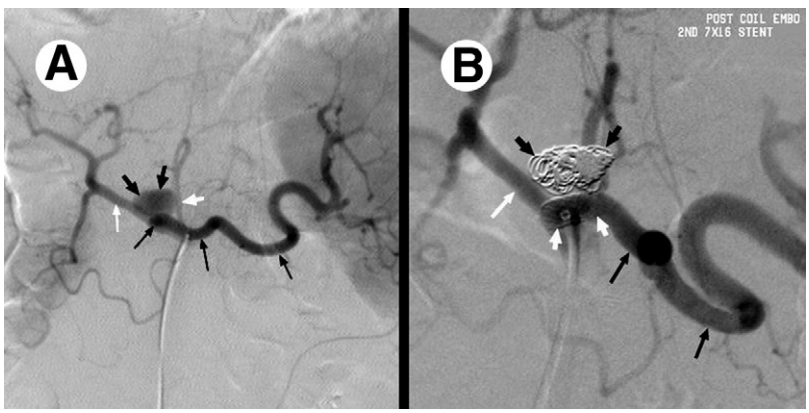

Figure 11 (A) Celiac angiogram confirms a pseudoaneurysm (large black arrows) arising from the proximal splenic artery (thin black arrows) in proximity to the left gastric (white arrowhead) and common hepatic (thin white arrow) arteries. (B) Following embolization with coils (small black arrows) that were deployed through the interstices of a noncovered stent (small white arrows), the pseudoaneurysm is occluded and flow is preserved in the splenic (thin black arrows) and common hepatic (thin white arrows) arteries.

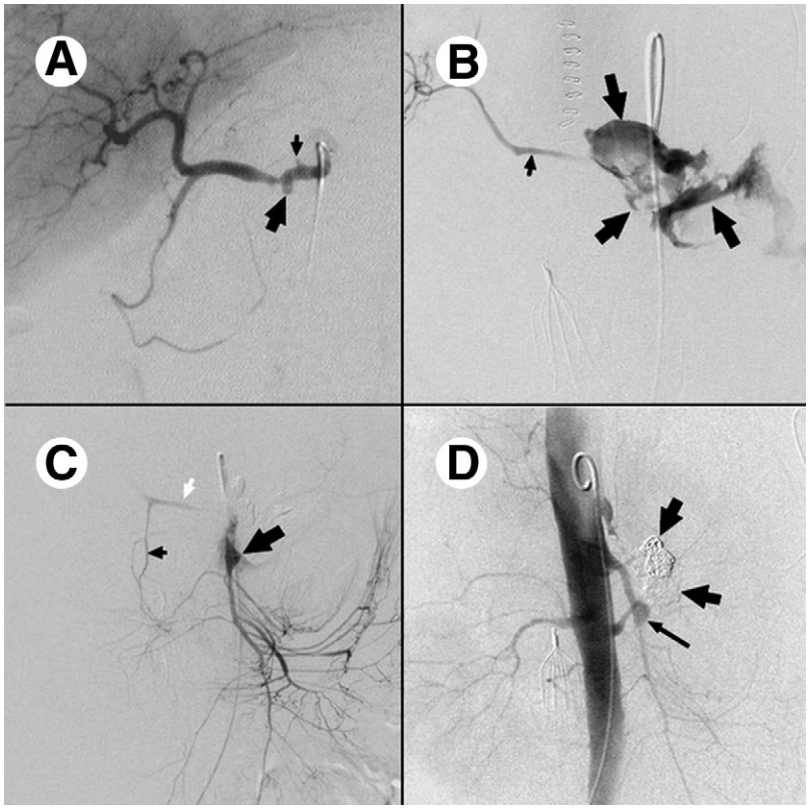

Figure 12 (A) Celiac arteriogram obtained in a patient with pancreatitis and a history of prior splenectomy shows focal contour irregularities (large and small black arrows) that represent pseudoaneurysms. (B) These were not treated and the patient later presented with acute upper GI hemorrhage, which was apparent on repeat angiography as massive extravasation (large black arrows). Marked vasoconstriction is seen in the common hepatic artery (small black arrow) in response to the bleeding. (C) Selective SMA angiography shows another pseudoaneurysm (large black arrow) in the main trunk, with retrograde filling of the gastroduodenal (small black arrowhead) and hepatic arteries (white arrow). (D) Lateral aortogram after embolization with coils (large black arrows) and n-butyl-cyanoacrylate ("glue") shows cessation of hemorrhage. The SMA pseudoaneurysm (thin black arrow) is again evident.

synthetic fibers maximize thrombogenicity. Microcoils can be deployed either with a saline flush or by using a wire-push technique. The former is more rapid and may achieve a more distal embolization from the microcatheter tip; however, the pushing wire technique is more precise and is thus preferred for deploying the initial coil.

Gelfoam pledgets and particulate agents may also be used successfully but are more difficult to control than microcoils.

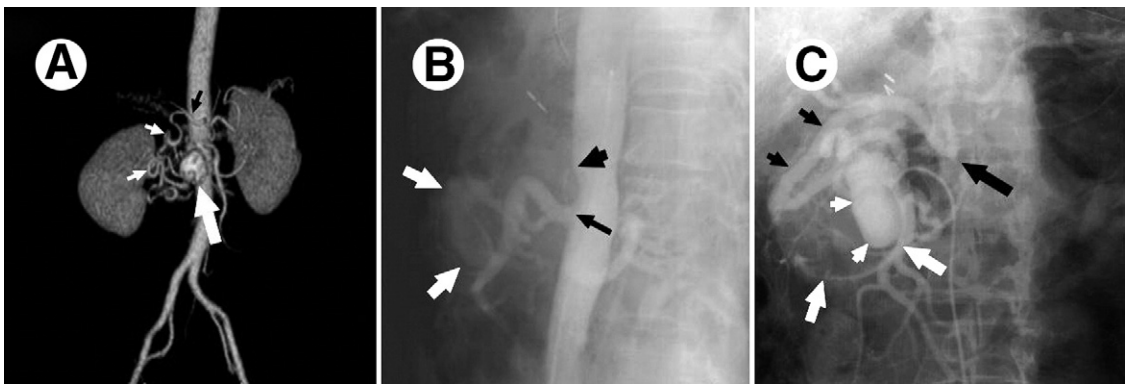

Figure 13 (A) 3D computed tomographic angiography image shows a large pseudoaneurysm (large white arrow) arising from one of the proximal inferior pancreaticoduodenal arteries, with the arcade (small white arrows) reconstituting the hepatic artery (small black arrow). (B) Lateral aortogram confirms SMA patency (black arrow) and celiac artery occlusion (black arrowhead). The pseudoaneurysm is faintly opacified (white arrows). (C) Selective SMA arteriogram shows the calcified rim (large white arrows) and contrast-filled (white arrowheads) central lumen of the pseudoaneurysm, with retrograde filling of the gastroduodenal artery (black arrowheads) via the vascular arcade. The celiac branches are also filled in retrograde fashion, demonstrating the point of celiac trunk occlusion (large black arrow).

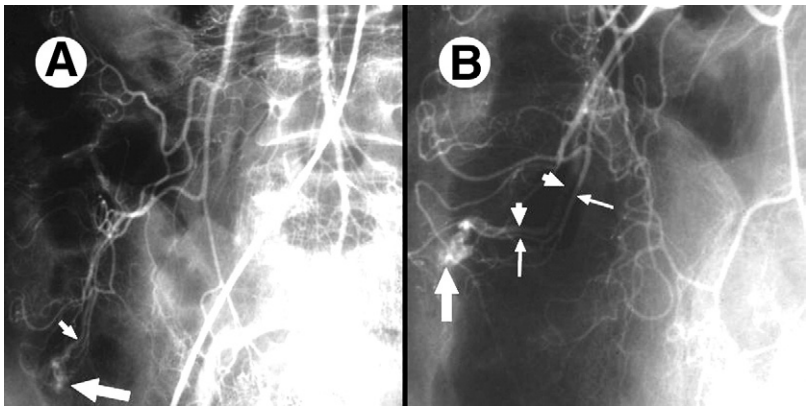

Figure 14 (A) Image from SMA arteriogram shows a vascular blush (large white arrow) in the right lower quadrant, with a prominent early draining vein (small white arrow). (B) A magnified view of the right lower quadrant demonstrates a vascular tangle (large white arrow), with simultaneous filling of the feeding artery (narrow white arrows) and the draining vein (white arrowheads), giving a characteristic "tram-track" sign. The findings are typical of angiodysplasia.

Gelfoam is a temporary agent and often cannot easily be deployed superselectively (Figs. 15 and 16). A disadvantage of the particulate agents is that small diameters may reach the intramural circulation distal to collaterals or may reflux into nontarget arteries.

Technical Challenges of Transcatheter Arterial Embolization

Because of the intermittent nature of GI hemorrhage, arteriography fails to demonstrate a distinct bleeding site in a considerable number of patients, and thus embolization is not possible. Furthermore, a negative arteriogram fails to guide emergency surgery and delays operative decision-making. In such circumstances, some investigators have advocated provocation of bleeding with vasodilators, anticoagulants, and/or thrombolytics in association with tagged red blood cell scans or angiography.[13,14] This may be appropriate for a patient who has undergone multiple blood transfusions and a prior exhaustive workup that has failed to localize the occult bleeding site. Additionally, there must be no contraindications to the administration of a thrombolytic agent.

Different methods of inducing bleeding and different rates of success have been reported. An optimal protocol has yet to be established and the procedure has also yet to become accepted by clinicians as part of the evaluation of GI bleeding. The technique continues to evolve as experience and comfort using thrombolytic agents in the setting of nonlocalized bleeding increases. One reported protocol used a combination of intravenous heparin, intra-arterial tolazoline, and intra-arterial tissue plasminogen activator (t-PA) to provoke bleeding. Doses used included 3000-10,000 U heparin, 25-100 mg intra-arterial tolazoline, and 10-50 mg intra-arterial t-PA (mean, 20.3 mg). The investigators also noted that more patients had provoked bleeding after smaller rather than larger doses of t-PA.[14] Tolazoline (Priscoline) was used for a vasodilatory effect but was withdrawn from the United States market in 2002 by Novartis Pharmaceuticals, the sole manufacturer of this drug. Alternative intra-arterial vasodilators that may be used include verapamil, 100-200 μg, and nitroglycerin, 100-300 μg, with the former showing the greater vasodilatory effects. We have occasionally used a similar transcatheter regimen in which we administered a vasodilator and a dose of 5000 U intra-arterial heparin and 5-10 mg

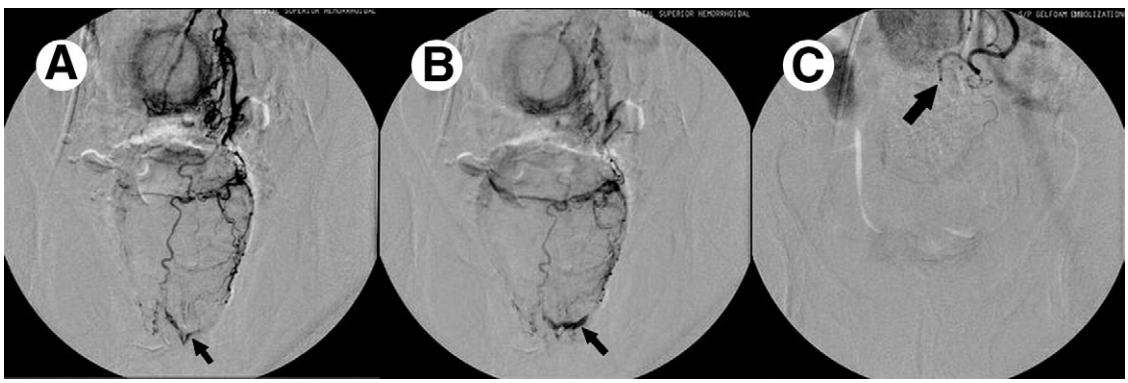

Figure 15 (A) Superior hemorrhoidal arteriogram in a patient with severe hemorrhage following rectal biopsy shows contrast extravasation (medium black arrow) that (B) rapidly increases (medium black arrow) during the DSA imaging. (C) Following transcatheter embolization with Gelfoam pledgets there is cessation of bleeding, but the level of occlusion is more proximal (large black arrow) than the superselective position that can usually be achieved with microcoils.

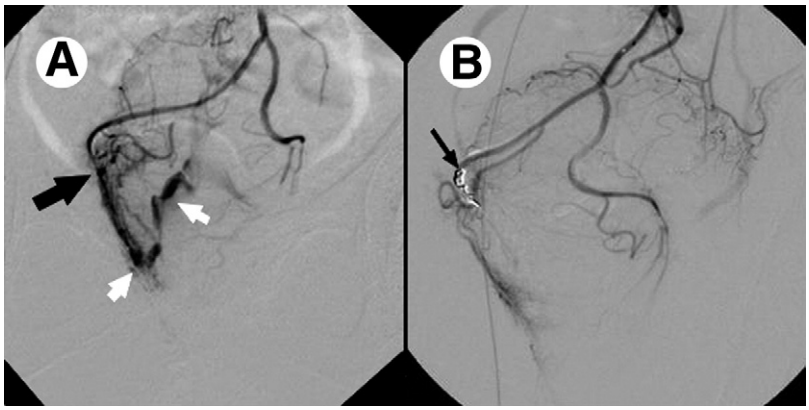

Figure 16 (A) Contrast extravasation (white arrows) denoting rectal hemorrhage from a terminal branch (large black arrow) of the superior hemorrhoidal artery could be treated with a more superselective catheterization (B) and microcoil embolization (thin black arrow) than when gelfoam was used for embolization in a similar location (Fig. 15).

intra-arterial t-PA, with resultant provocation of bleeding that allowed for subsequent successful transcatheter embolization.

Superselective embolization of the arterial supply to the bleeding source may be technically demanding, particularly in older patients who may have significant atherosclerotic disease. The mesenteric vasculature and the various arterial arcades are often tortuous, and the smaller arteries are prone to vasospasm and care must be taken to avoid dissection.

An arterial bleeding site may receive a dual blood supply because of the rich collateral arcades that characterize the mesenteric circulation. One must therefore catheterize and inject both potential sources of perfusion to the lesion and be prepared to embolize 2 separate vessels if necessary. Although this dual approach will control the hemorrhage, it will also increase the risk of bowel ischemia.[15]

While angiodysplasia and AVMs may initially respond to embolization, recurrent hemorrhage is frequent and, as noted, surgical resection of the involved bowel segment is often required. A small bowel AVM is much more easily localized at surgery if an embolization coil has been placed distally in the arterial branch that supplies the lesion, so that it is palpable or visible to the surgeon.[16]

Results of Transcatheter Arterial Embolization

The current technique of embolization in the treatment of acute GI hemorrhage successfully controls bleeding in about 80%-90% of patients.[17-21] Recurrent hemorrhage is infrequent, with the exception of angiodysplasia, AVMs, and inflammatory lesions. Recurrences can usually be angiographically re-evaluated and, if a bleeding source is identified, treated with repeat embolization.

Complications of Transcatheter Arterial Embolization

Transcatheter arterial embolization for the treatment of acute GI hemorrhage is safe, with major adverse events occurring in less than 2% of patients. A fraction of patients embolized superselectively will develop minor, asymptomatic, and self-limited ischemic changes, such as small ulcers that can only be detected incidentally via objective follow-up methods, such as by endoscopy, pathologic surgical specimen, or a radiographic imaging examination. Additionally, superselective microcoil embolization is unlikely to result in delayed infarction. As previously noted, the risk of ischemia is higher when particulate agents are used, because of the more distal embolization that can be achieved. If major bowel ischemia occurs several months to years later, it is more likely attributable to a new and acute insult, such as thromboembolic disease affecting the mesenteric arterial bed.

Nontarget embolization with microcoils is rare, as the coils are introduced only after a microcatheter has been successfully negotiated into the target vessel. One must carefully choose appropriate sized microcoils however, as a coil that is oversized relative to the target vessel may displace the microcatheter from its superselective position (Fig. 17). This could lead to deployment of the microcoil in a nontarget location. Similarly, undersized coils may fail to adequately occlude the target vessel or may lodge distal to the lesion that is to be treated.

Vasopressin Infusion Therapy

Vasopressin (Pitressin) is a naturally occurring hormone that causes constriction of both the mesenteric arteries and the smooth muscle of the bowel wall. The intra-arterial transcatheter infusion of vasopressin proximal to a mesenteric arterial bleeding site will reduce blood flow, thereby lowering the perfusion pressure and permitting clot formation at the lesion (Fig. 18). There are several situations in which this form of treatment for GI bleeding should not be used. These include bleeding that originates from a large diameter artery, such as the gastroduodenal, splenic, or proximal superior mesenteric arteries, or that occurs at a site with a dual blood supply, such as the duodenum. It is also contraindicated in patients who have severe coronary artery disease, extreme hypertension, limb ischemia, or cardiac arrhythmias. Superselective embolotherapy is now used preferentially over vasopressin infusion for treating GI hemorrhage because embolization poses fewer risks and can be completed more rapidly than a vasopres-

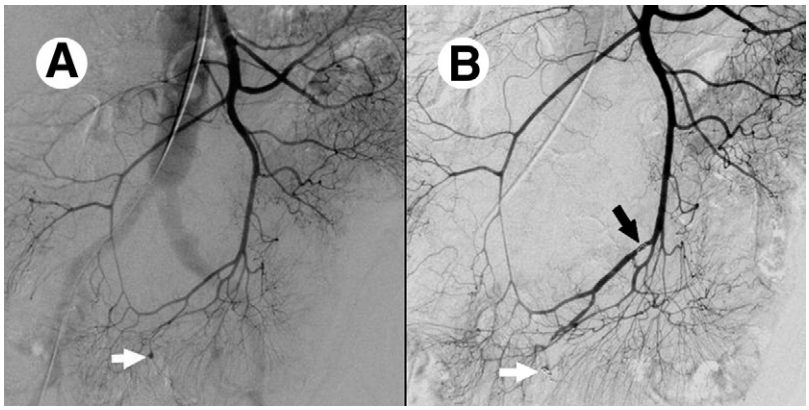

Figure 17 (A) SMA arteriogram in a patient with acute onset of rectal hemorrhage shows a focal collection (large white arrow) supplied by an arcade of the ileocolic branch of the SMA. (B) Superselective microcoil transcatheter embolization successfully controlled hemorrhage (large white arrow), but during the embolization the microcatheter tip was displaced and a microcoil deployed in a nontarget location (large black arrow).

sin infusion protocol. Vasopressin may still be useful in certain situations, despite the numerous side effects and the high rebleeding rates. One may consider using vasopressin for treating lesions that are inaccessible to a microcatheter, for diffuse mucosal oozing, and for controlling multiple sites of hemorrhage in high-risk surgical patients.

Technique of Vasopressin Infusion Therapy

Vasopressin is typically infused into the central vessel (eg, celiac, SMA, or IMA) that supplies the bleeding site via a catheter that has been placed proximally; to avoid bowel ischemia and potential infarction, a distal infusion should not be attempted. One hundred unified atomic mass units of vasopressin are mixed in 500 mL normal saline and the infusion is started at 0.2 U/min for 20 minutes using an arterial infusion pump set at 60 mL/h. If there is no cessation of bleeding, the dose is increased by 0.1 U/min up to a maximum of 0.4 U/min; each dosage change is followed by a repeat angiogram 20-30 minutes later to assess the effectiveness.[22] If the bleeding stops, the catheter is left in place for a 24-hour infusion at the effective dosage, with monitoring in the intensive care unit. After another repeat angiogram shows control of bleeding, the vasopressin infusion is gradually reduced over 24-48 hours and then vasopressin is replaced with an infusion of normal saline or 5% dextrose in water for 6-12 hours.

Results of Vasopressin Infusion Therapy

This form of therapy is particularly effective in controlling diverticular and gastric mucosal hemorrhage (Fig. 1), with

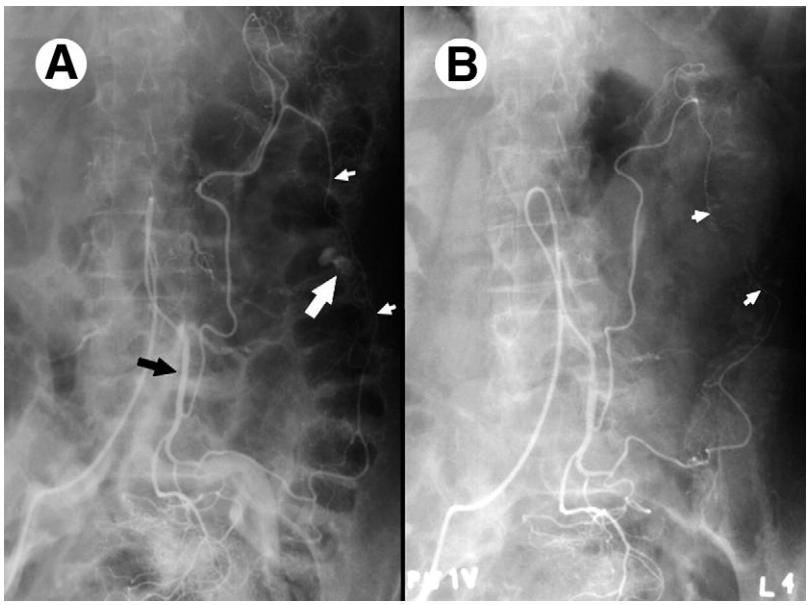

Figure 18 (A) Selective IMA (black arrow) arteriogram in a patient with lower GI bleeding shows contrast extravasation (large white arrow) that arises from the marginal artery (small white arrows). The appearance is typical of diverticular hemorrhage. (B) Repeat angiography following transarterial infusion of vasopressin at a rate of 0.4 U/min shows vasoconstriction (small white arrows) and control of bleeding.

initial success rates ranging from 60% to 90%.[22-25] As previously noted, however, there is a very high rate of rebleeding that may be up to 50%.

Complications of Vasopressin Infusion Therapy

Mild abdominal pain may occur at the initiation of the infusion and should be closely monitored, as persistence and/or worsening can be an indicator of bowel ischemia. If side effects develop during treatment, the vasopressin can be tapered to a lower dose or may need to be discontinued. Additional side effects and potential complications of vasopressin therapy include angina, myocardial infarction, hypertension, volume overload, abdominal cramps, and mesenteric ischemia. Some of the potential adverse effects may be treated or even pretreated. The simultaneous administration of intravenous, sublingual, or transdermal nitroglycerin may prevent or reverse the cardiotoxic side effects of vasopressin infusion.[26]

Conclusions

Technical refinements both in diagnostic angiography and in transcatheter arterial embolization have strengthened this therapy for the emergency management of acute GI hemorrhage. Superselective catheterization using a coaxial system that allows for microcoil embolization has emerged as an effective and safe alternative to emergency surgery. Until new medical and technological advances provide a superior alternative, acute GI hemorrhage that is refractory to medical and endoscopic management should be evaluated by mesenteric angiography and should be treated with attempted superselective microcoil embolization. Catheter-based technology in the hands of an experienced operator who possesses advanced microcatheter-based skills will likely remain an essential component in the emergency management of acute GI hemorrhage.

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